PASSMEDICINE PART 2

NEUROLOGY
OPHTHALMOLOGY
PSYCHIATRY

2024

A 68-year-old man is admitted to the unit with an exacerbation of severe pain around his abdomen. He describes it as a tight band like pain which is burning in character. He has a past medical history of metastatic prostate cancer. His drug history includes morphine modified release (100mg, twice daily), duloxetine (60mg, twice daily), pregabalin (200mg, three times per day). He states that the pain initially responded well to the analgesics he is on however over the past few weeks it has got progressively worse and culminated in a severe attack of pain today.

After discussion with the palliative care consultant, you elect to start the patient on an NMDA receptor antagonist. What medication will you start?

○ Fentanyl

○ Codeine

○ Paracetamol

○ Gabapentin

○ Ketamine

Submit answer

Reference ranges

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1 -

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After discussion with the palliative care consultant, you elect to start the patient on an NMDA receptor antagonist. What medication will you start?

Fentanyl	12%
Codeine	2%
Paracetamol	1%
Gabapentin	17%
Ketamine	67%

Ketamine can be used as a third line agent in the management of neuropathic pain which is poorly responsive to titrated opioids and oral adjuvant analgesics

Important for me Less important

The patient describes classic radicular pain associated with thoracic radiculopathy. It is described as a tight band like pain which may be burning, tingling, or shooting in character. He is on a large dose of opioids and two neuropathic agents which act on different pathways.

He is already on a strong opioid so it would be inappropriate to use a middle strength opioid (e.g. codeine, dihydrocodeine, or buprenorphine). Fentanyl would only be used in stable pain, or with specific other indications such as renal disease. In this case it is unlikely to control his pain.

He is already on two neuropathic agents so it would be inappropriate to start gabapentin.

Ketamine can be used for neuropathic pain poorly responsive to titrated opioids and oral adjuvant analgesics (e.g. antidepressant and/or anticonvulsant) particularly when there is abnormal pain sensitivity (e.g. allodynia, hyperalgesia or hyperpathia). Ketamine is an NMDA receptor antagonists.

Side effects to be aware of include hypertension, raised intracranial pressure and hallucinations.

Gabapentin was designed to mimic the neurotransmitter GABA. It does not, however, bind to GABA receptors. Its mechanism of action is to block voltage-gated calcium channels.

👍 👎 Discuss (4) Improve

Next question ›

Neuropathic pain ★

Neuropathic pain may be defined as pain which arises following damage or disruption of the nervous system. It is often difficult to treat and responds poorly to standard analgesia.

Examples include:
- diabetic neuropathy
- post-herpetic neuralgia
- trigeminal neuralgia
- prolapsed intervertebral disc

NICE updated their guidance on the management of neuropathic pain in 2013:
- first-line treatment*: amitriptyline, duloxetine, gabapentin or pregabalin
    - if the first-line drug treatment does not work try one of the other 3 drugs
    - in contrast to standard analgesics, drugs for neuropathic pain are typically used as monotherapy, i.e. if not working then drugs should be switched, not added
- tramadol may be used as 'rescue therapy' for exacerbations of neuropathic pain
- topical capsaicin may be used for localised neuropathic pain (e.g. post-herpetic neuralgia)
- pain management clinics may be useful in patients with resistant problems

*please note that for some specific conditions the guidance may vary. For example carbamazepine is used first-line for trigeminal neuralgia

AI + Q 123

Next question ›

B *I* A T↕

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Links

NICE 👍 13 👎 4

2013 Neuropathic pain guidelines

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Media

HEART ATTACK
REFERRED PAIN
ACUTE PAIN
CHRONIC PAIN
PAIN
BRAIN
SPINAL CORD
C FIBRES
A-DELTA FIBRES
RECEPTORS
PERIPHERAL NERVES

Understanding The Causes of Pain and Pain Management

Zero To Finals - YouTube 👍 1 👎 0

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Question 2 of 409

A 34-year-old female presents to the clinic. She states that she sometimes has difficulties leaving the house due to fear of being in situations where she cannot escape. Some examples she gives includes being in busy places such as shopping malls and public transport. She states that when she he is at home and in her place of work she functions well, with regards to both productivity and interpersonal relationships.

What is the most likely diagnosis?

- ○ Panic disorder
- ○ Generalized anxiety disorder
- ○ Agoraphobia
- ○ Depression
- ○ Social phobia

Submit answer

Reference ranges

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What is the most likely diagnosis?

Panic disorder	3%
Generalized anxiety disorder	4%
Agoraphobia	75%
Depression	0%
Social phobia	17%

Agoraphobia describes a fear of open spaces. It may or may not be associated with panic disorder

Important for me Less important

The clinical features are suggestive of agoraphobia which is described as fear of open spaces and being trapped in situations which are difficult to escape from.

Agoraphobia may or may not be associated with panic disorder. However, there is no evidence that this patient is having panic attacks.

Generalized anxiety disorder describes a syndrome where patients feel anxiety in a range of different situations including work and personal life. Social phobia describes a condition in which patients have a fear of being in social situations e.g. a fear of embarrassing themselves. The patient functions well with others both at work and home suggesting an alternative diagnosis.

There is nothing to suggest that the patient is depressed.

Discuss (1) Improve

Next question ›

Agoraphobia ★

Agoraphobia is primarily describes a fear of open spaces but also includes related aspects, e.g. the presence of crowds or the difficulty of escaping to a safe place

+ Q 123

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Question 3 of 409

A 64-year-old man presents to the endocrinology clinic with a palpable neck mass. He has noticed this incidentally. He denies any problems in swallowing, speaking or breathing. On more detailed history he does admit to a 3kg weight loss over the last 4 months which he cannot explain but is happy with as he has always been overweight. He has a past medical history of appendicectomy, hypertension, high cholesterol. He takes aspirin and atorvastatin. Thyroid function tests are normal. On examination, he has a roughly 3cm mass in the anterior neck triangle on the left side. It is firm and non-tender. What is the most appropriate next investigation?

○ CT neck ×

○ US neck ×

○ MRI neck ×

○ CT neck, chest, abdomen and pelvis ×

○ Nuclear scan whole body ×

Submit answer

Reference ranges

Score: **0%**

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Question 3 of 409 ✗

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CT neck	7%
US neck	71%
MRI neck	4%
CT neck, chest, abdomen and pelvis	17%
Nuclear scan whole body	1%

Ultrasound neck is the most appropriate 1st line imaging for a neck mass

Important for me Less important

This patient has a new thyroid mass and is clinically and biochemically euthyroid. The best investigation is ultrasound, which can help determine features suggestive of malignancy in thyroid cancer. CT and MRI of the neck can be useful if there is a goitre to determine the extent of the goitre. Further CT scanning can be used in staging his disease to see if there is any malignant spread.

Discuss (5) Improve

Next question ›

Neck lumps ★

The table below gives characteristic exam question features for conditions causing neck lumps:

Condition	**Notes**
Reactive lymphadenopathy	By far the most common cause of neck swellings. There may be a history of local infection or a generalised viral illness
Lymphoma	Rubbery, painless lymphadenopathy The phenomenon of pain whilst drinking alcohol is very uncommon There may be associated night sweats and splenomegaly
Thyroid swelling	May be hypo-, eu- or hyperthyroid symptomatically Moves upwards on swallowing
Thyroglossal cyst	More common in patients < 20 years old Usually midline, between the isthmus of the thyroid and the hyoid bone Moves upwards with protrusion of the tongue May be painful if infected
Pharyngeal pouch	More common in older men Represents a posteromedial herniation between thyropharyngeus and cricopharyngeus muscles Usually not seen but if large then a midline lump in the neck that gurgles on palpation Typical symptoms are dysphagia, regurgitation, aspiration and chronic cough
Cystic hygroma	A congenital lymphatic lesion (lymphangioma) typically found in the neck, classically on the left side Most are evident at birth, around 90% present before 2 years of age
Branchial cyst	An oval, mobile cystic mass that develops between the sternocleidomastoid muscle and the pharynx Develop due to failure of obliteration of the second branchial cleft in embryonic development Usually present in early adulthood
Cervical rib	More common in adult females Around 10% develop thoracic outlet syndrome
Carotid aneurysm	Pulsatile lateral neck mass which doesn't move on swallowing

AI + Q 123

Next question ›

B *I* A

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Links

Clinical Knowledge Summaries 👍 21 👎 8

Neck lumps

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Media

Thyroglossal duct cyst

Osmosis - YouTube 👍 0 👎 0

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A 72-year-old man is brought to hospital by his wife. He had a headache the day before and now is increasingly vague. He has Parkinson's disease and has been taking his medications consistently over the past month and is usually able to walk with a stick. He was born in Russia and has been exposed to tuberculosis in his younger years. He immigrated twenty years ago to the UK and has not travelled abroad in the last year. There is no history of memory problems.

On examination, he responds to voice and is able to obey commands. He cannot recall a history and is very sleepy. There is no neck stiffness and the tone in the limbs is normal though power is difficult to assess. He does not appear to be in any discomfort. His chest is clear and abdomen is soft but he has a temperature of 38.2ºC. His pupils are responsive, there is no facial droop and his speech is clear with no slurring.

Na^+	139 mmol/l
K^+	4.2 mmol/l
Urea	4.1 mmol/l
Creatinine	65 µmol/l
CRP	70 mg/l

Hb	120 g/l
Platelets	400 * 10^9/l
WBC	13 * 10^9/l

Lumbar puncture	
Protein	0.6g/l
Glucose	3.2 mmol/l
Cells	500 mononuclear cells per mm^3
CT head	no acute intracranial or extracranial bleed demonstrated
MRI head	pending

What is the likely diagnosis?

○ Cerebral abscess ×

○ Encephalitis ×

○ Parkinson's dementia ×

○ Subdural empyema ×

○ Tuberculous meningitis ×

Submit answer

Reference ranges

Score: **0%**

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2 -
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Question 4 of 409 ✗

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Lumbar puncture	
Protein	0.6g/l
Glucose	3.2 mmol/l
Cells	500 mononuclear cells per mm^3
CT head	no acute intracranial or extracranial bleed demonstrated
MRI head	pending

What is the likely diagnosis?

Cerebral abscess	13%
Encephalitis	45%
Parkinson's dementia	4%

Subdural empyema 9%

Tuberculous meningitis 29%

Clouded consciousness and fever -> encephalitis

Important for me Less important

This case demonstrates the difficulties in diagnosis particularly when the patient cannot cooperate for full examination findings. He has fever and reduced consciousness. A cerebral abscess usually has focal neurology which is not seen here. Parkinson's dementia would present more gradually and not have fever. Subdural empyema would be seen as an old subdural on the CT scan. Tuberculous meningitis is possible but usually presents more gradually and often with cranial nerve palsies. Therefore encephalitis is the most likely diagnosis.

👍 👎 Discuss (9) Improve

Next question >

Encephalitis ★

Features
- fever, headache, psychiatric symptoms, seizures, vomiting
- focal features e.g. aphasia
- peripheral lesions (e.g. cold sores) have no relation to the presence of HSV encephalitis

Pathophysiology
- HSV-1 is responsible for 95% of cases in adults
- typically affects temporal and inferior frontal lobes

Investigation
- cerebrospinal fluid
    - lymphocytosis
    - elevated protein
    - PCR for HSV, VZV and enteroviruses
- neuroimaging
    - medial temporal and inferior frontal changes (e.g. petechial haemorrhages)
    - normal in one-third of patients
    - MRI is better

- EEG
    - lateralised periodic discharges at 2 Hz

Management

- intravenous aciclovir should be started in all cases of suspected encephalitis

123

Next question >

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Question 5 of 409

A 28-year-old male presents with feet, hand and perioral paresthesia associated with progressive weakness in his lower limbs over approximately two weeks. On examination he has normal tone and reduced reflexes in his lower limbs, reduced proprioception in feet and hands, and proximal weakness in his lower limbs. His MRI spine was unremarkable.

Other results:

CSF protein	2.0g/L
HIV	negative
Lyme serology	negative
Epstein-Barr virus	negative

What is the correct treatment?

- ○ Intravenous immunoglobulins ×
- ○ Intravenous methylprednisolone ×
- ○ Cyclophosphamide infusion ×
- ○ Pyridostigmine ×
- ○ Doxycycline ×

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Reference ranges

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Other results:

CSF protein	2.0g/L
HIV	negative
Lyme serology	negative
Epstein-Barr virus	negative

What is the correct treatment?

Intravenous immunoglobulins	79%
Intravenous methylprednisolone	16%
Cyclophosphamide infusion	1%
Pyridostigmine	4%
Doxycycline	1%

IVIG is generally used first-line for Guillain-Barre syndrome

Important for me   Less important

The history given is one typical of Guillain-Barre syndrome with the characteristic albuminocytological dissociation within the CSF (a raised protein with a normal white cell count). After assessing for respiratory dysfunction, arrhythmias and autonomic dysfunction the appropriate treatment is intravenous immunoglobulins. This has been shown to hasten recovery in patients who are either not spontaneously improving or unable to walk unaided if used within 4 weeks of neuropathic symptom onset, with better outcomes for earlier use. Plasma exchange is also another option in these patients but intravenous immunoglobulins tend to be used for reason s outlined below.

Intravenous methylprednisolone has not been shown to be beneficial, and would tend to be used in a multiple sclerosis flare. Equally, immunosuppression with cyclophosphamide is not typically used.

Use of doxycycline may be considered if Lyme disease is diagnosed, which manifest with a range of neurological signs and symptoms. Pyridostigmine would used in myasthenia gravis patients, and therefore would not be used in this case.

Discuss (6) | Improve

Next question

Guillain-Barre syndrome: management

Guillain-Barre syndrome describes an immune-mediated demyelination of the peripheral nervous system often triggered by an infection (classically *Campylobacter jejuni*).

Management
- IV immunoglobulins (IVIG) or plasma exchange can be used
    - IVIG is as effective as plasma exchange better tolerated and easier to administer
    - therefore IVIG is generally used first-line for Guillain-Barre syndrome where patients require immunomodulatory therapy
    - no benefit in combining both treatments
- steroids and immunosuppressants have not been shown to be beneficial
- FVC regularly to monitor respiratory function

Prognosis
- severe motor problems persist in about 15%
- around 5% die

AI + Q 123

Next question

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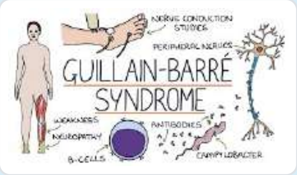
NERVE CONDUCTION STUDIES
PERIPHERAL NERVES
GUILLAIN-BARRÉ SYNDROME
WEAKNESS
NEUROPATHY
ANTIBODIES
B-CELLS
CAMPYLOBACTER

Understanding Guillain-Barre Syndrome

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SYMPTOMS - BASED on NERVES AFFECTED

Guillain-Barre syndrome

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Question 6 of 409

A 37-year-old man presents to the emergency department with a six-hour history of an acutely painful right eye associated with photophobia and reduced visual acuity. He has had no previous episodes. He has no relevant past medical history and occasionally takes codeine for back pain.

On examination, diffuse redness is noted throughout the right eye. The right pupil is smaller than the left and he is unable to count fingers in the right eye although visual acuity in the left eye is unimpaired.

What is the most likely diagnosis?

○ Acute angle closure glaucoma ×
○ Anterior uveitis ×
○ Bacterial conjunctivitis ×
○ Cluster headache ×
○ Scleritis ×

Submit answer

Reference ranges

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On examination, diffuse redness is noted throughout the right eye. The right pupil is smaller than the left and he is unable to count fingers in the right eye although visual acuity in the left eye is unimpaired.

What is the most likely diagnosis?

Acute angle closure glaucoma	44%
Anterior uveitis	49%
Bacterial conjunctivitis	0%
Cluster headache	2%
Scleritis	5%

Anterior uveitis presents with acutely painful red eye, photophobia, small pupil, reduced visual acuity. It is often associated with pus in the anterior chamber (a hypopyon)

Important for me Less important

This man has presented with a case of anterior uveitis, as confirmed by the typical findings on examination. Note that anterior uveitis can be both unilateral or bilateral, largely dependant on the cause. Although this condition is associated with a hypopyon, this is only sometimes present. A further clue is the presence of back pain in a young man which points towards a history of ankylosing spondylitis, a condition closely associated with anterior uveitis.

Acute angle-closure glaucoma does present with an acutely painful red eye however is commonly associated with a headache, nausea, and vomiting, and seeing halos. It is also more likely in the older population and particularly people with a history of presbyopia or chronic open-angle glaucoma.

Bacterial conjunctivitis does cause redness but is usually associated with itching and purulent discharge. Additionally, it is unlikely to be associated with the additional features this man has exhibited (miosis, photosensitivity, and reduced visual acuity).

Scleritis again is another cause of an acutely painful red eye however is unlikely to cause photophobia and miosis. Although scleritis is related to autoimmune conditions (such as systemic lupus erythematosus and rheumatoid arthritis), it is less associated with ankylosing spondylitis.

A cluster headache causes pain behind the eye and can be associated with redness and watering of the eye as well as miosis however attacks are more likely to be frequent and short-lived rather than persisting for six hours.

👍 👎 Discuss (2) Improve

Next question

Anterior uveitis

Anterior uveitis is one of the important differentials of a red eye. It is also referred to as iritis. Anterior uveitis describes inflammation of the anterior portion of the uvea - iris and ciliary body. It is associated with HLA-B27 and may be seen in association with other HLA-B27 linked conditions (see below).

Features

- acute onset
- ocular discomfort & pain (may increase with use)
- pupil may be small +/- irregular due to sphincter muscle contraction
- photophobia (often intense)
- blurred vision
- red eye
- lacrimation
- ciliary flush: a ring of red spreading outwards
- hypopyon; describes pus and inflammatory cells in the anterior chamber, often resulting in a visible fluid level
- visual acuity initially normal → impaired

Associated conditions

- ankylosing spondylitis
- reactive arthritis
- ulcerative colitis, Crohn's disease
- Behcet's disease
- sarcoidosis: bilateral disease may be seen

Management

- urgent review by ophthalmology
- cycloplegics (dilates the pupil which helps to relieve pain and photophobia) e.g. Atropine, cyclopentolate
- steroid eye drops

+ Q 123

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A 20-year-old woman presents to a general neurology clinic. Over the last year, she has noted that she is becoming increasingly clumsy with heavy objects. She describes struggling to wash her hair and complete certain house hold chores such as hanging washing up.

Neurological examination findings of the upper limbs have been summarised below. Power has been recorded as per the Medical Research Council (MRC) grading system.

	Left arm	**Right arm**
Tone	normal	normal
Power:		
shoulder abduction	3/5	3/5
shoulder adduction	4/5	4/5
elbow flexion	4/5	5/5
elbow extension	5/5	5/5
wrist flexion	5/5	5/5
wrist extension	5/5	5/5
Reflexes:		
Biceps	normal	normal
Triceps	normal	normal
Supinator	normal	unable to elicit
Sensation	intact	intact

When examining her cranial nerves it is noted there is drooping of her eyelids and decreased facial expression. Tests for fatigability were unremarkable. There were no other abnormal findings.

What is the most likely diagnosis?

○ Myasthenia gravis

○ Facioscapulohumeral dystrophy

○ Multiple sclerosis ×

○ Miller-Fisher syndrome ×

○ Becker's muscular dystrophy ×

Submit answer

Reference ranges

Score: **0%**

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elbow extension	5/5	5/5
wrist flexion	5/5	5/5
wrist extension	5/5	5/5
Reflexes:		
Biceps	normal	normal
Triceps	normal	normal
Supinator	normal	unable to elicit
Sensation	intact	intact

When examining her cranial nerves it is noted there is drooping of her eyelids and decreased facial expression. Tests for fatigability were unremarkable. There were no other abnormal findings.

What is the most likely diagnosis?

Myasthenia gravis 8%

Facioscapulohumeral dystrophy 77%

Multiple sclerosis	3%
Miller-Fisher syndrome	6%
Becker's muscular dystrophy	6%

Facioscapulohumeral muscular dystrophy: facial weakness (e.g. difficulty closing eyes, smiling, blowing), winged scapula and weakness of the shoulderm upper arm and hip girdle muscles

Important for me Less important

The history here is typical of facioscapulohumeral dystrophy, the diagnosis is confirmed by the examination findings which confirm a proximal weakness affecting the limb girdle and subtle signs of weakness in the face.

The absence of fatigability in muscle power excludes myasthenia gravis. The clinical picture is not of Miller-Fisher syndrome which often presents as a triad of ophthalmoplegia, ataxia, and areflexia. Becker's muscular dystrophy is excluded as it typically affects the limb girdles in males who carry the X-linked gene. The presentation is also atypical of MS.

Discuss (2) Improve

Next question >

Facioscapulohumeral muscular dystrophy ★

Facioscapulohumeral muscular dystrophy (FSHMD) is an autosomal dominant form of muscular dystrophy. As the name suggests it typically affects the face, scapula and upper arms first. Symptoms typically present by the age of 20 years.

Features
- facial muscles are involved first - difficulty closing eyes, smiling, blowing etc
- weakness of the shoulder and upper arm muscles
- abnormal prominence of the borders of the shoulder blades - 'winging'
- lower limb: hip girdle weakness, foot drop

+ Q 123

Next question >

Textbooks

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Score: **24.1%**

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A 43-year-old woman presents to the emergency department with fevers and confusion. She has no past medical history and does not take any regular medications.

On examination, her Glasgow coma scale is 13/15 (E3V4M5). There is no localizing neurological signs. Cardiovascular, respiratory and abdominal assessment is unremarkable. Her temperature is 38.8ºC.

An urgent MRI brain is organized, which demonstrates oedema of both temporal lobes.

A lumbar puncture is arranged.

What set of results is most likely to be found?

- ○ Normal white cell count / elevated protein
- ○ Normal white cell count / normal protein
- ○ Raised lymphocytes / elevated protein
- ○ Raised lymphocytes / normal protein
- ○ Raised neutrophils / elevated protein

Submit answer

Reference ranges

Score: **0%**

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An urgent MRI brain is organized, which demonstrates oedema of both temporal lobes.

A lumbar puncture is arranged.

What set of results is most likely to be found?

Normal white cell count / elevated protein	5%
Normal white cell count / normal protein	2%
Raised lymphocytes / elevated protein	57%
Raised lymphocytes / normal protein	32%
Raised neutrophils / elevated protein	5%

Herpes simplex encephalitis - the CSF usually shows a lymphocytosis amd elevated protein

Important for me Less important

Raised lymphocytes / elevated protein is the correct answer. The patient presents with fever and confusion and is pyrexial with a low GCS on examination. In this context, the MRI findings of bilateral temporal lobe oedema are highly suggestive of herpes simplex encephalitis. A lumbar puncture would typically demonstrate lymphocytosis and elevated protein.

Normal white cell count / elevated protein is incorrect. The normal white cell count points away from infection. Protein content in the CSF is usually raised either due to either an increased permeability of the blood brain barrier or synthesis of proteins within the spinal canal due to inflammatory cells. The differential for this set of results is broad and includes numerous inflammatory causes. It is however, a less likely set of results in HSV encephalitis.

Raised lymphocytes / normal protein is incorrect. This set of results may be compatible with a viral infection but in HSV encephalitis the protein is more likely raised than normal.

Raised neutrophils / elevated protein is incorrect. This set of results is more consistent with a bacterial infection.

Normal white cell count / normal protein is incorrect. This is a normal result and unlikely in HSV encephalitis.

👍 👎 Discuss (2) Improve

Next question ›

Herpes simplex encephalitis ★

Herpes simplex (HSV) encephalitis is a common topic in the exam. The virus characteristically affects the temporal lobes - questions may give the result of imaging or describe temporal lobe signs e.g. aphasia.

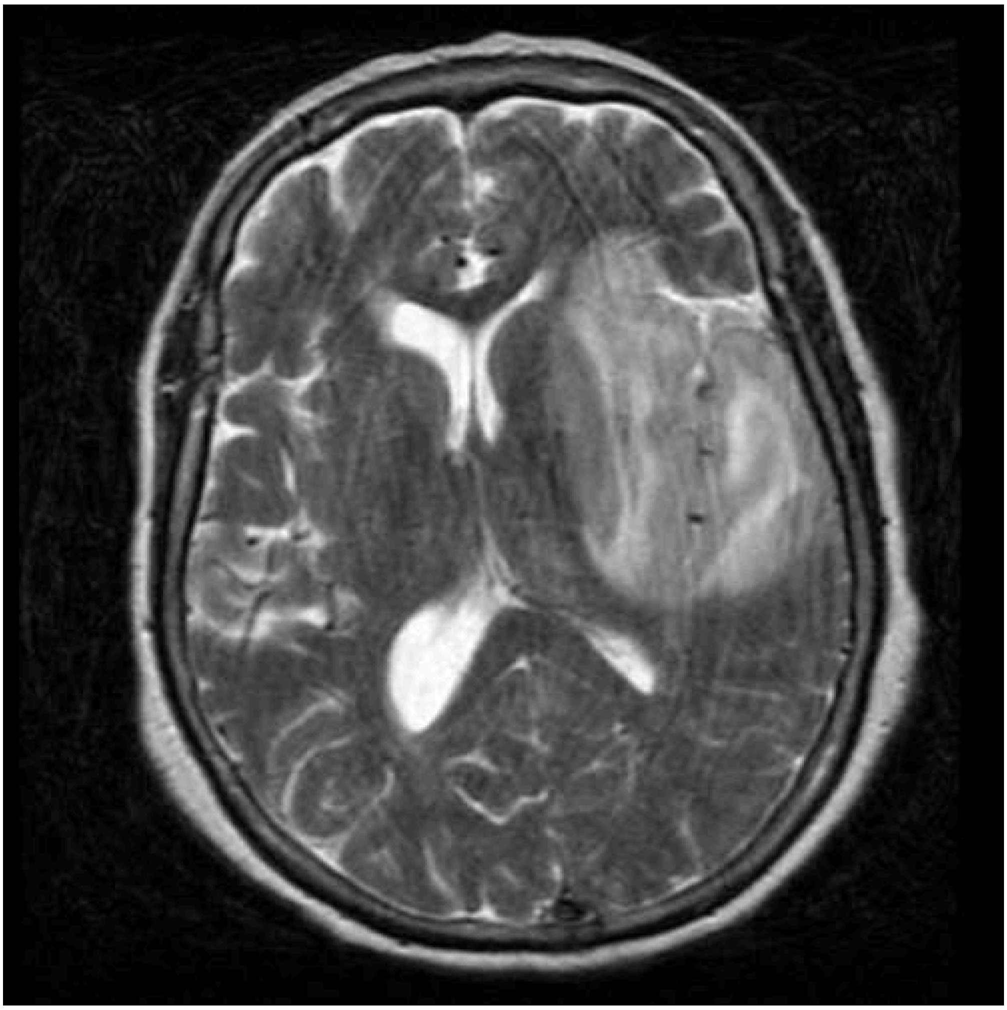
Features
- fever, headache, psychiatric symptoms, seizures, vomiting
- focal features e.g. aphasia
- peripheral lesions (e.g. cold sores) have no relation to the presence of HSV encephalitis

Pathophysiology
- HSV-1 is responsible for 95% of cases in adults
- typically affects temporal and inferior frontal lobes

Investigation
- CSF: lymphocytosis, elevated protein
- PCR for HSV
- CT: medial temporal and inferior frontal changes (e.g. petechial haemorrhages) - normal in one-third of patients
- MRI is better
- EEG pattern: lateralised periodic discharges at 2 Hz

Treatment
- intravenous aciclovir

The prognosis is dependent on whether aciclovir is commenced early. If treatment is started promptly the mortality is 10-20%. Left untreated the mortality approaches 80%



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MRI of a patient with HSV encephalitis. There is hyperintensity of the affected white matter and cortex in the medial temporal lobes and insular cortex.

+ Q 123

Next question

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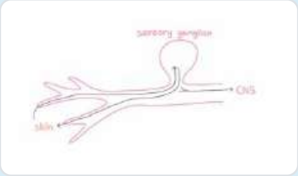
Media


sensory ganglion
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Herpes simplex encephalitis

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A 47-year-old woman was admitted from a rehabilitation unit after appearing unwell. She has a past medical history of a road traffic accident 1 year prior and spinal cord injury at the C5 level which has left her quadriplegic.

Her regular medications include laxido, codeine and paracetamol.

On examination from the end of the bed, there is marked flushing of the skin across the face and shoulders and she appears diaphoretic and extremely agitated. Her temperature is 37.6ºC, oxygen saturation 97% on air, heart rate 102 beats per minute, respiratory rate 21 breaths per minute and blood pressure 206/130 mmHg. Auscultation of the chest reveals good bilateral air entry and vesicular breath sounds. Her heart sounds are normal with no added murmurs. There is a bounding pulse peripherally and central capillary refill time of 2 seconds. There is no swelling of the lower legs and the abdomen is soft with a rounded supra-pubic mass. There is a long term catheter in-situ connected to an empty catheter bag.

Initial investigations are displayed below:

ECG	Sinus tachycardia

Hb	126 g/L	Male: (135-180) Female: (115 - 160)
Platelets	$368 * 10^9$/L	(150 - 400)
WBC	$9.3 * 10^9$/L	(4.0 - 11.0)
Na^+	141 mmol/L	(135 - 145)
K^+	4.3 mmol/L	(3.5 - 5.0)
Urea	5.3 mmol/L	(2.0 - 7.0)
Creatinine	84 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Troponin T	5ng/L	(<14)

Chest X-ray	no focal consolidation or collapse

What would be the next most appropriate management step given the likely diagnosis?

○ Treatment dose of low molecular weight heparin followed by a CTPA ×

○ IV co-amoxiclav and gentamicin ×

○ Antihypertensives ×

○ Abdominal X-ray ×

○ Antihypertensives and bladder scan ×

Submit answer

Reference ranges ⌵

Score: **0%**

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Creatinine	84 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Troponin T	5ng/L	(<14)

Chest X-ray	no focal consolidation or collapse

What would be the next most appropriate management step given the likely diagnosis?

Treatment dose of low molecular weight heparin followed by a CTPA	5%
IV co-amoxiclav and gentamicin	3%

Antihypertensives	5%
Abdominal X-ray	5%
Antihypertensives and bladder scan	81%

Management of autonomic dysreflexia involves removal/control of the stimulus and treatment of any life-threatening hypertension and/or bradycardia

Important for me Less important

Patients with a high cervical spinal cord injury are at risk of autonomic dysreflexia which can be distressing and potentially cause life-threatening hypertension. The management of autonomic dysreflexia would be to identify and remove the offending stimulus - in this case, a blocked catheter causing urinary retention - while treating extreme hypertension.

Although the patient is at risk of venous thromboembolism given her immobility. The clinical picture is more in keeping with autonomic dysreflexia secondary to urinary retention and therefore option 1 is incorrect.

The clinical picture and investigation results are not consistent with urinary sepsis and therefore option 2 is incorrect.

Antihypertensives alone do not address the underlying cause and therefore option 3 is incorrect.

An abdominal X-ray may be an appropriate further down the line if you are suspecting faecal impaction as a cause for the urinary retention or for the autonomic dysreflexia. In this case, the clinical picture is more suggestive of acute urinary retention due to a blocked catheter. To confirm and manage this retention would be the immediate priority and therefore option 4 is incorrect.

Discuss (4) Improve

Next question >

Autonomic dysreflexia

This clinical syndrome occurs in patients who have had a spinal cord injury at, or above T6 spinal level. Briefly, afferent signals, most commonly triggered by faecal impaction or urinary retention (but many other triggers have been reported) cause a sympathetic spinal reflex via thoracolumbar outflow. The usual, centrally mediated, parasympathetic response however is prevented by the cord lesion.

Features
- the result is an unbalanced physiological response, characterised by extreme hypertension
- flushing and sweating above the level of the cord lesion
- agitation
- in untreated cases, severe consequences of extreme hypertension have been reported, e.g. haemorrhagic stroke.

Management of autonomic dysreflexia involves removal/control of the stimulus and treatment of any life-threatening hypertension and/or bradycardia.

AI + Q 123

Next question >

B *I*

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Links

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Autonomic Dysreflexia

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Media

Autonomic
Dysreflexia
with
Dr Mike

Autonomic Dysreflexia

Dr Matt & Dr Mike - YouTube 👍 36 👎 2

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A 60-year-old female presents to the Emergency Department with altered sensation in both hands and reduced visual acuity in the left eye. She states that these symptoms have slowly worsened over a week. She has no issues with bladder or bowel activity.

On examination, she is haemodynamically stable. Of note, she has a sensory loss throughout her arms and legs, but no clear sensory level. Additionally, she has brisk reflexes in both the upper and lower limbs. She also has a visual acuity of 6/36 in the left eye, but normal vision in the right eye, with normal visual fields.

Her MRI scan reveals multiple continuous segments of inflamed spinal cord throughout the cervical region and left optic nerve inflammation. There are no cerebral lesions. Her cerebrospinal fluid (CSF) studies are negative for oligoclonal bands.

Which of the following tests will confirm the diagnosis?

- ○ dsDNA antibodies
- ○ Aquaporin-4 antibodies
- ○ ACh receptor antibodies
- ○ NMDA receptor antibodies
- ○ Neuronal antibodies

Submit answer

Reference ranges

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1 -
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Her MRI scan reveals multiple continuous segments of inflamed spinal cord throughout the cervical region and left optic nerve inflammation. There are no cerebral lesions. Her cerebrospinal fluid (CSF) studies are negative for oligoclonal bands.

Which of the following tests will confirm the diagnosis?

dsDNA antibodies	1%
Aquaporin-4 antibodies	79%
ACh receptor antibodies	2%
NMDA receptor antibodies	12%
Neuronal antibodies	6%

Neuromyelitis optica is associated with aquaporin 4 positive serum antibody

Important for me   Less important

The description of a patient with optic neuritis and longitudinal extensive cervical transverse myelitis with a CSF negative for oligoclonal bands is highly suggestive of neuromyelitis optica (NMO). The classic antibody associated with this disease is NMO-IgG or antibodies against aquaporin-4.

The 2006 diagnostic criteria for NMO are as follows:

Transverse myelitis and optic neuritis with at least 2 of the following features:

- MRI brain negative/nondiagnostic for multiple sclerosis

- MRI spinal cord lesion extending over 3 vertebral segments (LETM)

- NMO-IgG/Aquaporin-4 antibody seropositivity

Discuss (2) | Improve

Next question >

Neuromyelitis optica

Neuromyelitis optica (NMO) is a monophasic or relapsing-remitting demyelinating CNS disorder Although previously thought to be a variant of multiple sclerosis, it is now recognised to be a distinct disease, particularly prevalent in Asian populations. It typically involves the optic nerves and cervical spine, with imaging of the brain frequently normal. Vomiting is also a common presenting complaint.

Diagnosis requires bilateral optic neuritis, myelitis and 2 of the following 3 criteria:
- 1. Spinal cord lesion involving 3 or more spinal levels
- 2. Initially normal MRI brain
- 3. Aquaporin 4 positive serum antibody

Management
- immunosuppressant e.g. with anti-CD20 agent rituximab)

+ Q 123

Next question >

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Score: **24.1%**

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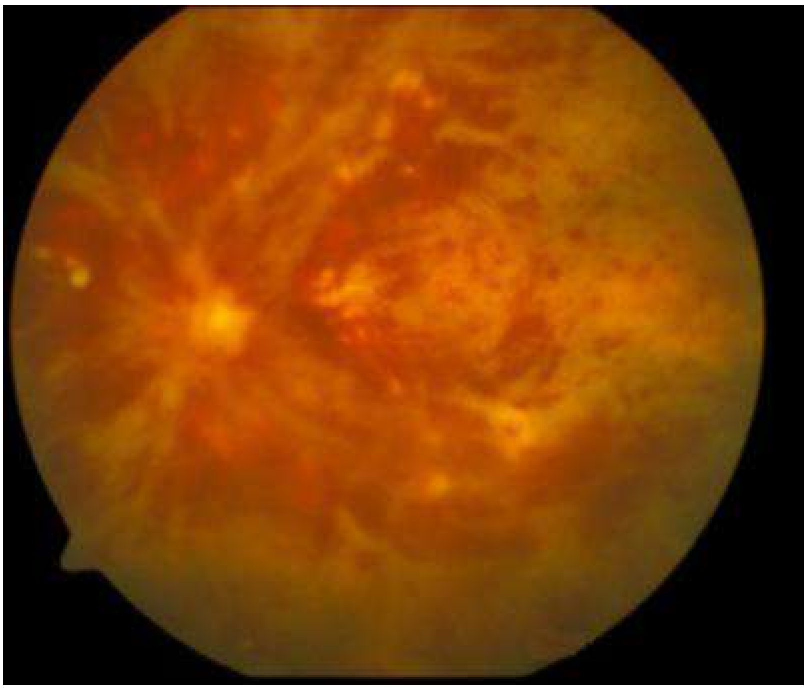
A 62-year-old woman presents with sudden loss of vision in her left eye. Fundoscopy reveals the following:



What is the diagnosis?

- ○ Retinal detachment
- ○ Ischaemic optic neuropathy
- ○ Vitreous haemorrhage
- ○ Central retinal vein occlusion
- ○ Central retinal artery occlusion

Submit answer

Reference ranges

Score: **0%**

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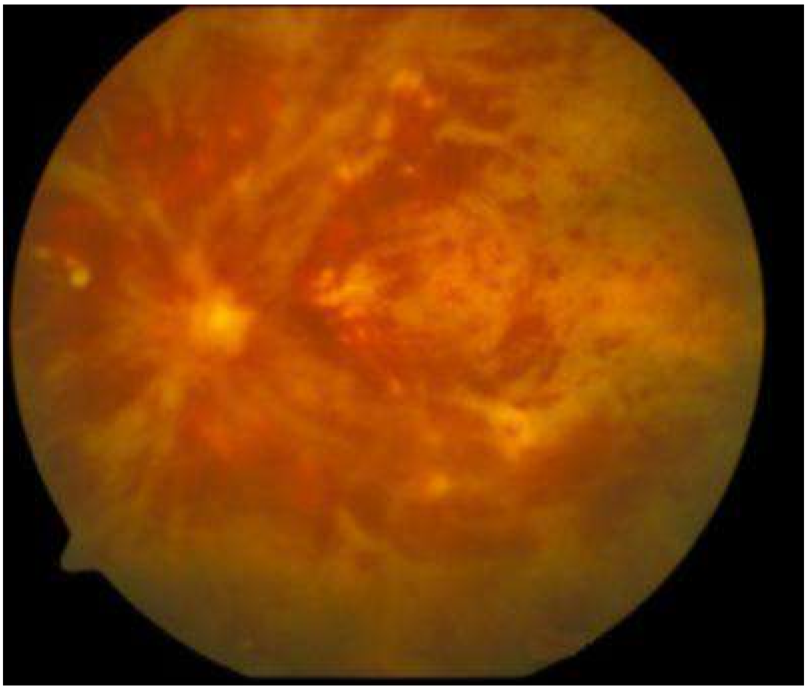


What is the diagnosis?

Retinal detachment	3%
Ischaemic optic neuropathy	3%
Vitreous haemorrhage	26%
Central retinal vein occlusion	64%
Central retinal artery occlusion	5%

Central retinal vein occlusion - sudden painless loss of vision, severe retinal haemorrhages on fundoscopy

Important for me Less important

This appearance is sometimes compared to a cheese and tomato pizza.

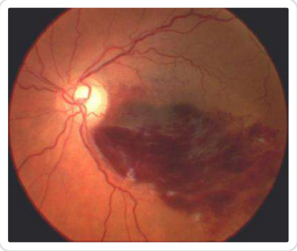
Central retinal vein occlusion ★

Central retinal vein occlusion (CRVO) is a differential for sudden painless loss of vision.

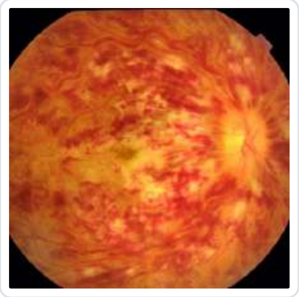
Risk factors
- increasing age
- hypertension
- cardiovascular disease
- glaucoma
- polycythaemia

Features
- sudden, painless reduction or loss of visual acuity, usually unilaterally
- fundoscopy
    - widespread hyperaemia
    - severe retinal haemorrhages - 'stormy sunset'

A key differential is branch retinal vein occlusion (BRVO) - this occurs when a vein in the distal retinal venous system is occluded and is thought to occur due to blockage of retinal veins at arteriovenous crossings. It results in a more limited area of the fundus being affected.



Management
- the majority of patients are managed conservatively
- indications for treatment in patients with CRVO include:
    - macular oedema - intravitreal anti-vascular endothelial growth factor (VEGF) agents
    - retinal neovascularization - laser photocoagulation



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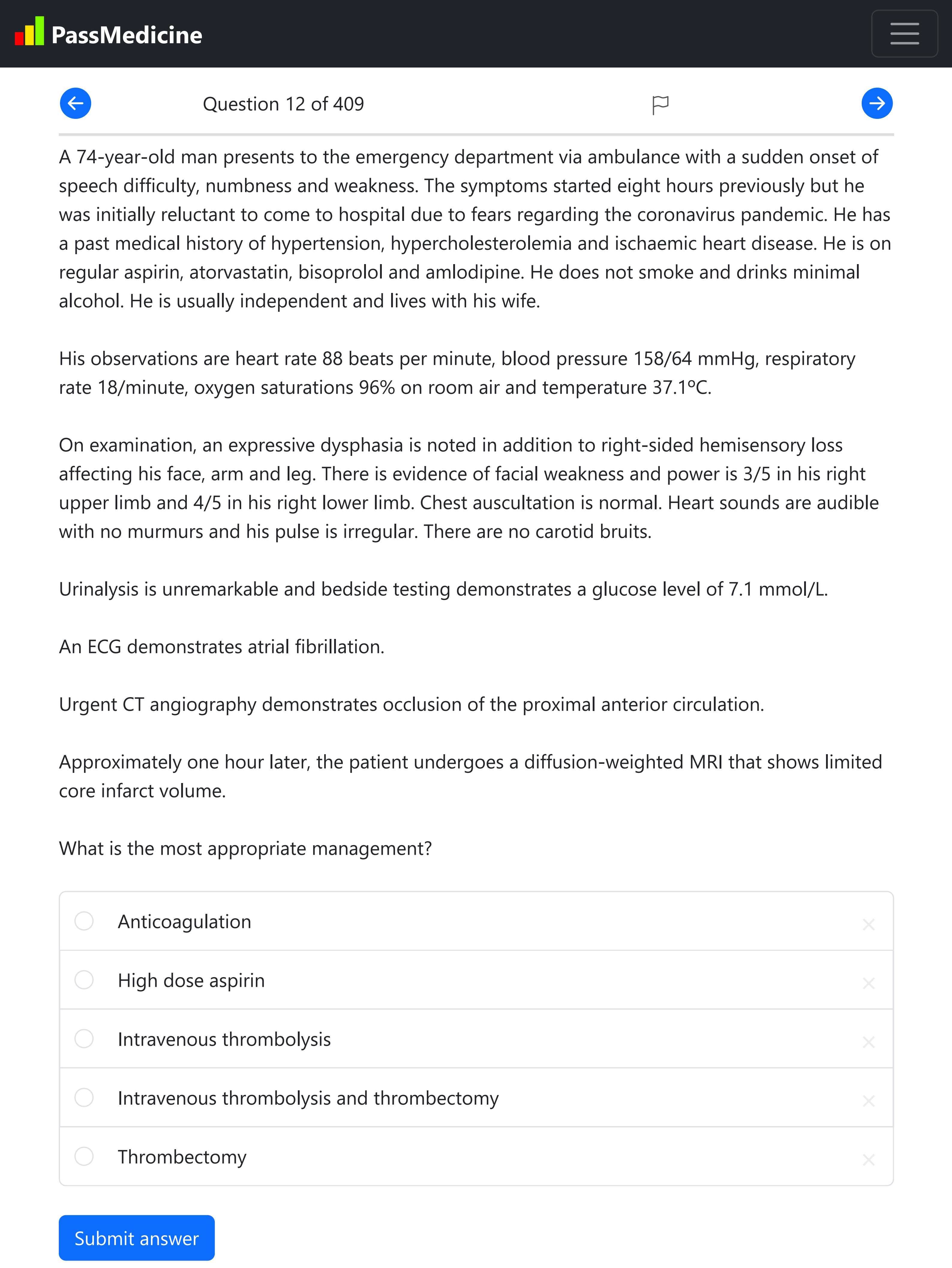

A 74-year-old man presents to the emergency department via ambulance with a sudden onset of speech difficulty, numbness and weakness. The symptoms started eight hours previously but he was initially reluctant to come to hospital due to fears regarding the coronavirus pandemic. He has a past medical history of hypertension, hypercholesterolemia and ischaemic heart disease. He is on regular aspirin, atorvastatin, bisoprolol and amlodipine. He does not smoke and drinks minimal alcohol. He is usually independent and lives with his wife.

His observations are heart rate 88 beats per minute, blood pressure 158/64 mmHg, respiratory rate 18/minute, oxygen saturations 96% on room air and temperature 37.1ºC.

On examination, an expressive dysphasia is noted in addition to right-sided hemisensory loss affecting his face, arm and leg. There is evidence of facial weakness and power is 3/5 in his right upper limb and 4/5 in his right lower limb. Chest auscultation is normal. Heart sounds are audible with no murmurs and his pulse is irregular. There are no carotid bruits.

Urinalysis is unremarkable and bedside testing demonstrates a glucose level of 7.1 mmol/L.

An ECG demonstrates atrial fibrillation.

Urgent CT angiography demonstrates occlusion of the proximal anterior circulation.

Approximately one hour later, the patient undergoes a diffusion-weighted MRI that shows limited core infarct volume.

What is the most appropriate management?

○ Anticoagulation

○ High dose aspirin

○ Intravenous thrombolysis

○ Intravenous thrombolysis and thrombectomy

○ Thrombectomy

Reference ranges

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Urinalysis is unremarkable and bedside testing demonstrates a glucose level of 7.1 mmol/L.

An ECG demonstrates atrial fibrillation.

Urgent CT angiography demonstrates occlusion of the proximal anterior circulation.

Approximately one hour later, the patient undergoes a diffusion-weighted MRI that shows limited core infarct volume.

What is the most appropriate management?

Anticoagulation	2%
High dose aspirin	9%
Intravenous thrombolysis	4%
Intravenous thrombolysis and thrombectomy	15%
Thrombectomy	69%

For thrombectomy in acute ischaemic stroke, an extended target time of 6-24 hours may be considered if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Important for me Less important

Anticoagulation is incorrect. This patient has had an ischaemic stroke likely secondary to new atrial fibrillation and will need anticoagulation in due course. However, anticoagulation is not a treatment for acute stroke and is usually delayed for two weeks after the initial event.

High dose aspirin is incorrect. This patient is outside the typical thrombolysis window of 4.5 hours but is already established on aspirin treatment and there is another more suitable option in this circumstance i.e. thrombectomy.

Intravenous thrombolysis is incorrect. This treatment needs to be administered with 4.5 hours of the onset of an ischaemic stroke. We are outside the thrombolysis window and therefore this is not an appropriate option.

Intravenous thrombolysis and thrombectomy is not the right answer. The combination of thrombectomy and intravenous thrombolysis is still time-limited by the thrombolysis window of 4.5 hours. The decision to offer thrombectomy alone is dependent on different criteria. This patient is outside the thrombolysis window.

Thrombectomy is the correct answer. An extended target time of 6-24 hours for thrombectomy alone may be considered if there is confirmed proximal occlusion of the anterior circulation by CT angiography and the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume. Our patient underwent a diffusion-weighted MRI demonstrating limited infarct core volume within 9 hours of symptom onset and has a confirmed proximal anterior circulation occlusion and therefore is an eligible candidate.

👍 👎 Discuss (8) Improve

Next question >

Stroke: management of acute ischaemic stroke ★

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
    - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for

    thrombolysis (see below)
    - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:

- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.

- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
    - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
    - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and

- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 11 👎 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 16

Stroke and TIA guidelines

Age and Ageing 👍 21 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

2023 National Clinical Guideline for Stroke

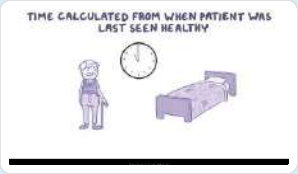
NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link

Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia
Acute ischaemic Stroke

GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1

Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

Khan Academy - YouTube 👍 1 👎 1

Report broken media

Score: **24.1%**

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A previously well 69 year old patient presents to the A+E department with a sudden onset of weakness. This was noticed by his wife who immediately called 999.

Neurological examination:

CN I-IV normal
CN V ophthalmic and maxillary divisions normal with reduced sensation in mandibular division on left side
CN VI normal
CN VII reduced power to left lower facial musculature
CN VIII normal
CN IX, X and XII weakness in swallow
CN XI weakness on turning the head to the left.

Right upper limb normal tone, power 5/5, normal reflexes and sensation. Normal finger pointing.
Left upper limb markedly increased tone, power 1/5 globally in all muscle groups, brisk reflexes and reduced sensation to light touch. Unable to move limb to determine ability to finger pointing.

Right lower limb normal tone, power 5/5, normal reflexes and sensation. Heel-knee-shin test normal.
Left lower limb slightly increased tone, power 3/5 globally, brisk reflexes and reduced sensations. Reduced ability to heel-knee-shin test when compared to right side.

Gait - not assessed due to weakness.
Romberg's test - not done due to weakness.

Where is the lesion most likely to be?

○ Left middle cerebral artery
○ Right middle cerebral artery
○ Posterior cerebral artery
○ Left anterior cerebral artery
○ Right anterior cerebral artery

Submit answer

Reference ranges ⌵

Score: **0%**

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Gait - not assessed due to weakness.
Romberg's test - not done due to weakness.

Where is the lesion most likely to be?

Left middle cerebral artery	7%
Right middle cerebral artery	54%
Posterior cerebral artery	20%
Left anterior cerebral artery	4%
Right anterior cerebral artery	15%

Contralateral hemiparesis and sensory loss with the upper extremity being more affected than the lower, contralateral homonymous hemianopia and aphasia - middle cerebral artery

Important for me Less important

The understanding of stroke territories is important both in clinical practice but also in the MRCP! In terms of limb weakness it must be remembered that an ischaemic event in the anterior cerebral territory caused contralateral hemiparesis (leg>arm) and the middle cerebral territory causes contralateral hemiparesis (face/arm>leg).

A posterior cerebral artery lesion causes contralateral homonymous hemianopia with amnesia and sensory loss.

Discuss (13) Improve

Next question

Stroke by anatomy

Site of the lesion	Associated effects
Anterior cerebral artery	Contralateral hemiparesis and sensory loss, lower extremity > upper
Middle cerebral artery	Contralateral hemiparesis and sensory loss, upper extremity > lower Contralateral homonymous hemianopia Aphasia
Posterior cerebral artery	Contralateral homonymous hemianopia with macular sparing Visual agnosia
Weber's syndrome (branches of the posterior cerebral artery that supply the midbrain)	Ipsilateral CN III palsy Contralateral weakness of upper and lower extremity
Posterior inferior cerebellar artery (lateral medullary syndrome, Wallenberg syndrome)	Ipsilateral: facial pain and temperature loss Contralateral: limb/torso pain and temperature loss Ataxia, nystagmus

Site of the lesion	Associated effects
Anterior inferior cerebellar artery (lateral pontine syndrome)	Symptoms are similar to Wallenberg's (see above), but: Ipsilateral: facial paralysis and deafness
Retinal/ophthalmic artery	Amaurosis fugax
Basilar artery	'Locked-in' syndrome

Lacunar strokes

- present with either isolated hemiparesis, hemisensory loss or hemiparesis with limb ataxia
- strong association with hypertension
- common sites include the basal ganglia, thalamus and internal capsule

123

Next question

Textbooks

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Links

Life in the Fast Lane 75 10

Brainstem Rules of 4

Suggest link Report broken link

Media

Management of ischaemic stroke

[Stroke](#)

Podmedics - YouTube 👍 0 👎 0

Brainstem Stroke

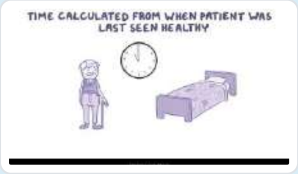
[Brainstem stroke syndromes](#)

Soton Brain Hub - YouTube 👍 13 👎 1

NEUROLOGY
MIDBRAIN SYNDROMES

[Midbrain Syndromes - Weberâ€™s Syndrome, Benediktâ€™s Syndrome and Parinaud Syndrome](#)

Armando Hasudungan - YouTube 👍 2 👎 1


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

[Stroke](#)

Osmosis - YouTube 👍 1 👎 1

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Score: **24.1%**

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A 56-year-old man presents to the neurology clinic with a 6-month history of loss of dexterity and new-onset dysphagia. The patient reports a history of hypertension for which he takes amlodipine 5mg once daily with no family history of note, he is a non-smoker and drinks 3 glasses of wine per week. Systems review reveals no history of rashes, joint pain, swelling or stiffness, no fevers, and no weight loss. A recent endoscopy showed no obvious abnormalities to explain the patient's dysphagia.

Physical examination demonstrates asymmetrical wasting of the deltoids with noticeable weakness of wrist flexion bilaterally (MRC grading 4/5).

Co-ordination is normal on the assessment of the upper and lower limbs, which decreased ankle jerk relaxes bilaterally. Plantars are downgoing bilaterally. Cranial nerve assessment is unremarkable, however, a bedside swallow assessment is suggestive of a delayed oropharyngeal phase.

Bloods tests ordered by the neurologist demonstrate:

Hb 140 g/L	Male: (135-180) Female: (115 - 160)
Platelets 220 * 10^9/L	(150 - 400)
WBC 5.0 * 10^9/L	(4.0 - 11.0)

Calcium 2.26 mmol/L	(2.1-2.6)
Phosphate 1.04 mmol/L	(0.8-1.4)
Magnesium 0.9 mmol/L	(0.7-1.0)
Thyroid stimulating hormone (TSH) 0.6 mU/L	(0.5-5.5)
Free thyroxine (T4) 10.1 pmol/L	(9.0 - 18)
Creatine kinase 1000 U/L	(35 - 250)

What is the most likely diagnosis?

○ Inclusion body myositis ×

○ Dermatomyositis ×

○ Motor neuron disease ×

○ Multiple sclerosis ×

○ Myasthenia gravis ×

Submit answer

Reference ranges ⌄

Score: **0%**

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Question 14 of 409

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Creatine kinase 1000 U/L	(35 - 250)

What is the most likely diagnosis?

Inclusion body myositis	65%
Dermatomyositis	6%
Motor neuron disease	25%
Multiple sclerosis	1%

Myasthenia gravis 3%

Older man + quadriceps and finger/wrist flexors weakness + raised CK → inclusion body myositis

Important for me Less important

Inclusion body myositis is characterized by frequently asymmetrical muscle weakness, with a mixed proximal and distal distribution of weakness. Dysphagia affects approximately 9% of patients at presentation.

Dermatomyositis usually features symmetrical muscle weakness with a characteristic rash. This patient has neither.

Motor neuron disease presents with upper motor neuron signs, with hyperreflexia and extensor plantars. This patient has neither.

Multiple sclerosis could possibly explain the patient's symptoms, however, it would be very unlikely in a male patient of late middle age.

Myasthenia gravis usually presents with symmetrical muscle weakness which is fatiguable. This patient has an asymmetrical weakness with no mention of fatiguability.

Discuss (6) Improve

Next question

Inclusion body myositis ★

Overview

- cause of myopathy
- associated with cytoplasmic inclusions on muscle biopsy

Features

- typically affects older males
- can affect both proximal and distal muscles
- characteristically affects quadriceps and finger/wrist flexors are usually more severely affected than extensor counterparts

123

Next question

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Media

INCLUSION BODY MYOPATHY

Inclusion body myositis

Osmosis - YouTube 👍 7 👎 2

Report broken media

Score: **24.1%**

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Question 15 of 409

A 24-year-old male presents to the acute medical unit with confusion. His mother states that over the past day he has been behaving very strangely. His past medical history includes epilepsy, asthma, and depression. His drug history includes salbutamol, sertraline, and sodium valproate. On examination, you note myoclonus and his Glasgow coma score is reduced at 13 (M6, V4, E3).

Blood results are as follows:

Hb	132 g/l	Na^+	142 mmol/l	Bilirubin	18 µmol/l
Platelets	328 * 10^9/l	K^+	4.1 mmol/l	ALP	72 u/l
WBC	8.7 * 10^9/l	Urea	6.8 mmol/l	ALT	184 u/l
Neuts	7.4 * 10^9/l	Creatinine	72 µmol/l	INR	1.1
Lymphs	1.2 * 10^9/l	Albumin	42 g/l	Plasma ammonia	72 µmol/L (normal range 11-32 µmol/L)

What is the most likely cause of this presentation?

○ Hepatic encephalopathy

○ Sertraline

○ Small intestinal bacterial overgrowth

○ Sodium valproate

○ Intracerebral hemorrhage

Submit answer

Reference ranges

Score: **0%**

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Lymphs	1.2 * 10^9/l	Albumin	42 g/l	Plasma ammonia	72 µmol/L (normal range 11-32 µmol/L)

What is the most likely cause of this presentation?

Hepatic encephalopathy	23%
Sertraline	12%
Small intestinal bacterial overgrowth	4%
Sodium valproate	61%
Intracerebral hemorrhage	1%

Sodium valproate is a well known cause of hyperammonemic encephalopathy

Important for me Less important

The patient has features of encephalopathy and raised serum ammonia levels. Valproate-induced hyperammonaemic encephalopathy (VHE) is an unusual complication characterised by a decreasing level of consciousness, focal neurological deficits, cognitive slowing, vomiting, drowsiness, and lethargy. Asymptomatic hyperammonaemia occurs in 15-50% of patients on valproate.

Treatment with L-carnitine may be beneficial in reducing ammonia levels.

Although the ALT is raised, this is a fairly common finding and may be entirely explained as a side effect of valproate therapy. The true liver function is normal as demonstrated by normal albumin, INR, and bilirubin. Therefore hepatic encephalopathy is an incorrect answer.

Small intestinal bacterial overgrowth can lead to acquired hyperammonaemia, however, there is nothing to suggest that this is the diagnosis in this case.

Discuss (7) | Improve

Next question

Sodium valproate

Sodium valproate is used in the management of epilepsy and is first-line therapy for generalised seizures. It works by increasing GABA activity.

Adverse effects
- teratogenic
    - neural tube defects
    - maternal use of sodium valproate is associated with a significant risk of neurodevelopmental delay in children
    - guidance is now clear that sodium valproate should not be used during pregnancy and in women of childbearing age unless clearly necessary. Women of childbearing age should not start treatment without specialist neurological or psychiatric advice.
- P450 inhibitor
- gastrointestinal: nausea
- increased appetite and weight gain
- alopecia: regrowth may be curly
- ataxia
- tremor
- hepatotoxicity
- pancreatitis
- thrombocytopaenia
- hyponatraemia
- hyperammonemic encephalopathy: L-carnitine may be used as treatment if this develops

AI + Q 123

Next question

Textbooks

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Score: **24.1%**

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A 48-year-old man is reviewed in psychiatry clinic after a referral from his general practitioner with eccentric behaviour. He has no past medical history and does not take any regular medications. There is no history of drug or alcohol abuse.

On examination, he appeared unkempt and dressed exclusively in black clothing. He was accompanied by his mother.

During the course of the interview, it transpires that others have always felt he was 'odd' and he has difficulty forming social relationships. He becomes anxious in social situations. He is obsessed with ghosts and has sought to purchase ghost-hunting equipment. He occasionally feels the spirits of ghosts in his home as a cold breeze.

His affect is inappropriate throughout, avoiding eye contact while laughing at unusual points in conversation.

What is the correct description of this type of personality disorder?

○ Antisocial

○ Avoidant

○ Borderline

○ Schizoid

○ Schizotypal

Submit answer

Reference ranges

Score: **0%**

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His affect is inappropriate throughout, avoiding eye contact while laughing at unusual points in conversation.

What is the correct description of this type of personality disorder?

Antisocial	6%
Avoidant	8%
Borderline	4%
Schizoid	32%
Schizotypal	50%

Schizotypal personality disorder is characterised by social isolation and odd beliefs

Important for me Less important

Schizotypal is the correct answer. This personality disorder is characterized by social isolation, odd or eccentric behaviour, inappropriate affect, unusual perceptual disturbances (the cold breeze in this scenario) and odd beliefs and magical thinking (ghost-hunting in this scenario). This patient demonstrates most of these characteristics.

Avoidant is incorrect. While this personality disorder can also result in social isolation, it is characterized by a craving for others that is absent in this context. Most of the avoidant behaviours in this disorder are as a consequence of fears of embarrassment or ridicule.

Borderline is incorrect. This is characterized by fear of abandonment, unstable interpersonal relationships, impulsivity and chronic feelings of emptiness. Patients often demonstrate recurrent suicidal behaviour.

Schizoid is incorrect. While there is also a preference for solitude in this condition and few friends or confidants other than family, abnormal perceptions and magical thinking are absent. Coldness is predominant rather than eccentricity.

Antisocial is incorrect. The key characteristics of this personality disorder are repeated failure to respect lawful behaviours, deception, irritability and aggressiveness, which are not mentioned in this case.

👍 | 👎 | 💬 Discuss (1) | Improve

Next question >

Personality disorders ★

Personality disorders (PD) are a group of mental health conditions characterised by persistent patterns of thinking, feeling, and behaving that are significantly different from cultural expectations. These patterns can lead to distress or problems in personal, social, and occupational functioning. It is thought that around 1 in 20 people have a personality disorder.

For many years patients were diagnosed with a specific 'named' personality disorder, such as narcissistic or paranoid. This approach was changed significantly with the introduction of the ICD-11 classification which marked a shift towards a more dimensional model, such as severity and specific problems. This allows for a more flexible and comprehensive understanding of personality pathology.

The majority of patients you'll encounter with personality disorders will be 'labelled' using the previous classification system. We've therefore included details of this classification at the end of the notes.

ICD-11 classification of personality disorders

Key Features of Personality Disorder (ICD-11)

- Persistent Pattern: The individual's patterns of cognition, emotional experience, behaviour, and interpersonal functioning deviate from cultural expectations. These patterns are stable over time and span across various personal and social situations.
- Impairment: The deviation results in significant problems or dysfunctions in the person’s life, especially in relationships, work, or social functioning.

- Duration: These characteristics are stable over time, beginning in adolescence or early adulthood, and are not transient.
- Distress or Dysfunction: The impairment may result in distress to the individual or others. These patterns are not explained by another mental disorder, a medical condition, or substance misuse.

Severity Classification in ICD-11

In the ICD-11, personality disorders are classified based on the degree of severity. This provides a broader and more flexible approach:

Mild Personality Disorder:
- Some impairments in functioning, often limited to specific areas of life (e.g., intimate relationships or work).
- Symptoms may be noticeable to others but do not cause pervasive distress or dysfunction.
- The individual may still maintain relatively stable relationships and occupational roles.

Moderate Personality Disorder:
- More significant impairments in multiple areas of life (personal, social, and work).
- The individual may struggle with maintaining close relationships, and there may be greater interpersonal difficulties.
- More distressing symptoms are present, but the individual can manage day-to-day functioning with some effort or support.

Severe Personality Disorder:
- Profound impairments in all areas of life.
- Individuals may experience pervasive difficulties in interpersonal relationships, self-identity, and coping mechanisms.
- Significant distress, dysfunction, and reduced quality of life are common.
- Individuals may require intensive and long-term therapeutic interventions.

Personality Disorder Trait Domains:

In addition to severity, ICD-11 allows clinicians to specify trait domains that represent the predominant problematic areas in the individual's personality. These are:
- Negative Affectivity:
  - Tendency to experience a wide range of negative emotions such as anxiety, depression, guilt, and anger.
  - Individuals may be prone to mood swings, insecurity, and emotional lability.
- Detachment:
  - Avoidance of social interactions, emotional withdrawal, and limited pleasure from relationships.
  - Individuals may appear cold, aloof, and isolated.
- Dissociality:

  - Disregard for the rights and feelings of others, lack of empathy, and difficulty forming prosocial relationships.
  - mpulsivity and manipulative behaviours are common traits.
- Disinhibition:
  - Impulsiveness, risk-taking, and difficulty controlling behaviours.
  - Individuals may struggle with planning and foresight, leading to reckless or irresponsible actions.
- Anankastia:
  - Preoccupation with orderliness, control, and perfectionism.
  - Individuals may be rigid, stubborn, and excessively focused on rules and details.
- Borderline Pattern (also recognised in ICD-11):
  - An additional qualifier for those showing emotional instability, intense and unstable interpersonal relationships, a fluctuating sense of identity, and impulsivity.
  - This pattern is akin to borderline personality disorder as recognised in ICD-10 and DSM-5.

Previous classification of personality disorders

They are typically categorised as belonging to one of three clusters:

Cluster A: 'Odd or Eccentric'	**Cluster B: 'Dramatic, Emotional, or Erratic'**	**Cluster C: 'Anxious and Fearful'**
• Paranoid • Schizoid • Schizotypal	• Antisocial • Borderline (Emotionally Unstable) • Histrionic • Narcissistic	• Obsessive-Compulsive • Avoidant • Dependent

Cluster A: 'Odd or Eccentric'

Paranoid
- Hypersensitivity and an unforgiving attitude when insulted
- Unwarranted tendency to questions the loyalty of friends
- Reluctance to confide in others
- Preoccupation with conspirational beliefs and hidden meaning
- Unwarranted tendency to perceive attacks on their character

Schizoid
- Indifference to praise and criticism
- Preference for solitary activities
- Lack of interest in sexual interactions

- Lack of desire for companionship
- Emotional coldness
- Few interests
- Few friends or confidants other than family

Schizotypal

- Ideas of reference (differ from delusions in that some insight is retained)
- Odd beliefs and magical thinking
- Unusual perceptual disturbances
- Paranoid ideation and suspiciousness
- Odd, eccentric behaviour
- Lack of close friends other than family members
- Inappropriate affect
- Odd speech without being incoherent

Cluster B: 'Dramatic, Emotional, or Erratic'

Antisocial

- Failure to conform to social norms with respect to lawful behaviours as indicated by repeatedly performing acts that are grounds for arrest;
- More common in men;
- Deception, as indicated by repeatedly lying, use of aliases, or conning others for personal profit or pleasure;
- Impulsiveness or failure to plan ahead;
- Irritability and aggressiveness, as indicated by repeated physical fights or assaults;
- Reckless disregard for the safety of self or others;
- Consistent irresponsibility, as indicated by repeated failure to sustain consistent work behaviour or honour financial obligations;
- Lack of remorse, as indicated by being indifferent to or rationalizing having hurt, mistreated, or stolen from another

Borderline - also known as Emotionally Unstable

- Efforts to avoid real or imagined abandonment
- Unstable interpersonal relationships which alternate between idealization and devaluation
- Unstable self image
- Impulsivity in potentially self damaging area (e.g. Spending, sex, substance abuse)
- Recurrent suicidal behaviour
- Affective instability
- Chronic feelings of emptiness
- Difficulty controlling temper
- Quasi psychotic thoughts

Histrionic

- Inappropriate sexual seductiveness
- Need to be the centre of attention
- Rapidly shifting and shallow expression of emotions
- Suggestibility
- Physical appearance used for attention seeking purposes
- Impressionistic speech lacking detail
- Self dramatization
- Relationships considered to be more intimate than they are

Narcissistic

- Grandiose sense of self importance
- Preoccupation with fantasies of unlimited success, power, or beauty
- Sense of entitlement
- Taking advantage of others to achieve own needs
- Lack of empathy
- Excessive need for admiration
- Chronic envy
- Arrogant and haughty attitude

Cluster C: 'Anxious and Fearful'

Obsessive-compulsive

- Is occupied with details, rules, lists, order, organization, or agenda to the point that the key part of the activity is gone
- Demonstrates perfectionism that hampers with completing tasks
- Is extremely dedicated to work and efficiency to the elimination of spare time activities
- Is meticulous, scrupulous, and rigid about etiquettes of morality, ethics, or values
- Is not capable of disposing worn out or insignificant things even when they have no sentimental meaning
- Is unwilling to pass on tasks or work with others except if they surrender to exactly their way of doing things
- Takes on a stingy spending style towards self and others; and shows stiffness and stubbornness

Avoidant

- Avoidance of occupational activities which involve significant interpersonal contact due to fears of criticism, or rejection.
- Unwillingness to be involved unless certain of being liked
- Preoccupied with ideas that they are being criticised or rejected in social situations
- Restraint in intimate relationships due to the fear of being ridiculed
- Reluctance to take personal risks due to fears of embarrassment
- Views self as inept and inferior to others
- Social isolation accompanied by a craving for social contact

Dependent

- Difficulty making everyday decisions without excessive reassurance from others
- Need for others to assume responsibility for major areas of their life
- Difficulty in expressing disagreement with others due to fears of losing support
- Lack of initiative
- Unrealistic fears of being left to care for themselves
- Urgent search for another relationship as a source of care and support when a close relationship ends
- Extensive efforts to obtain support from others
- Unrealistic feelings that they cannot care for themselves

Management

Personality disorders are difficult to treat and in the past have been considered 'untreatable' by definition.. However, a number of approaches have been shown to help patients, including:

- psychological therapies: dialectical behaviour therapy
- treatment of any coexisting psychiatric conditions

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Next question >

Textbooks

High-yield textbook

Extended textbook

Media

Personality disorders by clusters

Personality disorders by clusters

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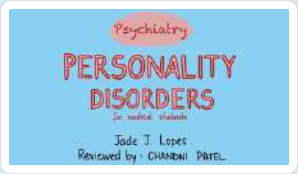
Psychiatry
PERSONALITY DISORDERS
for medical students
Jade J. Lopes
Reviewed by: CHANDNI PATEL

Personality Disorders and DSH

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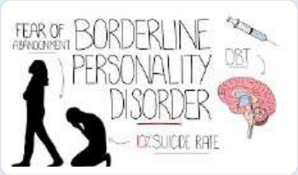
FEAR OF ABANDONMENT
BORDERLINE PERSONALITY DISORDER
DBT
10% SUICIDE RATE

Borderline Personality Disorder Explained

Rhesus Medicine - YouTube 👍 1 👎 0

Cluster C personality disorders (avoidant, obsessive-compulsive, dependent)

Osmosis - YouTube 👍 0 👎 0

Cluster A personality disorders (paranoid, schizoid, schizotypal)

Osmosis - YouTube 👍 1 👎 1

Cluster B personality disorders (antisocial, borderline, histrionic, narcissistic)

Osmosis - YouTube 👍 1 👎 1

Report broken media

Score: **24.1%**

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Question 17 of 409

A 16-year-old female is brought to the general practitioner by her mother, who is concerned about the difficulties her daughter has in forming successful friendships and personal relationships. The patient herself denies this is a problem and reports that she has many 'best friends'.

During the course of the consultation, the GP becomes aware that she is inappropriately sexually seductive and seems to use her physical appearance to seek attention. In addition, she tends to dramatize events and stories and always places herself as the central protagonist. Many of her stories seem to involve emotional overreaction her part.

What is the most accurate description of this personality disorder?

- ○ Antisocial
- ○ Borderline
- ○ Dependent
- ○ Histrionic
- ○ Narcissistic

Submit answer

Reference ranges

Score: **0%**

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What is the most accurate description of this personality disorder?

Antisocial	1%
Borderline	10%
Dependent	2%
Histrionic	72%
Narcissistic	15%

Histrionic personality disorder is characterised by excessive attention-seeking and emotional overreaction

Important for me Less important

A histrionic personality disorder is correct. This personality disorder is characterized by inappropriate sexual seductiveness, self-dramatization, attention-seeking behaviour and emotional overreaction. Additionally, relationships are considered to be more intimate than they are in reality., In this case, the patient considered herself to have many 'best friends', while her mother disagrees with that assessment.

An antisocial personality disorder is incorrect. This personality disorder is characterized by repeatedly breaking the law, deception, irresponsibility and a lack of remorse.

A borderline personality disorder is incorrect. This personality disorder is characterized by an unstable self-image and efforts to avoid abandonment. Impulsivity is common, along with difficulties in controlling temper and recurrent suicidal behaviour.

A dependent personality disorder is incorrect. People with this personality disorder have difficulty making everyday decisions without excessive reassurance from others and have unrealistic fears about being left to care for themselves. They often commence an urgent search for another relationship as a source of care when a close relationship ends.

A narcissistic personality disorder is incorrect. This personality disorder is characterized by a grandiose sense of self-importance and a sense of entitlement. People who exhibit these features often take advantage of others to achieve their own needs and lack empathy.

👍 👎 Discuss (4) Improve

Next question >

Personality disorders ★

Personality disorders (PD) are a group of mental health conditions characterised by persistent patterns of thinking, feeling, and behaving that are significantly different from cultural expectations. These patterns can lead to distress or problems in personal, social, and occupational functioning. It is thought that around 1 in 20 people have a personality disorder.

For many years patients were diagnosed with a specific 'named' personality disorder, such as narcissistic or paranoid. This approach was changed significantly with the introduction of the ICD-11 classification which marked a shift towards a more dimensional model, such as severity and specific problems. This allows for a more flexible and comprehensive understanding of personality pathology.

The majority of patients you'll encounter with personality disorders will be 'labelled' using the previous classification system. We've therefore included details of this classification at the end of the notes.

ICD-11 classification of personality disorders

Key Features of Personality Disorder (ICD-11)

- Persistent Pattern: The individual's patterns of cognition, emotional experience, behaviour, and interpersonal functioning deviate from cultural expectations. These patterns are stable over time and span across various personal and social situations.
- Impairment: The deviation results in significant problems or dysfunctions in the person’s life, especially in relationships, work, or social functioning.
- Duration: These characteristics are stable over time, beginning in adolescence or early adulthood, and are not transient.

- Distress or Dysfunction: The impairment may result in distress to the individual or others. These patterns are not explained by another mental disorder, a medical condition, or substance misuse.

Severity Classification in ICD-11

In the ICD-11, personality disorders are classified based on the degree of severity. This provides a broader and more flexible approach:

Mild Personality Disorder:
- Some impairments in functioning, often limited to specific areas of life (e.g., intimate relationships or work).
- Symptoms may be noticeable to others but do not cause pervasive distress or dysfunction.
- The individual may still maintain relatively stable relationships and occupational roles.

Moderate Personality Disorder:
- More significant impairments in multiple areas of life (personal, social, and work).
- The individual may struggle with maintaining close relationships, and there may be greater interpersonal difficulties.
- More distressing symptoms are present, but the individual can manage day-to-day functioning with some effort or support.

Severe Personality Disorder:
- Profound impairments in all areas of life.
- Individuals may experience pervasive difficulties in interpersonal relationships, self-identity, and coping mechanisms.
- Significant distress, dysfunction, and reduced quality of life are common.
- Individuals may require intensive and long-term therapeutic interventions.

Personality Disorder Trait Domains:

In addition to severity, ICD-11 allows clinicians to specify trait domains that represent the predominant problematic areas in the individual's personality. These are:
- Negative Affectivity:
  - Tendency to experience a wide range of negative emotions such as anxiety, depression, guilt, and anger.
  - Individuals may be prone to mood swings, insecurity, and emotional lability.
- Detachment:
  - Avoidance of social interactions, emotional withdrawal, and limited pleasure from relationships.
  - Individuals may appear cold, aloof, and isolated.
- Dissociality:
  - Disregard for the rights and feelings of others, lack of empathy, and difficulty forming prosocial relationships.

    - mpulsivity and manipulative behaviours are common traits.
- Disinhibition:
    - Impulsiveness, risk-taking, and difficulty controlling behaviours.
    - Individuals may struggle with planning and foresight, leading to reckless or irresponsible actions.
- Anankastia:
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They are typically categorised as belonging to one of three clusters:

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Cluster A: 'Odd or Eccentric'

Paranoid
- Hypersensitivity and an unforgiving attitude when insulted
- Unwarranted tendency to questions the loyalty of friends
- Reluctance to confide in others
- Preoccupation with conspirational beliefs and hidden meaning
- Unwarranted tendency to perceive attacks on their character

Schizoid
- Indifference to praise and criticism
- Preference for solitary activities
- Lack of interest in sexual interactions
- Lack of desire for companionship
- Emotional coldness

- Few interests
- Few friends or confidants other than family

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- Odd beliefs and magical thinking
- Unusual perceptual disturbances
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Cluster B: 'Dramatic, Emotional, or Erratic'

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- Failure to conform to social norms with respect to lawful behaviours as indicated by repeatedly performing acts that are grounds for arrest;
- More common in men;
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- Efforts to avoid real or imagined abandonment
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- Inappropriate sexual seductiveness
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- Preoccupation with fantasies of unlimited success, power, or beauty
- Sense of entitlement
- Taking advantage of others to achieve own needs
- Lack of empathy
- Excessive need for admiration
- Chronic envy
- Arrogant and haughty attitude

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Obsessive-compulsive

- Is occupied with details, rules, lists, order, organization, or agenda to the point that the key part of the activity is gone
- Demonstrates perfectionism that hampers with completing tasks
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- Avoidance of occupational activities which involve significant interpersonal contact due to fears of criticism, or rejection.
- Unwillingness to be involved unless certain of being liked
- Preoccupied with ideas that they are being criticised or rejected in social situations
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Dependent

- Difficulty making everyday decisions without excessive reassurance from others
- Need for others to assume responsibility for major areas of their life
- Difficulty in expressing disagreement with others due to fears of losing support
- Lack of initiative
- Unrealistic fears of being left to care for themselves
- Urgent search for another relationship as a source of care and support when a close relationship ends
- Extensive efforts to obtain support from others
- Unrealistic feelings that they cannot care for themselves

Management

Personality disorders are difficult to treat and in the past have been considered 'untreatable' by definition.. However, a number of approaches have been shown to help patients, including:

- psychological therapies: dialectical behaviour therapy
- treatment of any coexisting psychiatric conditions

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Personality disorders by clusters

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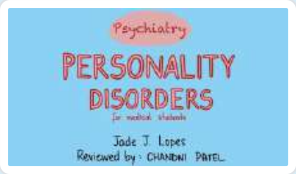
Psychiatry
PERSONALITY DISORDERS
for medical students
Jade J. Lopes
Reviewed by: CHANDNI PATEL

Personality Disorders and DSH

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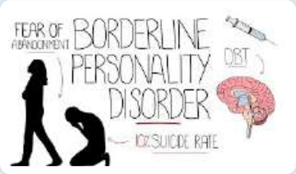
FEAR OF ABANDONMENT
BORDERLINE PERSONALITY DISORDER
DBT
10% SUICIDE RATE

Borderline Personality Disorder Explained

Rhesus Medicine - YouTube 👍 1 👎 0

Cluster C personality disorders (avoidant, obsessive-compulsive, dependent)

Osmosis - YouTube 👍 0 👎 0

Cluster A personality disorders (paranoid, schizoid, schizotypal)

Osmosis - YouTube 👍 1 👎 1

Cluster B personality disorders (antisocial, borderline, histrionic, narcissistic)

Osmosis - YouTube 👍 1 👎 1

Report broken media

Score: **24.1%**

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Question 18 of 409

A 25-year-old woman is reviewed in the neurology outpatient clinic. She has a past medical history of idiopathic intracranial hypertension and she reports a recurrence of headaches. She takes regular acetazolamide.

On examination, fundoscopy reveals papilloedema. Her body mass index is 31 kg/m². Her visual acuity is normal.

A decision is made to perform a therapeutic lumbar puncture in the left lateral position.

The opening pressure is 29 €‰25 cmCSF and 10ml of cerebrospinal fluid is removed.

The patient subsequently develops a headache.

What intervention could have reduced the likelihood of developing this complication?

○ Bed rest following procedure ×

○ Decreased quantity of CSF removal ×

○ Increased fluid intake post procedure ×

○ Performing the procedure in a sitting up position ×

○ Re-insertion of the stylet prior to removing the needle ×

Submit answer

Reference ranges

Score: **0%**

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A decision is made to perform a therapeutic lumbar puncture in the left lateral position.

The opening pressure is 29 €‰25 cmCSF and 10ml of cerebrospinal fluid is removed.

The patient subsequently develops a headache.

What intervention could have reduced the likelihood of developing this complication?

Bed rest following procedure	25%
Decreased quantity of CSF removal	7%
Increased fluid intake post procedure	15%
Performing the procedure in a sitting up position	4%
Re-insertion of the stylet prior to removing the needle	50%

Lumbar puncture: Re-insertion of the stylet prior to removing the needle reduces the incidence of post-LP headaches

Important for me Less important

Re-insertion of the stylet prior to removing the needle is the correct answer. This is an intervention that has been shown to decrease the incidence of post LP headaches.

Bed rest following procedure is not the correct answer. This has not been shown to decrease the incidence of post-LP headaches.

Decreased quantity of CSF removal is incorrect. This patient has had a standard amount of CSF removed (10ml). It has not been shown that varying the amount of CSF removed has any effect on the incidence of post LP headaches.

Increased fluid intake post-procedure is incorrect. This has not been shown to reduce the incidence of post-LP headaches.

Performing the procedure in a sitting up position is incorrect. This has now been shown to reduce the incidence of post-LP headaches. Additionally, we cannot measure an opening pressure in this position so it would be unsuitable in this context.

👍 👎 Discuss (2) Improve

Next question >

Post-lumbar puncture headache ★

Headache following lumbar puncture (LP) occurs in approximately one-third of patients. The pathophysiology of is unclear but may relate to a 'leak' of CSF following dural puncture. Post-LP headaches are more common in young females with a low body mass index

Typical features
- usually develops within 24-48 hours following LP but may occur up to one week later
- may last several days
- worsens with upright position
- improves with recumbent position

Factors which may contribute to headache	Factors which do not contribute to headache
• Needle type: conventional 'cutting' needles increase the risk compared to pencil point (atraumatic) needles • Increased needle size • Direction of bevel • Not replacing the stylet • Increased number of LP attempts	• Increased volume of CSF removed • Bed rest following procedure • Increased fluid intake post procedure • Opening pressure of CSF • Position of patient

Management
- supportive initially (analgesia, rest)
- if pain continues for more than 72 hours then specific treatment is indicated, to prevent subdural haematoma
- treatment options include: blood patch, epidural saline and intravenous caffeine

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **24.1%**

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Question 19 of 409

You have been referred a 45-year-old man by the Accident and Emergency doctors with a severe headache. The headache woke him up at 3am, and he describes it as the worst headache he's ever had. He has had two episodes like this over the past three days that have followed a very similar pattern, each lasting around 70 minutes before going. The pain is mainly around the left eye and temple and is sharp in nature. On examining him you note that his left eye is watering and swollen, and there's some redness and mild bruising just above the eye. When you ask him about this bruising he says that the pain was so bad he bashed his head against the fridge door to try and help take it away.

How would you treat his headache?

- ○ Oral triptan
- ○ Indometacin
- ○ 100% oxygen and indometacin
- ○ 100% oxygen
- ○ 100% oxygen and nasal triptan

Submit answer

Reference ranges

Score: **0%**

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Question 19 of 409 ✓

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How would you treat his headache?

Oral triptan	2%
Indometacin	6%
100% oxygen and indometacin	14%
100% oxygen	33%
100% oxygen and nasal triptan	46%

Cluster headache - acute treatment: subcutaneous sumatriptan + 100% O2

Important for me Less important

What is described above is the classical cluster headache. NICE guidelines characterise cluster headaches as one-sided headache in or around the eye or temporal region associated with signs of autonomic dysfunction on the same side.

Attacks of pain:

- Usually last for 15-180 minutes
- Almost always described as the most severe pain known
- Tend to recur at the same time each day
- Often wake the person shortly after falling asleep

Signs of symptoms of autonomic dysfunction include:

- Rhinorrhoea or nasal congestion
- Red eye and/or lacrimation

- Facial or forehead sweating or flushing
- Constriction of the pupil and/or ptosis
- Eyelid oedema
- A sense of aural fullness

The suggested first line treatment for cluster headaches is 100% oxygen at a flow rate of at least 12 litres per minute and a subcutaneous or nasal triptan. Evidence from one small study suggests that subcutaneous sumatriptan provides faster relief of symptoms than intranasal sumatriptan.

Indometacin is used in paroxysmal hemicrania which has similar characteristics to cluster headaches, but attacks tend to be shorter (2-45 minutes) and more frequent (up to 40/day) and more common in women.

Oral triptans have no evidence base in cluster headaches.

👍 👎 Discuss (15) Improve

Next question >

Cluster headache ★

Cluster headaches are known to be one of the most painful conditions that patients can have the misfortune to suffer. The name relates to the pattern of the headaches - they typically occur in clusters lasting several weeks, with the clusters themselves typically once a year.

Cluster headaches are more common in men (3:1) and smokers. Alcohol may trigger an attack and there also appears to be a relation to nocturnal sleep.

Features
-intense sharp, stabbing pain around one eye
  - pain typical occurs once or twice a day, each episode lasting 15 mins - 2 hours
  - the patient is restless and agitated during an attack due to the severity

- clusters typically last 4-12 weeks
- accompanied by redness, lacrimation, lid swelling
- nasal stuffiness
- miosis and ptosis in a minority

Investigations
- most patients will have neuroimaging - underlying brain lesions are sometimes found even if the clinical symptoms are typical for cluster headache
- MRI with gadolinium contrast is the investigation of choice

Management
- NICE recommend seeking specialist advice from a neurologist if a patient develops cluster headaches
- acute
  - 100% oxygen (80% response rate within 15 minutes)
  - subcutaneous triptan (75% response rate within 15 minutes)
- prophylaxis
  - verapamil is the drug of choice
  - there is also some evidence to support a tapering dose of prednisolone

Some neurologists use the term trigeminal autonomic cephalgia to group a number of conditions including cluster headache, paroxysmal hemicrania and short-lived unilateral neuralgiform headache with conjunctival injection and tearing (SUNCT). It is recommended such patients are referred for specialist assessment as specific treatment may be required, for example it is known paroxysmal hemicrania responds very well to indomethacin

123

Next question >

Textbooks

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Links

NICE

2012 Headache guidelines

17 10

Suggest link

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Media

SEVERE SYMPTOMS
HIGH FLOW 100% OXYGEN
CLUSTER HEADACHES
PREVENTION
SUBCUT TRIPTANS
HOME OXYGEN

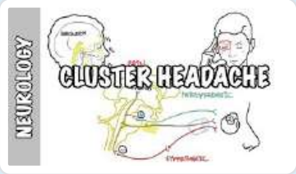
Cluster headaches

Zero To Finals - YouTube 👍 0 👎 0

CLUSTER HEADACHE

Cluster headache

Townsend Teaching - YouTube 👍 0 👎 0


NEUROLOGY
CLUSTER HEADACHE

Cluster Headaches - symptoms, pathophysiology, treatment

Armando Hasudungan - YouTube 👍 1 👎 1

Report broken media

Score: **24.1%**

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A 16-year-old boy attends GP with their parents for a review of constipation. They have improved well and are taking regular movicol with a soft motion every 1-2 days. The parents asked about a rash across the face. He has a background of autism and learning difficulty.

On examination, the boy has raised papules across the nose and cheeks with a warty appearance that measure 1-2mm. This does not extend across the back or chest. He has 2 patches of hypopigmentation on his lower back that are 3cm x 2cm and 1cm x 5cm. There are three hyperpigmented patches over the abdomen.

These have not resolved despite using over the counter treatments for acne.

Hb	134 g/l	Na$^+$	139 mmol/l	Bilirubin	5 µmol/l
Platelets	456 * 10^9/l	K$^+$	4.2 mmol/l	ALP	100 u/l
WBC	4.5 * 10^9/l	Urea	4.2 mmol/l	ALT	24 u/l
Neuts	2.2 * 10^9/l	Creatinine	78 µmol/l	γGT	78 u/l
Lymphs	0.1 10^9/l			Albumin	35 g/l

What treatment would you start for the facial lesions?

- ○ Sirolimus
- ○ Retinoid
- ○ Lymecycline
- ○ Benzoyl peroxide
- ○ Excision

Submit answer

Reference ranges

Score: **0%**

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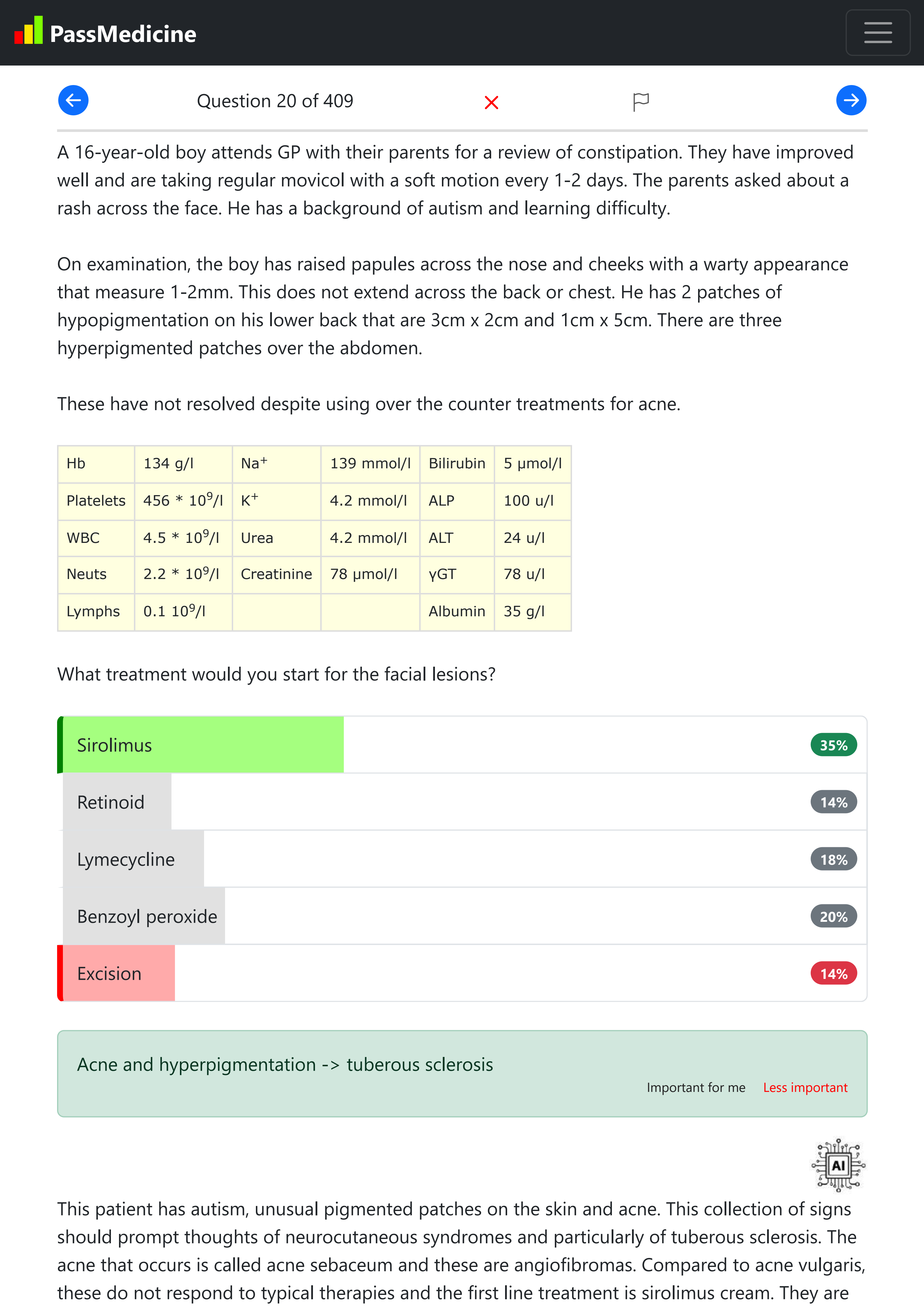

Question 20 of 409

A 16-year-old boy attends GP with their parents for a review of constipation. They have improved well and are taking regular movicol with a soft motion every 1-2 days. The parents asked about a rash across the face. He has a background of autism and learning difficulty.

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Neuts	2.2 * 10^9/l	Creatinine	78 µmol/l	γGT	78 u/l
Lymphs	0.1 10^9/l			Albumin	35 g/l

What treatment would you start for the facial lesions?

Sirolimus	35%
Retinoid	14%
Lymecycline	18%
Benzoyl peroxide	20%
Excision	14%

Acne and hyperpigmentation -> tuberous sclerosis

Important for me Less important

This patient has autism, unusual pigmented patches on the skin and acne. This collection of signs should prompt thoughts of neurocutaneous syndromes and particularly of tuberous sclerosis. The acne that occurs is called acne sebaceum and these are angiofibromas. Compared to acne vulgaris, these do not respond to typical therapies and the first line treatment is sirolimus cream. They are too small and numerous to be excised. This boy should be referred to genetics to confirm the diagnosis and screen for further malignancies that he will be at risk of.

Discuss (5) Improve

Next question

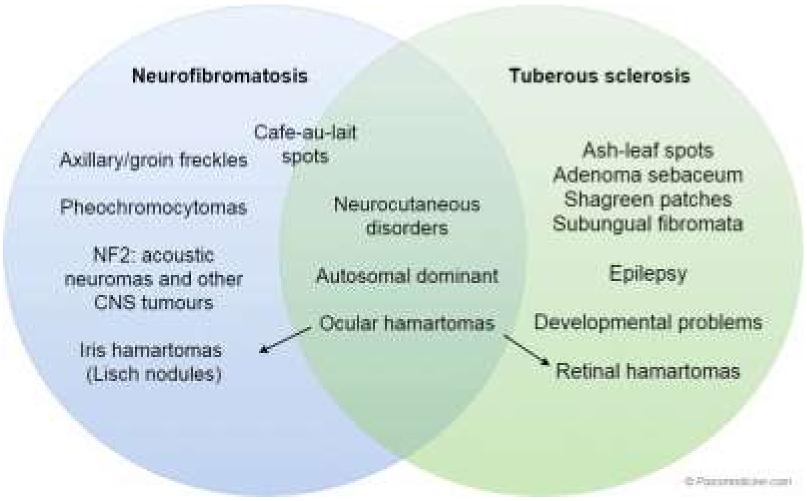
Tuberous sclerosis

Tuberous sclerosis (TS) is a genetic condition of autosomal dominant inheritance. Like neurofibromatosis, the majority of features seen in TS are neurocutaneous.

Cutaneous features
- depigmented 'ash-leaf' spots which fluoresce under UV light
- roughened patches of skin over lumbar spine (Shagreen patches)
- adenoma sebaceum (angiofibromas): butterfly distribution over nose
- fibromata beneath nails (subungual fibromata)
- cafe-au-lait spots* may be seen

Neurological features
- developmental delay
- epilepsy (infantile spasms or partial)
- intellectual impairment

Also
- retinal hamartomas: dense white areas on retina (phakomata)
- rhabdomyomas of the heart
- gliomatous changes can occur in the brain lesions
- polycystic kidneys, renal angiomyolipomata
- lymphangioleiomyomatosis: multiple lung cysts


Neurofibromatosis
Tuberous sclerosis
Axillary/groin freckles
Cafe-au-lait spots
Pheochromocytomas
NF2: acoustic neuromas and other CNS tumours
Iris hamartomas (Lisch nodules)
Neurocutaneous disorders
Autosomal dominant
Ocular hamartomas
Ash-leaf spots
Adenoma sebaceum
Shagreen patches
Subungual fibromata
Epilepsy
Developmental problems
Retinal hamartomas

Comparison of neurofibromatosis and tuberous sclerosis. Note that whilst they are both autosomal dominant neurocutaneous disorders there is little overlap otherwise

*these of course are more commonly associated with neurofibromatosis. However a 1998 study of 106 children with TS found cafe-au-lait spots in 28% of patients

123

Next question >

Textbooks

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Links

Patient.info 👍 6 👎 12

Tuberous sclerosis review

Suggest link Report broken link

Media

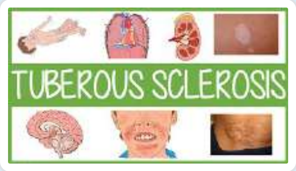
TUBEROUS SCLEROSIS
Osmosis.org

Tuberous sclerosis

Osmosis - YouTube 👍 11 👎 0

Angiomyolipoma

Osmosis - YouTube 👍 3 👎 0


TUBEROUS SCLEROSIS

Tuberous Sclerosis Complex

Townsend Teaching - YouTube 👍 3 👎 0

Tuberous
Sclerosis

Tuberous Sclerosis Complex (TSC)

Pixorize - YouTube 👍 2 👎 2

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Score: **24.1%**

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Question 21 of 409

An 18-year-old female is admitted to hospital with worsening lower limb weakness. She states that it started in her ankles, but now she is unable to stand from a squatting position which is abnormal for her. On examination, she has reduced power in ankle plantar and dorsiflexion bilaterally as well as some mild weakness in her hip extensors. Her hand grip strength is normal.

A cerebrospinal fluid (CSF) study is carried out and based on the clinical syndrome and the result she is diagnosed with Guillain-Barre Syndrome (GBS).

Which of the following treatments should be commenced in the acute setting?

○ Intravenous methylprednisolone ×

○ Intravenous immunoglobulin ×

○ Oral prednisone ×

○ Rituximab ×

○ Pyridostigmine ×

Submit answer

Reference ranges

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Question 21 of 409

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A cerebrospinal fluid (CSF) study is carried out and based on the clinical syndrome and the result she is diagnosed with Guillain-Barre Syndrome (GBS).

Which of the following treatments should be commenced in the acute setting?

Intravenous methylprednisolone	6%
Intravenous immunoglobulin	91%
Oral prednisone	2%
Rituximab	0%
Pyridostigmine	1%

IVIG is generally used first-line for Guillain-Barre syndrome

Important for me Less important

Immunomodulatory treatment has been proven to hasten recovery in GBS. Intravenous immunoglobulin (IVIG) and plasma exchange have proved equally effective, however, IVIG is often the initial treatment for practical reasons.

Corticosteroids (oral and intravenous) have not been found to have a clinical benefit in GBS. Consequently, this class of drugs is not currently employed in the treatment of the syndrome.

Discuss (1) Improve

Next question

Guillain-Barre syndrome: management

Guillain-Barre syndrome describes an immune-mediated demyelination of the peripheral nervous system often triggered by an infection (classically *Campylobacter jejuni*).

Management

- IV immunoglobulins (IVIG) or plasma exchange can be used
    - IVIG is as effective as plasma exchange better tolerated and easier to administer
    - therefore IVIG is generally used first-line for Guillain-Barre syndrome where patients require immunomodulatory therapy
    - no benefit in combining both treatments
- steroids and immunosuppressants have not been shown to be beneficial
- FVC regularly to monitor respiratory function

Prognosis

- severe motor problems persist in about 15%
- around 5% die

123

Next question

Textbooks

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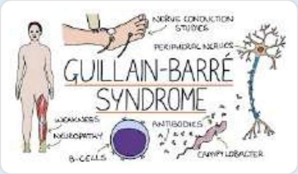
Extended textbook

Media


NERVE CONDUCTION STUDIES
PERIPHERAL NERVES
GUILLAIN-BARRÉ SYNDROME
WEAKNESS
NEUROPATHY
ANTIBODIES
B-CELLS
CAMPYLOBACTER

Understanding Guillain-Barre Syndrome

Zero To Finals - YouTube 3 1

Guillain-Barre syndrome

Osmosis - YouTube 👍 1 👎 1

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Score: **24.1%**

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Question 22 of 409

You are working in a neurology outpatient clinic seeing a patient referred from a local GP clinic. He's a 42-year-old man who has been troubled by severe headaches over the past half a year. These headaches are the worse that he's ever had in his life, describing them as far worse that the compound fracture he sustained three years ago. These headaches tend to happen most nights at around 2am just after he falls asleep. He often paces around his kitchen for a couple of hours and often resorts to bashing his head against the fridge the pain is so bad. When probed further he mentioned that he gets a sense of fullness in his right ear (which is the side that the headache most often occurs on). He remembers having a similar problem a couple of years ago that lasted a few months before resolving on their own.

What medication is most likely to prevent these headaches?

○ Oral triptan

○ Home oxygen

○ Pizotifen

○ Propranolol

○ Verapamil

Submit answer

Reference ranges

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What medication is most likely to prevent these headaches?

Oral triptan	3%
Home oxygen	4%
Pizotifen	4%
Propranolol	16%
Verapamil	74%

Verapamil is used for long-term prophylaxis of cluster headaches

Important for me Less important

NICE guidelines suggest that one should diagnose a cluster headache if:

- Pain lasts 15 minutes to 3 hours and is associated with intense restlessness and agitation.
- Episodes occur with a frequency between one every other day and eight times a day.
- Commonly wakes the person from sleep within 2 hours of going to sleep.
- At least five episode of pain have occurred.
- Headache is associated with at least one autonomic feature occurring on the same side as the pain.

Acute treatment for cluster headaches:

- Subcutaneous or nasal triptan
- High flow oxygen 12L/min - this should be set up at home

Prophylactic treatment:

- Verapamil

👍 👎 💬 Discuss (5) Improve

Next question >

Cluster headache ★

Cluster headaches are known to be one of the most painful conditions that patients can have the misfortune to suffer. The name relates to the pattern of the headaches - they typically occur in clusters lasting several weeks, with the clusters themselves typically once a year.

Cluster headaches are more common in men (3:1) and smokers. Alcohol may trigger an attack and there also appears to be a relation to nocturnal sleep.

Features
-intense sharp, stabbing pain around one eye

- pain typical occurs once or twice a day, each episode lasting 15 mins - 2 hours
- the patient is restless and agitated during an attack due to the severity

- clusters typically last 4-12 weeks
- accompanied by redness, lacrimation, lid swelling
- nasal stuffiness
- miosis and ptosis in a minority

Investigations

- most patients will have neuroimaging - underlying brain lesions are sometimes found even if the clinical symptoms are typical for cluster headache
- MRI with gadolinium contrast is the investigation of choice

Management

- NICE recommend seeking specialist advice from a neurologist if a patient develops cluster headaches
- acute
  - 100% oxygen (80% response rate within 15 minutes)
  - subcutaneous triptan (75% response rate within 15 minutes)
- prophylaxis
  - verapamil is the drug of choice
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Some neurologists use the term trigeminal autonomic cephalgia to group a number of conditions including cluster headache, paroxysmal hemicrania and short-lived unilateral neuralgiform headache with conjunctival injection and tearing (SUNCT). It is recommended such patients are referred for specialist assessment as specific treatment may be required, for example it is known paroxysmal hemicrania responds very well to indomethacin

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Textbooks

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Links

NICE 👍 17 👎 10

2012 Headache guidelines

Suggest link

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Media

SEVERE SYMPTOMS
HIGH FLOW 100% OXYGEN
CLUSTER HEADACHES
PREVENTION
SUBCUT TRIPTANS
HOME OXYGEN

Cluster headaches

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CLUSTER HEADACHE

Cluster headache

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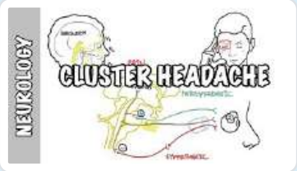
NEUROLOGY
CLUSTER HEADACHE

Cluster Headaches - symptoms, pathophysiology, treatment

Armando Hasudungan - YouTube 👍 1 👎 1

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Score: **24.1%**

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Question 23 of 409

A 20-year-old man presented to his general practitioner following an episode of collapse. He reported that he was attending a comedy night when he suddenly collapsed to the floor. He denied loss of consciousness and had a quick recovery from the event. He is normally fit and well, though reports that he is having difficulty at university as he is struggling to stay awake.

On examination, his pulse was regular at 60bpm and ECG demonstrated normal sinus rhythm. Blood pressure was 134/70mmHg on sitting and 125/65mmHg on standing.

What is the most likely cause of his collapse?

○ Transient ischemic attack (TIA)

○ Neuromuscular dystrophy

○ Cataplexy

○ Conversion disorder

○ Stokes-Adams syndrome

Submit answer

Reference ranges

Score: **0%**

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On examination, his pulse was regular at 60bpm and ECG demonstrated normal sinus rhythm. Blood pressure was 134/70mmHg on sitting and 125/65mmHg on standing.

What is the most likely cause of his collapse?

Transient ischemic attack (TIA)	1%
Neuromuscular dystrophy	1%
Cataplexy	86%
Conversion disorder	3%
Stokes-Adams syndrome	9%

Cataplexy occurs during heightened emotion. Differential to consider with collapse of unknown cause

Important for me Less important

Cataplexy is a sudden, transient episode of muscular weakness without loss of consciousness. Typically it occurs during heightened emotions, most commonly laughter such as the comedy clue in the question. Often cataplexy co-occurs with narcolepsy which typically presents as patients recurrently falling asleep during the day, as with this patient.

Stokes-Adams syndrome is a transient AV block and results in loss of consciousness, a more progressive history would be expected with neuromuscular dystrophy and although TIAs do present with transient focal motor weakness the patient's age and the bilateral nature of his symptoms make this unlikely.

Discuss (3) Improve

Next question >

Cataplexy ★

Cataplexy describes the sudden and transient loss of muscular tone caused by strong emotion (e.g. laughter, being frightened). Around two-thirds of patients with narcolepsy have cataplexy.

Features range from buckling knees to collapse.

AI + Q 123

Next question >

B I A

Textbooks

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Score: **24.1%**

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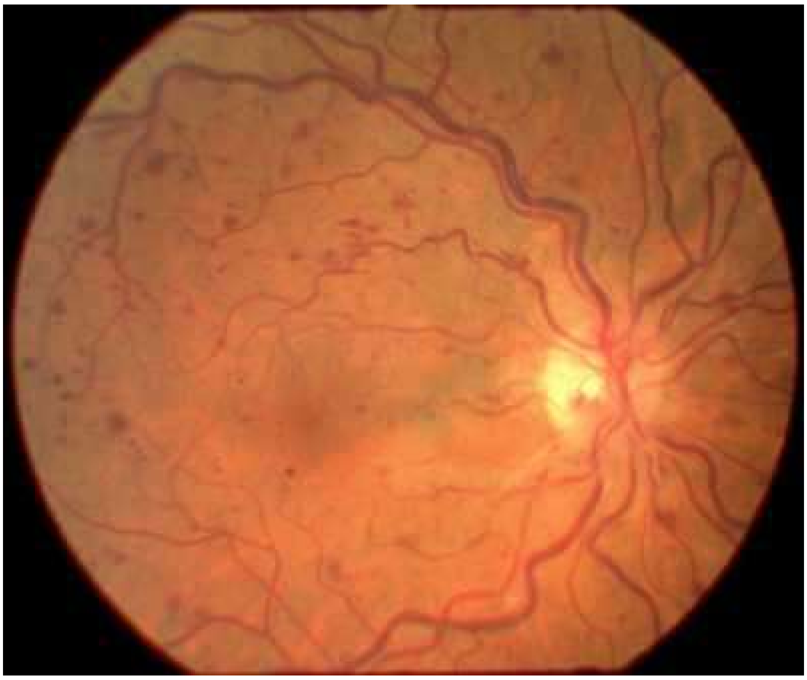
Question 24 of 409

A 73-year-old man complained of a one day history of blurred vision in the right eye. He has a past history of poorly controlled hypertension and polycythaemia but otherwise enjoys good health. Visual acuity is 6/6 in the left eye and 6/18 in the right eye. Fundoscopy of the right eye is as follows:



What is the most likely diagnosis?

- ○ Ischaemic optic neuropathy
- ○ Central retinal artery occlusion
- ○ Papilloedema
- ○ Central retinal vein occlusion
- ○ Macular degeneration

Submit answer

Reference ranges

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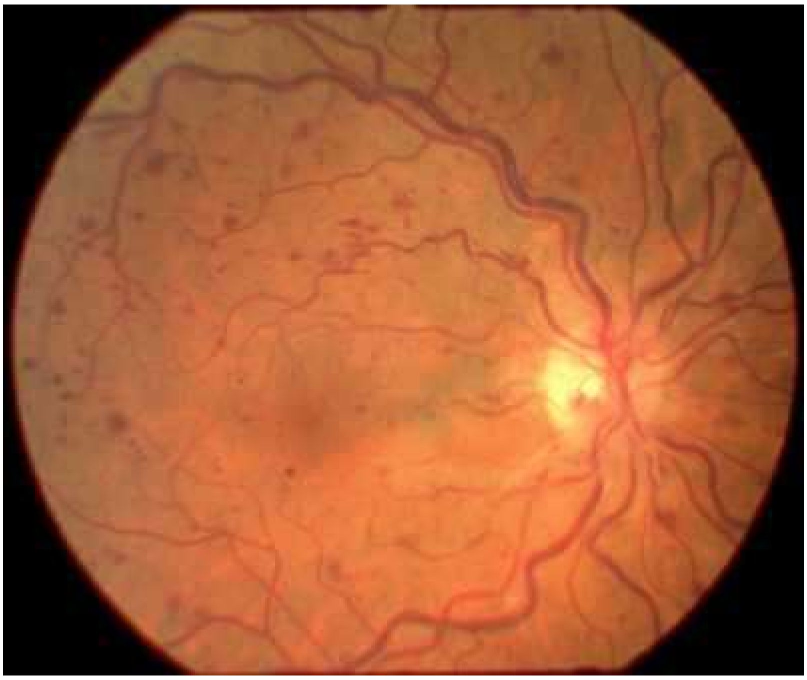


What is the most likely diagnosis?

Ischaemic optic neuropathy	24%
Central retinal artery occlusion	12%
Papilloedema	15%
Central retinal vein occlusion	38%
Macular degeneration	11%

Central retinal vein occlusion - sudden painless loss of vision, severe retinal haemorrhages on fundoscopy

Important for me   Less important

This slide shows a non-ischaemic central retinal vein occlusion. There are dilated retinal veins and widespread multiple blot haemorrhages.

👍 👎 Discuss (4) Improve

Next question ›

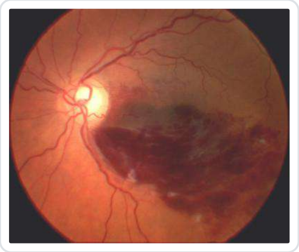
Central retinal vein occlusion ★

Central retinal vein occlusion (CRVO) is a differential for sudden painless loss of vision.

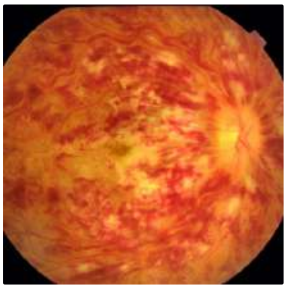
Risk factors
- increasing age
- hypertension
- cardiovascular disease
- glaucoma
- polycythaemia

Features
- sudden, painless reduction or loss of visual acuity, usually unilaterally
- fundoscopy
  - widespread hyperaemia
  - severe retinal haemorrhages - 'stormy sunset'

A key differential is branch retinal vein occlusion (BRVO) - this occurs when a vein in the distal retinal venous system is occluded and is thought to occur due to blockage of retinal veins at arteriovenous crossings. It results in a more limited area of the fundus being affected.



Management
- the majority of patients are managed conservatively
- indications for treatment in patients with CRVO include:
  - macular oedema - intravitreal anti-vascular endothelial growth factor (VEGF) agents
  - retinal neovascularization - laser photocoagulation



123

Next question >

B I A T↕

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A 43-year-old gentleman is admitted to the Emergency Department with a 2-week history of worsening drowsiness and confusion. His wife states that he had initially complained of increased tiredness and had started taking to his bed immediately after arriving home from work. One week ago he stopped going to work altogether and his wife reports that he has been sleeping for most of the day since then. This morning she had difficulty waking him, and when she did manage to rouse him he seemed confused with apparent slurring of his speech.

His past medical history is remarkable for epilepsy and bipolar disorder.

On examination, he is responsive only to pain. His vital signs are within normal limits and examination of the chest is unremarkable. His abdomen is soft, with no obvious tenderness and no organomegaly. Bowel sounds are present.

A set of blood tests are taken:

Hb	152 g/l	Na^+	139 mmol/l	Bilirubin	18 µmol/l
Platelets	278 * 10^9/l	K^+	4.3 mmol/l	ALP	117 u/l
WBC	8.1 * 10^9/l	Urea	5.2 mmol/l	ALT	19 u/l
Neuts	5.4 * 10^9/l	Creatinine	93 µmol/l	γGT	48 u/l
Lymphs	1.8 * 10^9/l			Albumin	41 g/l
Eosin	0.2 * 10^9/l			Ammonia	197 µmol/l

His wife later remarks that his medications have recently been altered by his GP. Which of the following is most likely to be responsible?

- ○ Lithium
- ○ Sodium valproate
- ○ Venlafaxine
- ○ Diazepam
- ○ Quetiapine

Submit answer

Reference ranges

Score: **0%**

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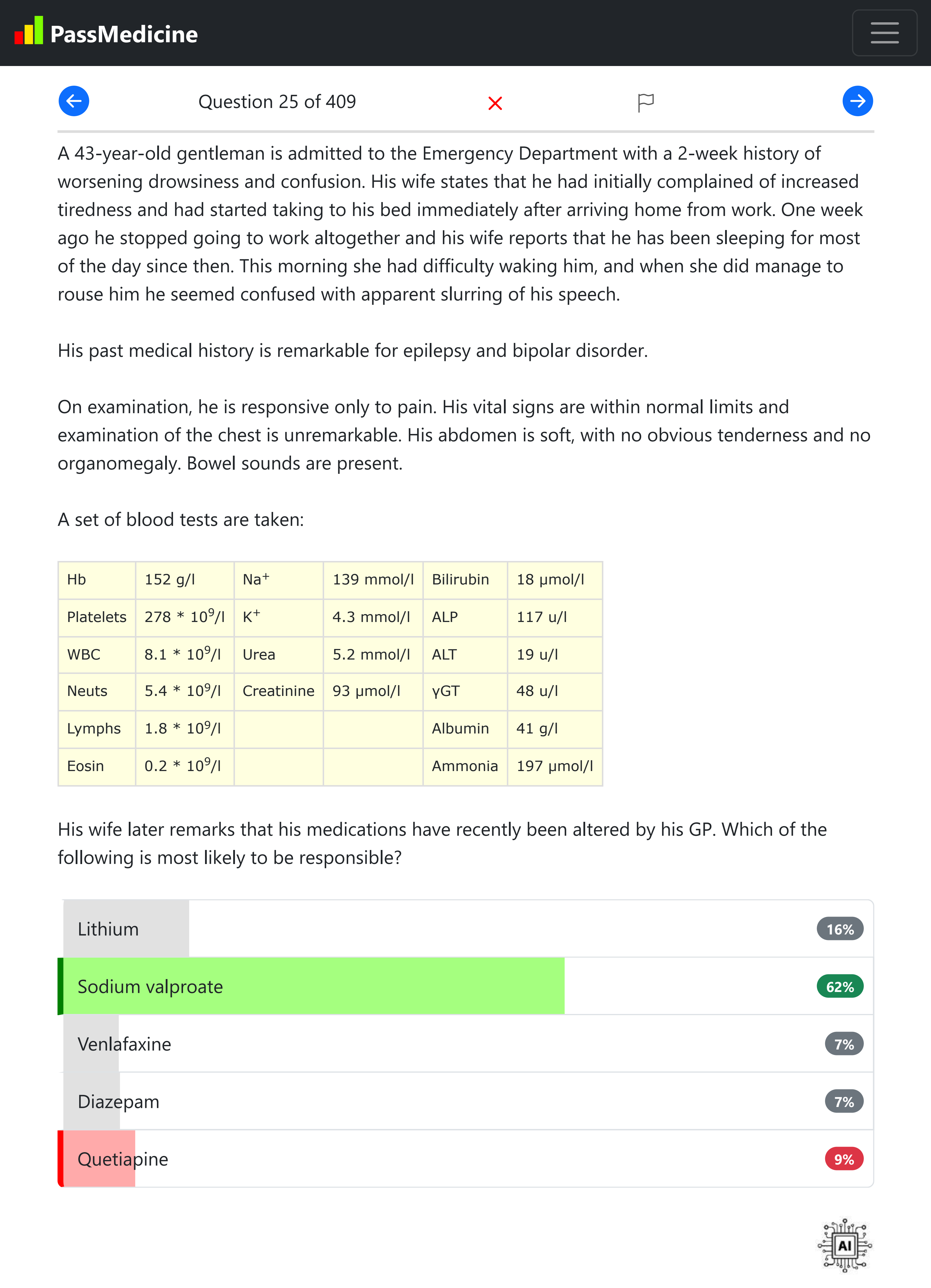

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Eosin	0.2 * 10^9/l			Ammonia	197 µmol/l

His wife later remarks that his medications have recently been altered by his GP. Which of the following is most likely to be responsible?

Lithium	16%
Sodium valproate	62%
Venlafaxine	7%
Diazepam	7%
Quetiapine	9%

The patient's presenting symptoms are relatively non-specific and, taken in high enough doses, any of the medications listed could cause impairment of consciousness. The patient's grossly abnormal ammonia level is the critical detail in this case and confirms a diagnosis of valproate-associated hyperammonaemic encephalopathy (VHE).

VHE is a well-documented complication of valproate therapy and can occur in patients with therapeutic as well supratherapeutic plasma valproate levels. Valproate inhibits carbamoyl phosphate synthetase I, an enzyme essential for ammonia metabolism in the liver, leading to elevated plasma ammonium levels. Interestingly, asymptomatic hyperammonaemia has been shown to occur in a large proportion of patients taking sodium valproate.

Reference: Wadzinski, J., Franks, R., Roane, D. and Bayard, M. (2007). Valproate-associated Hyperammonemic Encephalopathy. The Journal of the American Board of Family Medicine, 20(5), pp.499-502.

👍 👎 Discuss (7) Improve

Next question ›

Sodium valproate

Sodium valproate is used in the management of epilepsy and is first-line therapy for generalised seizures. It works by increasing GABA activity.

Adverse effects

- teratogenic
    - neural tube defects
    - maternal use of sodium valproate is associated with a significant risk of neurodevelopmental delay in children
    - guidance is now clear that sodium valproate should not be used during pregnancy and in women of childbearing age unless clearly necessary. Women of childbearing age should not start treatment without specialist neurological or psychiatric advice.
- P450 inhibitor
- gastrointestinal: nausea
- increased appetite and weight gain
- alopecia: regrowth may be curly
- ataxia
- tremor
- hepatotoxicity
- pancreatitis
- thrombocytopaenia
- hyponatraemia
- hyperammonemic encephalopathy: L-carnitine may be used as treatment if this develops

123

Next question ›

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A 23-year-old female has represented 2 days after discharge from the neurology team with a constant diffuse headache, worse on standing than lying down, without neck stiffness or photophobia, but associated with nausea and vomiting. 48 hours ago, she was an inpatient being investigated for intermittent headaches over the past 8 months, typically over the left side of her frontal and temporal regions, of sudden onset during any time of day, associated with double vision, lasting for 'hours' at a time. She was experiencing up to 5 episodes a week, with one episode witnessed during her admission, when the senior house officer noted bilateral restriction in vertical eye movements, adduction and unreactive pupils, spontaneously resolving after 3 hours. Her blood tests were unremarkable.

The patient underwent a CT head, demonstrating no intracranial lesions; and a lumbar puncture:

Opening pressure	12.3 cmH2O
WCC	2 /mm³
RBC	50 /mm³
Protein	0.45 g/l
Glucose	4.5 mmol/l (serum 6.7 mmol/l)
Oligoclonal bands	None present

The patient subsequently self-discharged, reporting no immediate headaches, back pain or lower limb paraesthesia after the lumbar puncture.

During this second admission, she undergoes an MRI head scan, demonstrating significant diffuse meningeal enhancement and bilateral shallow subdural haemorrhages. What is the appropriate treatment?

Oral fluids, caffeine and blood patch	54%
Repeat lumbar puncture	4%
Intravenous aciclovir	12%
Intravenous ceftriaxone	3%
Neurosurgical input	28%

There are two headaches here: the initial presentation appears to be an unusual migrainous phenomenon, possibly ophthalmoplegic migraine, while the second is clearly a low-pressure headache. The neurologists would have correct in initially ruling out any other possible causes of ocular paresis with a lumbar puncture but the CSF constituents are fairly unremarkable.

Do not be alarmed by the report of the MRI head: meningeal enhancement, thickening and shallow subdural haematoma are features of low-pressure headaches and do not represent meningitis or subdural haematomas regarding neurosurgical drainage. A repeat lumbar puncture would make exacerbate the problem and hence inappropriate. In view of the low-pressure headache occurring soon after a lumbar puncture, encouraging oral fluids, caffeine and a blood patch by the anaesthetists would be most appropriate.

Discuss (7) | Improve

Next question >

Post-lumbar puncture headache

Headache following lumbar puncture (LP) occurs in approximately one-third of patients. The pathophysiology of is unclear but may relate to a 'leak' of CSF following dural puncture. Post-LP headaches are more common in young females with a low body mass index

Typical features
- usually develops within 24-48 hours following LP but may occur up to one week later
- may last several days
- worsens with upright position
- improves with recumbent position

Factors which may contribute to headache	**Factors which do not contribute to headache**
• Needle type: conventional 'cutting' needles increase the risk compared to pencil point (atraumatic) needles • Increased needle size • Direction of bevel • Not replacing the stylet • Increased number of LP attempts	• Increased volume of CSF removed • Bed rest following procedure • Increased fluid intake post procedure • Opening pressure of CSF • Position of patient

Management
- supportive initially (analgesia, rest)

- if pain continues for more than 72 hours then specific treatment is indicated, to prevent subdural haematoma
- treatment options include: blood patch, epidural saline and intravenous caffeine

+ Q 123

Next question >

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A 17-year-old Caucasian male with no past medical history presents with his first episode of sudden onset left leg weakness and numbness on his anterior left thigh, of sudden onset and persistent after 4 days. On examination, you note 3/5 weakness on flexion of his left hip and loss of sensation to light touch, pain and temperature on his anterior left thigh in the sensory nerve root L1 distribution. A contrasted MRI scan of the patient's spine reveals a hyperintense T2 signal partially within the left side of the cord at the L1, with corresponding enhancement with gadolinium. No masses were observed. Further imaging of the brain is awaited. What is the most likely diagnosis at present?

Multiple sclerosis	29%
Left spinal cord tumour	6%
Left anterior spinal ischaemic stroke	17%
Left transverse myelitis	48%
Guillain Barre syndrome	1%

The history, examination and radiological evidence are strongly suggestive of partial L1 transverse myelitis. The key question is whether this represents a diagnosis of multiple sclerosis (MS) or not. The diagnosis of MS is determined by the McDonald criteria, first proposed in 2001 and most recently revised in 2010[1]. The lesions or clinical episodes suggestive of acute demyelinating episodes must be disseminated in both time and place. As a result, a patient presenting with only one symptomatic episode can be diagnosed with MS if their MRI scans demonstrate demyelinating plaques that are:

i) distinguished in age from the current symptomatic lesion
ii) In at least two of 4 areas typical of MS (periventricular, juxtacortical, infratentorial, spinal cord white matter).

In this case, only one episode in time and space has been demonstrated so far. Although a risk of future MS diagnosis is acknowledged and may go on to be proven following MRI of the head, the diagnosis remains transverse myelitis at present.

It is often quoted that in patients diagnosed with transverse myelitis after their first symptomatic episode, only a small proportion go on to be diagnosed with MS later in their lives. However, more recent evidence suggests the pattern of transverse myelitis is important: only 5-10% of patients with complete transverse myelitis will be diagnosed with MS, with a viral febrile illness the most likely underlying cause. Patients with incomplete transverse myelitis have up to 72% risk of MS-consistent plaques found on MRI head, with 60-90% subsequently being diagnosed MS within 5 years[2].

Old MS plaques appear better defined on T1 and T2 imaging than acute lesions. Contrast studies with gadolinium are also helpful: acute plaques tend to enhance to a greater degree on T1 imaging, correlated to acute inflammation that persists for up to 8 weeks and diminishes on treatment with glucocorticoids.

1. Montalban X, TintoreM, Swanton et al. MRI criteria for MS in patients with clinically isolated syndromes.
Neurology. 2010;74(5):427

2. Morrissey SP, Miller DH, Kendall BE et al. The significance of brain magnetic resonance imaging abnormalities at presentation with clinically isolated syndromes suggestive of multiple sclerosis. A 5-year follow-up study. Brain. 1993;116 (Pt 1):135

Discuss (8) Improve

Next question

Transverse myelitis

Causes of transverse myelitis

- viral infections: varicella-zoster, herpes simplex, cytomegalovirus, Epstein-Barr, influenza, echovirus, human immunodeficiency virus
- bacterial infections: syphilis, Lyme disease
- post-infectious (immune mediated)
- first symptom of multiple sclerosis (MS) or neuromyelitis optica (NMO)

+ Q 123

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TRANSVERSE MYELITIS
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Transverse myelitis

Osmosis - YouTube 👍 9 👎 0

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Question 28 of 409 ✓

A 32-year-old male presents with his 4th episode of worst ever headache in one week. He describes the headache to always be of sudden onset on the left side of his head, of 10 out of 10 severity and that he finds bright lights extremely distressing during these periods. The episodes last for around 30 minutes, typically after dinner. He also describes redness and swelling of his left eye and a blocked left nostril during the headaches, associated with tearing of his left eye.

He has no past medical history and family history of migraines. He denies illicit drug use, is a non-smoker and drinks two glasses of wine with dinner every night. Over the past 7 days, he has been self-medicating with paracetamol and ibuprofen. On examination, you notice no focal neurology, no meningism and fundoscopy is unremarkable.

What is the most likely diagnosis?

Subarachnoid haemorrhage	1%
First presentations of migraine	1%
SUNCT (short lasting unilateral neuralgiform headache with conjunctival injection or tearing)	39%
Medication overuse headache	3%
Cluster headaches	56%

The main differentials are between cluster headaches and SUNCT, both of which are headache syndromes resulting in severely painful headaches associated with autonomic symptoms. It would be unusual for autonomic symptoms such as conjunctival injection, lacrimation and rhinorrhoea with migraines. Subarachnoid haemorrhage headaches rarely re-occur without disastrous consequences, focal neurology and reduced GCS. Medication overuse headache are normally of onset after a longer period of analgesia use and are more chronic in character without autonomic symptoms.

In distinguishing cluster headaches and SUNCT, the former are more prevalent in younger males below the age of 40 while SUNCT is more common in older patients above the age of 40. Cluster headaches typically onset at night, last from 15 minutes to 3 hours, while SUNCT can occur at any time of day and typically lasts for seconds to minutes. Cluster headaches rarely onset more than 3 times per day while SUNCT has been described in up to 75 times per day. A transient Horners syndrome is typical during a cluster headache but may be lacking in SUNCT. Lastly, cluster headaches are classically, but not always, triggered by alcohol[1]. The combination of severe nocturnal onset unilateral headache following alcohol in a patient under 40 years old, lasting for 30 minutes, suggest cluster headaches over SUNCT.

1. Kruszewski P, Pareja JA, Caminero AB et al. Cluster headaches and SUNCT: similarities and differences. J Headache Pain 2001; 2:57-66

Discuss (7) Improve

Next question

Cluster headache

Cluster headaches are known to be one of the most painful conditions that patients can have the misfortune to suffer. The name relates to the pattern of the headaches - they typically occur in clusters lasting several weeks, with the clusters themselves typically once a year.

Cluster headaches are more common in men (3:1) and smokers. Alcohol may trigger an attack and there also appears to be a relation to nocturnal sleep.

Features
-intense sharp, stabbing pain around one eye
  - pain typical occurs once or twice a day, each episode lasting 15 mins - 2 hours
  - the patient is restless and agitated during an attack due to the severity

- clusters typically last 4-12 weeks
- accompanied by redness, lacrimation, lid swelling
- nasal stuffiness
- miosis and ptosis in a minority

Investigations
- most patients will have neuroimaging - underlying brain lesions are sometimes found even if the clinical symptoms are typical for cluster headache
- MRI with gadolinium contrast is the investigation of choice

Management
- NICE recommend seeking specialist advice from a neurologist if a patient develops cluster headaches
- acute
  - 100% oxygen (80% response rate within 15 minutes)
  - subcutaneous triptan (75% response rate within 15 minutes)
- prophylaxis
  - verapamil is the drug of choice

    - there is also some evidence to support a tapering dose of prednisolone

Some neurologists use the term trigeminal autonomic cephalgia to group a number of conditions including cluster headache, paroxysmal hemicrania and short-lived unilateral neuralgiform headache with conjunctival injection and tearing (SUNCT). It is recommended such patients are referred for specialist assessment as specific treatment may be required, for example it is known paroxysmal hemicrania responds very well to indomethacin

AI + Q 123

Next question >

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Links

NICE 17 10

2012 Headache guidelines

Suggest link Report broken link

Media

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HIGH FLOW 100% OXYGEN
CLUSTER HEADACHES
PREVENTION
SUBCUT TRIPTANS
HOME OXYGEN

Cluster headaches

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CLUSTER HEADACHE

Cluster headache

Townsend Teaching - YouTube 👍 0 👎 0

NEUROLOGY
CLUSTER HEADACHE

Cluster Headaches - symptoms, pathophysiology, treatment

Armando Hasudungan - YouTube 👍 1 👎 1

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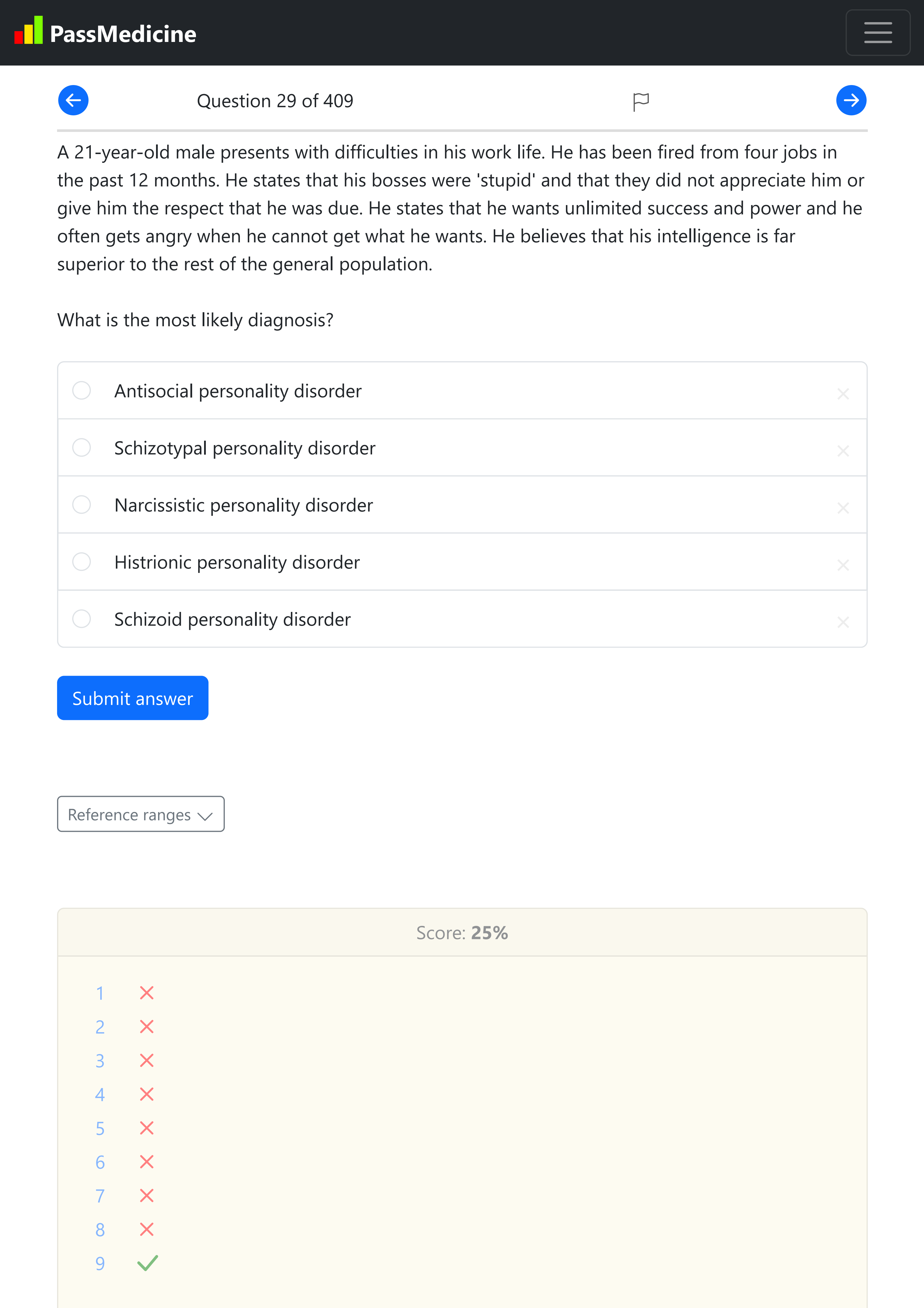

A 21-year-old male presents with difficulties in his work life. He has been fired from four jobs in the past 12 months. He states that his bosses were 'stupid' and that they did not appreciate him or give him the respect that he was due. He states that he wants unlimited success and power and he often gets angry when he cannot get what he wants. He believes that his intelligence is far superior to the rest of the general population.

What is the most likely diagnosis?

- ○ Antisocial personality disorder ×
- ○ Schizotypal personality disorder ×
- ○ Narcissistic personality disorder ×
- ○ Histrionic personality disorder ×
- ○ Schizoid personality disorder ×

Submit answer

Reference ranges

Score: **25%**

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What is the most likely diagnosis?

Antisocial personality disorder	2%
Schizotypal personality disorder	5%
Narcissistic personality disorder	84%
Histrionic personality disorder	5%
Schizoid personality disorder	4%

Narcissistic personality disorder is characterised by a grandiose sense of self importance

Important for me Less important

Narcissistic personality disorder is characterised by a grandiose sense of self importance. In addition, they also often have a deep need for excessive attention and admiration, troubled relationships, and a lack of empathy for others.

Schizotypal personality disorder is characterised by social isolation and odd beliefs. Schizoid personality disorder is similar in that a need for social isolation and apathy dominates, however strange beliefs are not a diagnostic criteria of this condition.

Histrionic personality disorder is characterised by excessive attention-seeking and emotional overreaction.

Discuss (3) Improve

Next question

Personality disorders ★

Personality disorders (PD) are a group of mental health conditions characterised by persistent patterns of thinking, feeling, and behaving that are significantly different from cultural expectations. These patterns can lead to distress or problems in personal, social, and occupational functioning. It is thought that around 1 in 20 people have a personality disorder.

For many years patients were diagnosed with a specific 'named' personality disorder, such as narcissistic or paranoid. This approach was changed significantly with the introduction of the ICD-11 classification which marked a shift towards a more dimensional model, such as severity and specific problems. This allows for a more flexible and comprehensive understanding of personality pathology.

The majority of patients you'll encounter with personality disorders will be 'labelled' using the previous classification system. We've therefore included details of this classification at the end of the notes.

ICD-11 classification of personality disorders

Key Features of Personality Disorder (ICD-11)

- Persistent Pattern: The individual's patterns of cognition, emotional experience, behaviour, and interpersonal functioning deviate from cultural expectations. These patterns are stable over time and span across various personal and social situations.
- Impairment: The deviation results in significant problems or dysfunctions in the person’s life, especially in relationships, work, or social functioning.
- Duration: These characteristics are stable over time, beginning in adolescence or early adulthood, and are not transient.
- Distress or Dysfunction: The impairment may result in distress to the individual or others. These patterns are not explained by another mental disorder, a medical condition, or substance misuse.

Severity Classification in ICD-11

In the ICD-11, personality disorders are classified based on the degree of severity. This provides a broader and more flexible approach:

Mild Personality Disorder:

- Some impairments in functioning, often limited to specific areas of life (e.g., intimate relationships or work).
- Symptoms may be noticeable to others but do not cause pervasive distress or dysfunction.
- The individual may still maintain relatively stable relationships and occupational roles.

Moderate Personality Disorder:

- More significant impairments in multiple areas of life (personal, social, and work).
- The individual may struggle with maintaining close relationships, and there may be greater interpersonal difficulties.
- More distressing symptoms are present, but the individual can manage day-to-day functioning with some effort or support.

Severe Personality Disorder:

- Profound impairments in all areas of life.
- Individuals may experience pervasive difficulties in interpersonal relationships, self-identity, and coping mechanisms.
- Significant distress, dysfunction, and reduced quality of life are common.
- Individuals may require intensive and long-term therapeutic interventions.

Personality Disorder Trait Domains:

In addition to severity, ICD-11 allows clinicians to specify trait domains that represent the predominant problematic areas in the individual's personality. These are:

- Negative Affectivity:
    - Tendency to experience a wide range of negative emotions such as anxiety, depression, guilt, and anger.
    - Individuals may be prone to mood swings, insecurity, and emotional lability.
- Detachment:
    - Avoidance of social interactions, emotional withdrawal, and limited pleasure from relationships.
    - Individuals may appear cold, aloof, and isolated.
- Dissociality:
    - Disregard for the rights and feelings of others, lack of empathy, and difficulty forming prosocial relationships.
    - mpulsivity and manipulative behaviours are common traits.
- Disinhibition:
    - Impulsiveness, risk-taking, and difficulty controlling behaviours.
    - Individuals may struggle with planning and foresight, leading to reckless or irresponsible actions.
- Anankastia:
    - Preoccupation with orderliness, control, and perfectionism.
    - Individuals may be rigid, stubborn, and excessively focused on rules and details.
- Borderline Pattern (also recognised in ICD-11):
    - An additional qualifier for those showing emotional instability, intense and unstable interpersonal relationships, a fluctuating sense of identity, and impulsivity.
    - This pattern is akin to borderline personality disorder as recognised in ICD-10 and DSM-5.

Previous classification of personality disorders

They are typically categorised as belonging to one of three clusters:

Cluster A: 'Odd or Eccentric'	Cluster B: 'Dramatic, Emotional, or Erratic'	Cluster C: 'Anxious and Fearful'
• Paranoid • Schizoid • Schizotypal	• Antisocial • Borderline (Emotionally Unstable) • Histrionic • Narcissistic	• Obsessive-Compulsive • Avoidant • Dependent

Cluster A: 'Odd or Eccentric'

Paranoid

- Hypersensitivity and an unforgiving attitude when insulted
- Unwarranted tendency to questions the loyalty of friends
- Reluctance to confide in others
- Preoccupation with conspirational beliefs and hidden meaning
- Unwarranted tendency to perceive attacks on their character

Schizoid

- Indifference to praise and criticism
- Preference for solitary activities
- Lack of interest in sexual interactions
- Lack of desire for companionship
- Emotional coldness
- Few interests
- Few friends or confidants other than family

Schizotypal

- Ideas of reference (differ from delusions in that some insight is retained)
- Odd beliefs and magical thinking
- Unusual perceptual disturbances
- Paranoid ideation and suspiciousness
- Odd, eccentric behaviour
- Lack of close friends other than family members
- Inappropriate affect
- Odd speech without being incoherent

Cluster B: 'Dramatic, Emotional, or Erratic'

Antisocial

- Failure to conform to social norms with respect to lawful behaviours as indicated by repeatedly performing acts that are grounds for arrest;
- More common in men;
- Deception, as indicated by repeatedly lying, use of aliases, or conning others for personal profit or pleasure;
- Impulsiveness or failure to plan ahead;
- Irritability and aggressiveness, as indicated by repeated physical fights or assaults;
- Reckless disregard for the safety of self or others;
- Consistent irresponsibility, as indicated by repeated failure to sustain consistent work behaviour or honour financial obligations;
- Lack of remorse, as indicated by being indifferent to or rationalizing having hurt, mistreated, or stolen from another

Borderline - also known as Emotionally Unstable

- Efforts to avoid real or imagined abandonment
- Unstable interpersonal relationships which alternate between idealization and devaluation
- Unstable self image
- Impulsivity in potentially self damaging area (e.g. Spending, sex, substance abuse)
- Recurrent suicidal behaviour
- Affective instability
- Chronic feelings of emptiness
- Difficulty controlling temper
- Quasi psychotic thoughts

Histrionic

- Inappropriate sexual seductiveness
- Need to be the centre of attention
- Rapidly shifting and shallow expression of emotions
- Suggestibility
- Physical appearance used for attention seeking purposes
- Impressionistic speech lacking detail
- Self dramatization
- Relationships considered to be more intimate than they are

Narcissistic

- Grandiose sense of self importance
- Preoccupation with fantasies of unlimited success, power, or beauty
- Sense of entitlement
- Taking advantage of others to achieve own needs
- Lack of empathy
- Excessive need for admiration
- Chronic envy

- Arrogant and haughty attitude

Cluster C: 'Anxious and Fearful'

Obsessive-compulsive

- Is occupied with details, rules, lists, order, organization, or agenda to the point that the key part of the activity is gone
- Demonstrates perfectionism that hampers with completing tasks
- Is extremely dedicated to work and efficiency to the elimination of spare time activities
- Is meticulous, scrupulous, and rigid about etiquettes of morality, ethics, or values
- Is not capable of disposing worn out or insignificant things even when they have no sentimental meaning
- Is unwilling to pass on tasks or work with others except if they surrender to exactly their way of doing things
- Takes on a stingy spending style towards self and others; and shows stiffness and stubbornness

Avoidant

- Avoidance of occupational activities which involve significant interpersonal contact due to fears of criticism, or rejection.
- Unwillingness to be involved unless certain of being liked
- Preoccupied with ideas that they are being criticised or rejected in social situations
- Restraint in intimate relationships due to the fear of being ridiculed
- Reluctance to take personal risks due to fears of embarrassment
- Views self as inept and inferior to others
- Social isolation accompanied by a craving for social contact

Dependent

- Difficulty making everyday decisions without excessive reassurance from others
- Need for others to assume responsibility for major areas of their life
- Difficulty in expressing disagreement with others due to fears of losing support
- Lack of initiative
- Unrealistic fears of being left to care for themselves
- Urgent search for another relationship as a source of care and support when a close relationship ends
- Extensive efforts to obtain support from others
- Unrealistic feelings that they cannot care for themselves

Management

Personality disorders are difficult to treat and in the past have been considered 'untreatable' by definition.. However, a number of approaches have been shown to help patients, including:

- psychological therapies: dialectical behaviour therapy
- treatment of any coexisting psychiatric conditions

123

Next question >

Textbooks

High-yield textbook

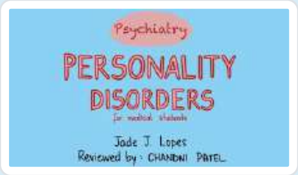
Extended textbook

Media

Personality disorders by clusters

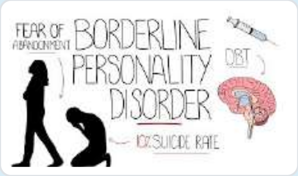
Personality disorders by clusters

MedLecturesMadeEasy - YouTube 👍 4 👎 0


Psychiatry
PERSONALITY DISORDERS
for medical students
Jade J. Lopes
Reviewed by: CHANDNI PATEL

Personality Disorders and DSH

MedFlix - YouTube 👍 1 👎 0


FEAR OF ABANDONMENT
BORDERLINE PERSONALITY DISORDER
DBT
↑ SUICIDE RATE

Borderline Personality Disorder Explained

Rhesus Medicine - YouTube 👍 1 👎 0

Cluster C personality disorders (avoidant, obsessive-compulsive, dependent)

Osmosis - YouTube 👍 0 👎 0

Cluster A personality disorders (paranoid, schizoid, schizotypal)

Osmosis - YouTube 👍 1 👎 1

Cluster B personality disorders (antisocial, borderline, histrionic, narcissistic)

Osmosis - YouTube 👍 1 👎 1

Report broken media

Score: **24.1%**

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Question 30 of 409

An 82-year-old man attends the clinic with weakness affecting his hands. A systematic enquiry was otherwise unremarkable He has no significant past medical history and takes no regular medications. On examination, you note bilateral weakness of the finger and wrist flexors (MRC grade 3/5) and hip flexion (MRC grade 4/5). The neurological examination was otherwise unremarkable.

Blood results are as follows:

Hb	144 g/L	Male: (135-180) Female: (115 - 160)
Platelets	168 * 10^9/L	(150 - 400)
WBC	7.2 * 10^9/L	(4.0 - 11.0)
Na^+	136 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	8.4 mmol/L	(2.0 - 7.0)
Creatinine	96 µmol/L	(55 - 120)
CRP	3 mg/L	(< 5)
Creatine kinase	1042 IU/L	(40 - 320)

What is the most likely diagnosis?

○ C6/7 radiculopathy

○ Inclusion body myositis

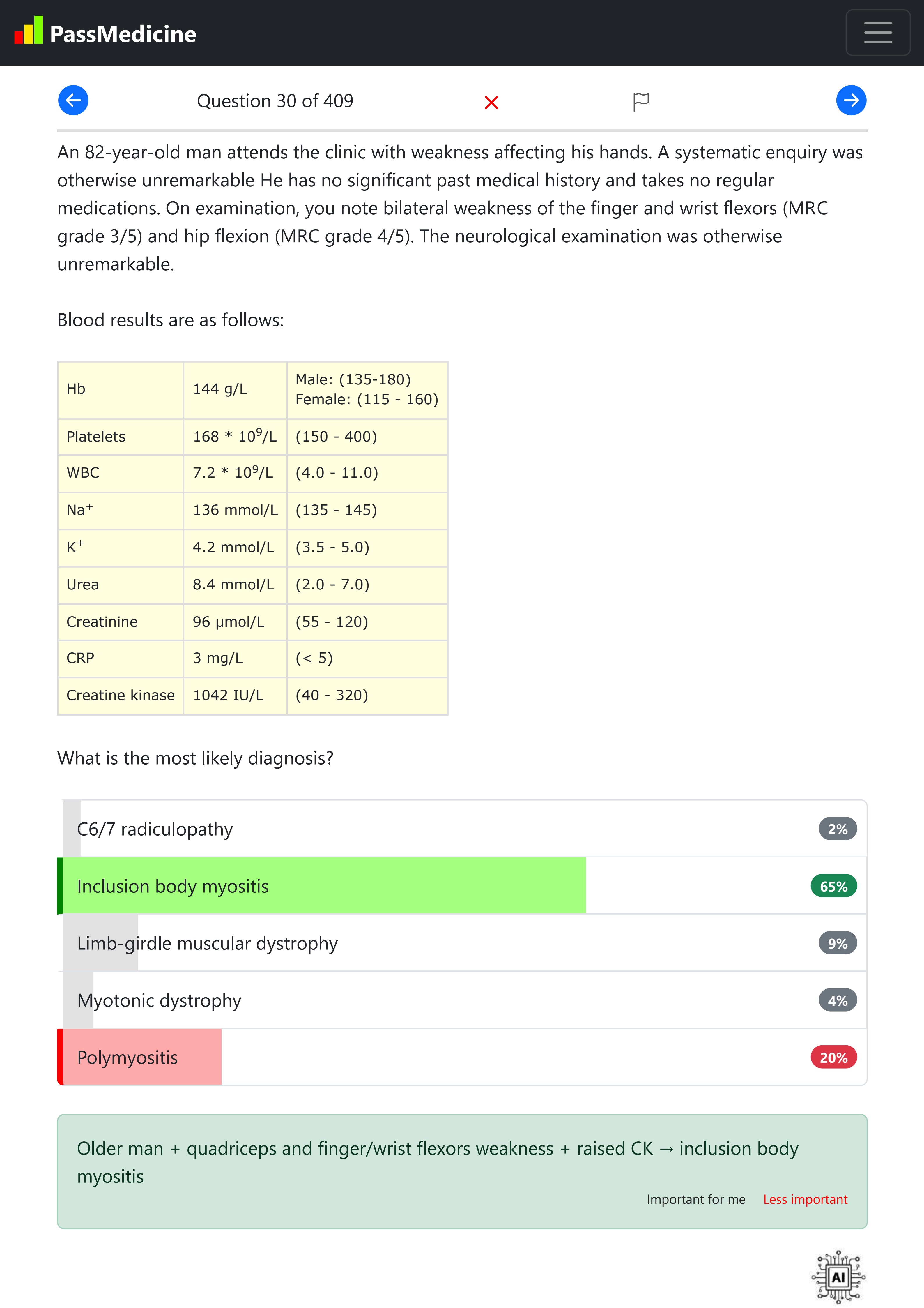
○ Limb-girdle muscular dystrophy

○ Myotonic dystrophy

○ Polymyositis

Submit answer

Reference ranges



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Urea	8.4 mmol/L	(2.0 - 7.0)
Creatinine	96 µmol/L	(55 - 120)
CRP	3 mg/L	(< 5)
Creatine kinase	1042 IU/L	(40 - 320)

What is the most likely diagnosis?

C6/7 radiculopathy	2%
Inclusion body myositis	65%
Limb-girdle muscular dystrophy	9%
Myotonic dystrophy	4%
Polymyositis	20%

Older man + quadriceps and finger/wrist flexors weakness + raised CK → inclusion body myositis

Important for me   Less important

Inclusion body myositis is correct. The presence of weakness of the finger/wrist flexors and hip flexors, and a raised CK in an elderly man is highly suggestive of inclusion body myositis.

C6/7 radiculopathy is incorrect. Although bilateral radiculopathy would explain the weakness in hand/wrist flexors, it would not explain the weakness in the hips. Furthermore, the raised CK is suggestive of a myopathy.

Limb-girdle muscular dystrophy is incorrect. Although this remains within the differential diagnosis, the pattern of muscle weakness is more in keeping with inclusion body myositis. Limb-girdle muscular dystrophy would classically affect the shoulder and hips, and weakness in the hands and wrists would be unusual.

Myotonic dystrophy is incorrect. The pattern of involvement, in this case, is not typical of myotonic dystrophy. Myotonic dystrophy classically presents with facial weakness and distal muscle weakness. Furthermore, the absence of any other hallmark feature of this condition (e.g. frontal balding, and grip myotonia) makes it a less likely diagnosis.

Polymyositis is incorrect. The pattern of involvement, in this case, is not typical of polymyositis. The hallmark of polymyositis is a progressive disease with symmetric involvement of the proximal girdle muscles (shoulder and pelvis muscles) and neck flexors. Myalgia is also common.

Discuss (3) Improve

Next question

Inclusion body myositis ★

Overview
- cause of myopathy
- associated with cytoplasmic inclusions on muscle biopsy

Features
- typically affects older males
- can affect both proximal and distal muscles
- characteristically affects quadriceps and finger/wrist flexors are usually more severely affected than extensor counterparts

+ Q 123

Next question

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INCLUSION BODY MYOPATHY

Inclusion body myositis

Osmosis - YouTube 👍 7 👎 2

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Score: **22%**

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A 32 year-old accountant presents to the neurology clinic.

For the last year he has been troubled by severe headaches. These occur in the morning, sometimes waking him from sleep. There is a sudden onset of stabbing pain behind the left eye, at the onset of which he feels the need to pace around the room, pounding his head in an effort to ease the pain. During the attacks his left eye is watery and his nose his congested. On more than one occasion he has noticed that his right pupil appears enlarged during the attacks. He tells you very clearly that the pain is so severe that at times during the attacks he has considered jumping out of the top floor of the office building where he works. Each attack lasts one or two hours, and between the attacks he feels fine.

He shows you a detailed headache dairy in which he has documented every attack over the last year. For example, in January he had a period of nine days in which attacks occurred several times daily, but in February and March he was headache free. During April he developed severe headaches which occurred almost every day until June. He then had another period of relief until August, and so on.

He has no other past medical history and no allergies.

Examination is unremarkable.

What is the best treatment for prophylaxis of this man's headaches?

○ 100% oxygen

○ Verapamil

○ Sumatriptan

○ Propranolol

○ Lithium

Submit answer

Reference ranges

A 32 year-old accountant presents to the neurology clinic.

For the last year he has been troubled by severe headaches. These occur in the morning, sometimes waking him from sleep. There is a sudden onset of stabbing pain behind the left eye, at the onset of which he feels the need to pace around the room, pounding his head in an effort to ease the pain. During the attacks his left eye is watery and his nose his congested. On more than one occasion he has noticed that his right pupil appears enlarged during the attacks. He tells you very clearly that the pain is so severe that at times during the attacks he has considered jumping out of the top floor of the office building where he works. Each attack lasts one or two hours, and between the attacks he feels fine.

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He has no other past medical history and no allergies.

Examination is unremarkable.

What is the best treatment for prophylaxis of this man's headaches?

100% oxygen	6%
Verapamil	82%
Sumatriptan	4%
Propranolol	7%
Lithium	0%

Verapamil is used for long-term prophylaxis of cluster headaches

Important for me   Less important

This is a classic description of episodic cluster headache.

Interestingly, the patient mentioned that his right pupil was enlarged, but most likely he is describing ipsilateral Horner's syndrome, which may occur during cluster attacks.

Verapamil is the established first-line agent for the prophylaxis of episodic and chronic cluster headache.

100% oxygen is the first-line treatment for acute attacks of cluster headache.

Sumatriptan is commonly used for acute attacks of cluster headache and migraine, often subcutaneously.

Propranolol is a first-line drug for the prophylaxis of migraine.

Lithium is a second-line agent for the prophylaxis of cluster headache.

Discuss (6) | Improve

Next question >

Cluster headache

Cluster headaches are known to be one of the most painful conditions that patients can have the misfortune to suffer. The name relates to the pattern of the headaches - they typically occur in clusters lasting several weeks, with the clusters themselves typically once a year.

Cluster headaches are more common in men (3:1) and smokers. Alcohol may trigger an attack and there also appears to be a relation to nocturnal sleep.

Features
-intense sharp, stabbing pain around one eye

  - pain typical occurs once or twice a day, each episode lasting 15 mins - 2 hours
  - the patient is restless and agitated during an attack due to the severity

- clusters typically last 4-12 weeks
- accompanied by redness, lacrimation, lid swelling
- nasal stuffiness
- miosis and ptosis in a minority

Investigations

- most patients will have neuroimaging - underlying brain lesions are sometimes found even if the clinical symptoms are typical for cluster headache
- MRI with gadolinium contrast is the investigation of choice

Management

- NICE recommend seeking specialist advice from a neurologist if a patient develops cluster headaches
- acute
    - 100% oxygen (80% response rate within 15 minutes)
    - subcutaneous triptan (75% response rate within 15 minutes)
- prophylaxis
    - verapamil is the drug of choice
    - there is also some evidence to support a tapering dose of prednisolone

Some neurologists use the term trigeminal autonomic cephalgia to group a number of conditions including cluster headache, paroxysmal hemicrania and short-lived unilateral neuralgiform headache with conjunctival injection and tearing (SUNCT). It is recommended such patients are referred for specialist assessment as specific treatment may be required, for example it is known paroxysmal hemicrania responds very well to indomethacin

AI + Q 123

Next question >

B I A

Textbooks

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Extended textbook

Links

NICE 👍 17 👎 10

2012 Headache guidelines

Suggest link

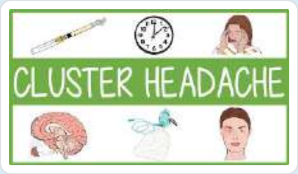
Report broken link

Media

SEVERE SYMPTOMS
HIGH FLOW 100% OXYGEN
CLUSTER HEADACHES
PREVENTION
SUBCUT TRIPTANS
HOME OXYGEN

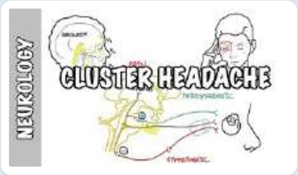
Cluster headaches

Zero To Finals - YouTube 👍 0 👎 0


CLUSTER HEADACHE

Cluster headache

Townsend Teaching - YouTube 👍 0 👎 0


NEUROLOGY
CLUSTER HEADACHE

Cluster Headaches - symptoms, pathophysiology, treatment

Armando Hasudungan - YouTube 👍 1 👎 1

Report broken media

Score: **22%**

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A 53-year-old lady was admitted to the medical admission unit having presented to the Emergency Department with a severe headache which had developed over the last five days. She described the headache as a continual dull pain across the front of her head and was significantly worsened when she sat up as well as when she coughed or strained. It was alleviated only when she lied down in a dark room; paracetamol 1g QDS and ibuprofen 400mg TDS did not provide any relief. She denied the presence of nausea or vomiting, her vision was not affected and other than feeling tired she did not complain of any other symptoms. Her past medical history was comprised of non-classical migraines and hypothyroidism for which she was prescribed levothyroxine 175mcg OD. Seven days ago she was admitted with a headache, fever and photophobia; review of her medical notes revealed the following results from the previous admission:

Hb	142 g/l
Platelets	326 * 10^9/l
WBC	11.2 * 10^9/l

Glucose	5.4 mmol/l
ESR	32 mm/hr
CRP	46 mg/l
PTT	12.2 (NR 12-14s)
APTT	44s (NR 300-46s)
Fibrinogen	5.2 (NR 2-4 g/l)

Urine MCS: NAD
Blood MCS: NAD

CT Head: normal intracranial appearances; no space occupying lesion or haemorrhage identified

Lumbar puncture:

Protein	0.3g (NR 0.2-0.4 g/L)
Glucose	3.6 mmol/l
WCC	8 (<5/mm3)
Opening pressure	15 (NR 10-20 cmH2O)
MCS	NAD

Examination revealed the presence of a female in a dark room lying flat. Her temperature was

36.6ºC, heart rate 77bpm, respiratory rate 16/min and blood pressure 136/78 mmHg. Examination her cardiovascular and respiratory systems were unremarkable. Examination of her neurological system revealed the presence of normal functioning cranial nerves 2-12 with unremarkable fundoscopy; there was no neck stiffness or photophobia objectively. There was no neck stiffness and Kernig's sign was negative. Examination of her peripheral neurological system was unremarkable.

What is the single next best management option?

- ○ Organise urgent cranial MR venogram scan ×
- ○ Organise urgent cranial MRI scan ×
- ○ Commence therapy with sumatriptan ×
- ○ Administer epidural blood patch ×
- ○ Send CSF for xanthochromia analysis ×

Submit answer

Reference ranges ﹀

Score: **24.1%**

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What is the single next best management option?

Organise urgent cranial MR venogram scan	36%
Organise urgent cranial MRI scan	5%
Commence therapy with sumatriptan	14%
Administer epidural blood patch	41%
Send CSF for xanthochromia analysis	4%

This lady has developed post-lumbar puncture headache (PLPH), a common complication of a lumbar puncture and is thought to be caused by excess leakage of cerebrovascular fluid causing a relatively low intracranial pressure and is aggravated significantly when assuming a non-supine posture. There are of course many other diagnoses that need to be considered, including meningitis, migraine, cortical vein thrombosis, intracerebral haemorrhage and cluster headaches. In most instances however the diagnosis of PLPH can be made clinically. Patients frequently believe the cause of their headaches is due to migraine and therefore report that avoiding bright light alleviates their headache when in fact it is lying down which is alleviating the headache. Management is conservative, including administering analgesia and sufficient fluids. If this fails to resolve the headache the gold standard management is an epidural blood patch. See http://www.paulchristomd.com/wp-content/themes/flowhub/pdf/PostduralPunctureHA.pdf for a comprehensive review.

Discuss (15) | Improve

Next question >

Post-lumbar puncture headache

Headache following lumbar puncture (LP) occurs in approximately one-third of patients. The pathophysiology of is unclear but may relate to a 'leak' of CSF following dural puncture. Post-LP headaches are more common in young females with a low body mass index

Typical features

- usually develops within 24-48 hours following LP but may occur up to one week later
- may last several days
- worsens with upright position
- improves with recumbent position

Factors which may contribute to headache	**Factors which do not contribute to headache**
• Needle type: conventional 'cutting' needles increase the risk compared to pencil point (atraumatic) needles • Increased needle size • Direction of bevel • Not replacing the stylet • Increased number of LP attempts	• Increased volume of CSF removed • Bed rest following procedure • Increased fluid intake post procedure • Opening pressure of CSF • Position of patient

Management

- supportive initially (analgesia, rest)
- if pain continues for more than 72 hours then specific treatment is indicated, to prevent subdural haematoma
- treatment options include: blood patch, epidural saline and intravenous caffeine

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **22%**

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Question 33 of 409 ✓

A 45-year-old woman is admitted to the neurology inpatient unit with a 3-day history of fever and headache. To exclude meningoencephalitis, a lumbar puncture is performed. Following the procedure, she developed a severe headache that was initially attributed to a post-lumbar puncture headache; however, it persists despite lying flat, adequate hydration, and analgesia. Subsequent computed tomography (CT) of the brain reveals a subdural haematoma, although the CT brain scan at admission was normal.

What could have prevented this patient's condition?

Fall risk assessment	10%
Increased volume of CSF removed	2%
Intravenous mannitol	3%
Use of cutting needle for LP	18%
Use of pencil-point needle for LP	66%

The use of pencil point (atraumatic) needles reduces the risk of post-lumbar puncture headache

Important for me Less important

The correct response is **use of pencil-point needle for LP** because the scenario depicts a patient who has developed a subdural haematoma after a lumbar puncture. Utilisation of an atraumatic pencil-point needle can mitigate this risk. There is an increased likelihood of subdural haematoma formation with larger gauge spinal needles. Additionally, an epidural blood patch can also reduce the incidence of post-lumbar puncture headaches. Notably, the headache associated with a subdural haematoma does not decrease with recumbency, intravenous hydration, or analgesics. Management involves an autologous blood patch, epidural saline or intravenous caffeine administration. Craniotomy and surgical decompression are seldom necessitated.

In this case, the subdural haematoma is attributable to the lumbar puncture rather than trauma. A **fall risk assessment** is imperative for preventing traumatic subdural haematomas and is crucial in managing patients with diminished consciousness levels; however, it is not pertinent to this situation.

The volume of cerebrospinal fluid (CSF) extracted does not contribute to post-lumbar puncture headache or subdural haematoma formation. Consequently, **increased volume of CSF removed** would not have prevented this patient's condition.

Intravenous mannitol is an incorrect choice as it plays no role in the prophylaxis against post-lumbar puncture subdural haematoma.

Since conventional spinal needles elevate the risk of post-lumbar puncture complications, opting for **use of cutting needle for LP** would not be effective in averting either post-lumbar puncture headaches or haematomas.

👍 👎 Discuss (2) Improve

Next question >

Post-lumbar puncture headache ★

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• Needle type: conventional 'cutting' needles increase the risk compared to pencil point (atraumatic) needles • Increased needle size • Direction of bevel • Not replacing the stylet • Increased number of LP attempts	• Increased volume of CSF removed • Bed rest following procedure • Increased fluid intake post procedure • Opening pressure of CSF • Position of patient

Management
- supportive initially (analgesia, rest)

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123

Next question >

Textbooks

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Score: **22%**

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A 19-year-old male presents to outpatient clinic with a 3-month history of worsening speech slurring. He finds it extremely embarrassing and is made fun of by his friends in college, who thinks he is turning up to school drunk. He is also finding that he is having difficulty with excessive drooling. Over the past 6 weeks, his has also noted increasing weakness in lifting his right arm. He has no other past medical history, is a non-smoker and drinks alcohol only socially.

On examination, his cranial nerves demonstrate weakness in raising his eyebrows, puffing his cheeks and in pursing his lips. He displays significant scapula winging bilaterally with mild wasting of his right deltoid muscle. The musculature of his left arm is normal. The power of his right shoulder abduction and adduction is 3/5, with 5/5 in all other movements. Reflexes are all present with a normal sensory examination.

His blood tests are as follows:

Hb	144 g/l
Platelets	388 * 10^9/l
WBC	5.8 * 10^9/l

Na^+	139 mmol/l
K^+	4.7 mmol/l
Urea	4.6 mmol/l
Creatinine	55 µmol/l
CRP	2mg/l
Creatine kinase	150 IU/l (50-335)
TSH	2.3 mu/l
HIV	negative

What is the most likely diagnosis?

- ○ Myoclonic epilepsy with ragged red fibres (MERRF) ×
- ○ Myotonic dystrophy type 1 ×
- ○ Polymyositis ×
- ○ Limb girdle muscular dystrophy ×

○ Facioscapulohumeral dystrophy ×

Submit answer

Reference ranges ⌵

Score: **24.1%**

1	✗
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CRP	2mg/l
Creatine kinase	150 IU/l (50-335)
TSH	2.3 mu/l
HIV	negative

What is the most likely diagnosis?

Myoclonic epilepsy with ragged red fibres (MERRF)	3%
Myotonic dystrophy type 1	6%
Polymyositis	1%
Limb girdle muscular dystrophy	2%

Facioscapulohumeral dystrophy 89%

Facioscapulohumeral muscular dystrophy: facial weakness (e.g. difficulty closing eyes, smiling, blowing), winged scapula and weakness of the shoulderm upper arm and hip girdle muscles

Important for me Less important

This is a difficult question regarding the cause of a myopathy, onset at late adolescence and progresses. Firstly, the normal CK, CRP and lack of systemic features point against a systemic inflammatory condition such as polymyositis. Although also involving proximal muscles and presenting typically without cutaneous manifestations, polymyositis is a multisystem disorder with cardiac, pulmonary features and oesophageal involvement.

Similarly, although myotonic dystrophy type 1 patients also present from age of 20 onwards, it is another multisystem disorder with cardiac structural and conduction abnormalities, respiratory muscle involvement, gastrointestinal symptoms, testicular failure and insulin resistance, amongst a large number of clinical features. Classically, patients with myotonic dystrophy display myotonia, a slowing of relaxation following normal muscular contraction.

An absence of myoclonus makes MERRF less likely. It is a mitochondrial disorder typically presenting with myopathy, peripheral neuropathy and frequently, ataxia, Wolff-Parkinson-White syndrome and bilateral deafness. The final assessment of the type of muscular dystrophy relies simply on the muscles involved: in this case, muscles involved are facial and upper limb girdle muscles. Be aware that muscular dystrophies such as facioscapulohumeral dystrophy can initially present asymmetrically before progressing to bilateral involvement.

Discuss (3) Improve

Next question

Facioscapulohumeral muscular dystrophy

Facioscapulohumeral muscular dystrophy (FSHMD) is an autosomal dominant form of muscular dystrophy. As the name suggests it typically affects the face, scapula and upper arms first. Symptoms typically present by the age of 20 years.

Features
- facial muscles are involved first - difficulty closing eyes, smiling, blowing etc
- weakness of the shoulder and upper arm muscles
- abnormal prominence of the borders of the shoulder blades - 'winging'

- lower limb: hip girdle weakness, foot drop

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **22%**

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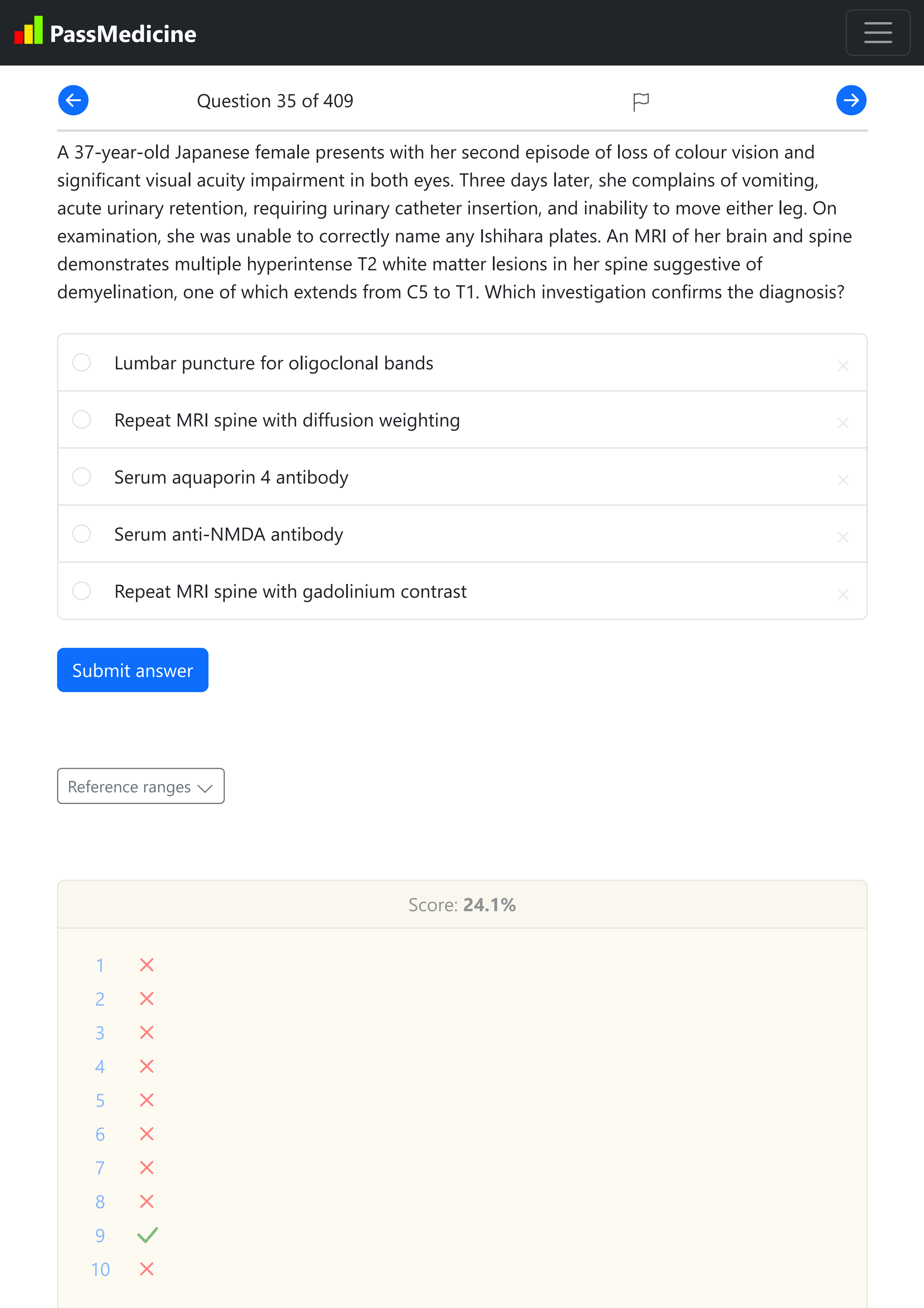

Question 35 of 409

A 37-year-old Japanese female presents with her second episode of loss of colour vision and significant visual acuity impairment in both eyes. Three days later, she complains of vomiting, acute urinary retention, requiring urinary catheter insertion, and inability to move either leg. On examination, she was unable to correctly name any Ishihara plates. An MRI of her brain and spine demonstrates multiple hyperintense T2 white matter lesions in her spine suggestive of demyelination, one of which extends from C5 to T1. Which investigation confirms the diagnosis?

- ○ Lumbar puncture for oligoclonal bands
- ○ Repeat MRI spine with diffusion weighting
- ○ Serum aquaporin 4 antibody
- ○ Serum anti-NMDA antibody
- ○ Repeat MRI spine with gadolinium contrast

Score: **24.1%**

1 ✗
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Question 35 of 409 ✗

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Lumbar puncture for oligoclonal bands	16%
Repeat MRI spine with diffusion weighting	7%
Serum aquaporin 4 antibody	59%
Serum anti-NMDA antibody	7%
Repeat MRI spine with gadolinium contrast	10%

Neuromyelitis optica is associated with aquaporin 4 positive serum antibody

Important for me Less important

The patient has presented with optic neuritis, myelitis and vomiting, strongly suggestive of a diagnosis of neuromyelitis optica (NMO), also known as Devics disease.

In this case, CSF oligoclonal bands neither confirms nor excludes a diagnosis MRI spine with DWI would demonstrate acute ischaemic infarcts if a spinal stroke was suspected. Gadolinium enhanced imaging does not add to the diagnostic criteria for NMO. Anti-NMDA antibody is a serum autoantibody for a subtype of autoimmune encephalitis.

Discuss (3) Improve

Next question ›

Neuromyelitis optica

Neuromyelitis optica (NMO) is a monophasic or relapsing-remitting demyelinating CNS disorder Although previously thought to be a variant of multiple sclerosis, it is now recognised to be a distinct disease, particularly prevalent in Asian populations. It typically involves the optic nerves and cervical spine, with imaging of the brain frequently normal. Vomiting is also a common presenting complaint.

Diagnosis requires bilateral optic neuritis, myelitis and 2 of the following 3 criteria:

- 1. Spinal cord lesion involving 3 or more spinal levels
- 2. Initially normal MRI brain
- 3. Aquaporin 4 positive serum antibody

Management

- immunosuppressant e.g. with anti-CD20 agent rituximab)

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **22%**

1 ✗
2 ✗
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A 72-year-old man is referred to the oncology team after presenting to the emergency department. The patient reports progressive lower thoracic back pain over the past three weeks. The pain had now reached an intensity that had prevented him from sleeping during the past two nights, despite over the counter analgesics. The patient reported that coughing caused a severe exacerbation of the pain, but denied radiation of the pain. The patient had previously suffered mild lower back pain after exercise, but could recall no episodes that compared in intensity to his present symptoms.

The patient's wife reported she felt the patient had been unsteady on his feet over the past few days, and reported that he had nearly fallen on several occasions. The patient denied any history of sphincter disturbance or sensory symptoms. On direct questioning, the patient revealed that he had been troubled by a severe cough over the past two months and had also lost 7 kilograms in weight. He denied any history of fevers or night sweats.

The patient had an unremarkable past medical history and took no regular medications. He was a retired builder, who had stopped smoking when he took retirement seven years previously.

Peripheral nerve examination of upper limbs: normal tone, normal power, normal coordination, normal sensation, reflexes intact and symmetrical.

Peripheral nerve examination of lower limbs: tone generally increased bilaterally with 5 beats of clonus in left lower limb; power generally slightly reduced (4+/5) bilaterally, although patient limited by back pain; normal coordination; light touch sensation reduced to mid-point between symphysis pubis and umbilicus; knee and ankle jerks brisk bilaterally, plantar response up-going in left lower limb.

Respiratory examination: borderline clubbing of fingers; reduced resonance and air entry in left upper zone.

Musculoskeletal examination: point tenderness in midline of back around level of T11 / T12 vertebrae.

What is the appropriate investigation of patient's back pain?

○ MRI whole-spine within 24 hours

○ MRI lumbar-thoracic spine within 24 hours

○ CT lumbar-thoracic spine within 24 hours

○ MRI whole-spine within 7 days ×

○ CT myelography within 7 days ×

Submit answer

Reference ranges ⌄

Score: **24.1%**

1	✗
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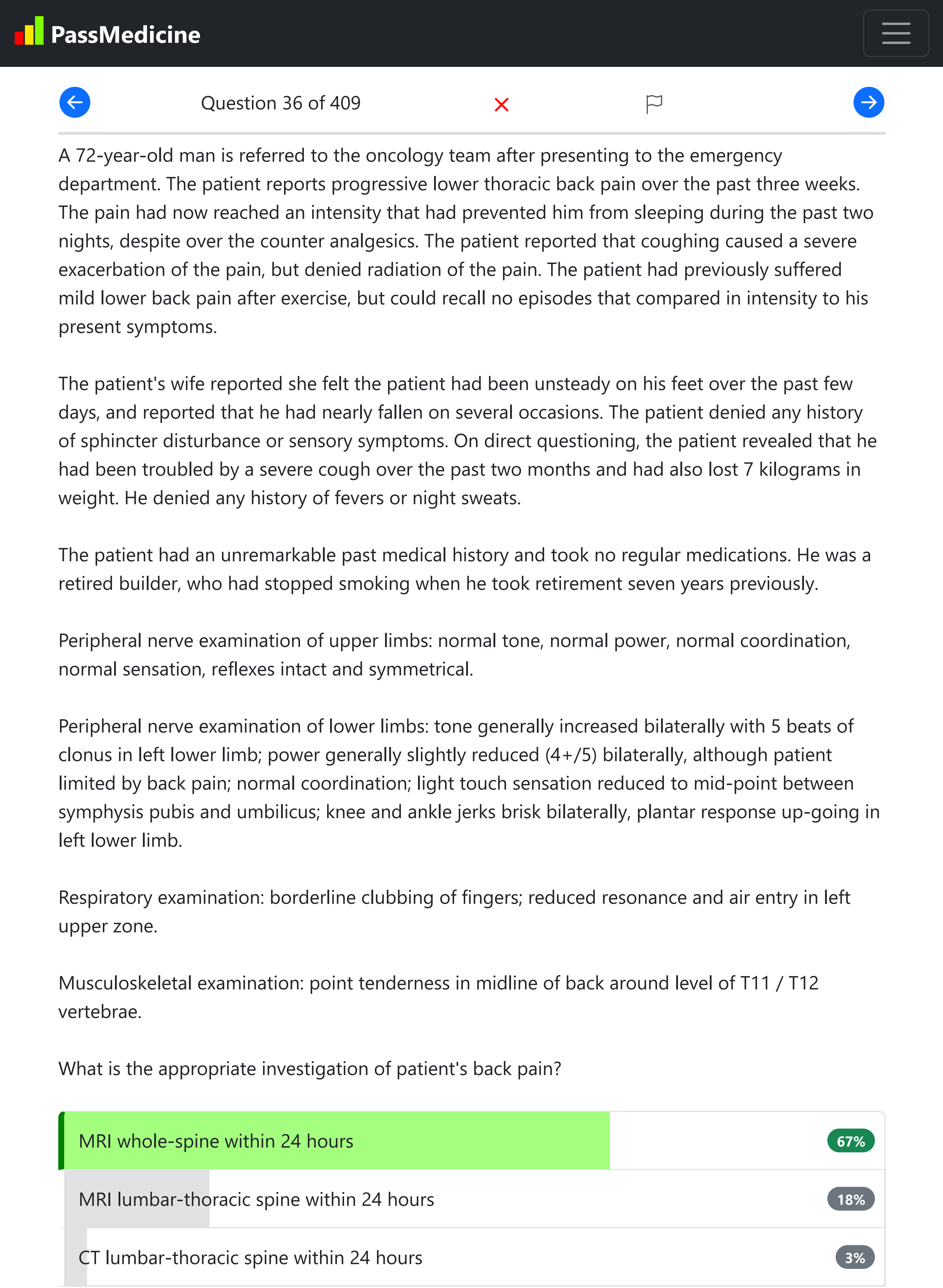

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Musculoskeletal examination: point tenderness in midline of back around level of T11 / T12 vertebrae.

What is the appropriate investigation of patient's back pain?

MRI whole-spine within 24 hours 67%

MRI lumbar-thoracic spine within 24 hours 18%

CT lumbar-thoracic spine within 24 hours 3%

MRI whole-spine within 7 days 12%

CT myelography within 7 days 1%

The presentation of this patient raises strong concerns about metastatic spinal cord compression as the cause for his symptoms. In the majority of cases, the condition is secondary to bony metastasis to the vertebral spine with collapse and extra-dural compression of the spinal cord.

In around 20 % of cases of metastatic spinal cord compression there is no pre-existing diagnosis of malignancy. While this is true in the case of this patient, there are several aspects of the above assessment that are highly suspicious of a primary lung cancer.

In 95 % of cases, back pain is the most common first symptom, and can be present for up to two months before other signs occur. The patient's history of progressive pain, exacerbated by coughing, and associated with midline tenderness is typical of pain secondary to a localised lesion; however, pain can also present in a radicular pattern due to nerve root compression.

The normal upper limb examination and presence of upper motor neurone signs in the lower limbs with a sensory level, would localise the lesion to the thoracic spinal cord, and correlates with the vertebral tenderness. Bladder and bowel symptoms are often a late feature of metastatic spinal cord compression, so the absence of autonomic dysfunction does not exclude the diagnosis.

United Kingdom NICE guidance is for MRI whole-spine to be performed within 24 hours in individuals with both pain and neurological symptoms suggestive of metastatic spinal cord compression. In the presence of pain without neurological symptoms, MRI whole-spine should be performed within 7 days. It is necessary to image the entire vertebral column, as multi-level involvement is present in 30-50 % of cases.

CT scanning is often used to aid surgical or radiotherapy treatment planning. CT myelography is occasionally used in individuals with contra-indications to MRI scanning.

Al-Qurainy, Collis E. Metastatic spinal cord compression: diagnosis and management. BMJ 2016;353:i2539.

Discuss (4) | Improve

Next question

Spinal cord lesions ★

The diagram belows shows cross-section view of the spinal cord:

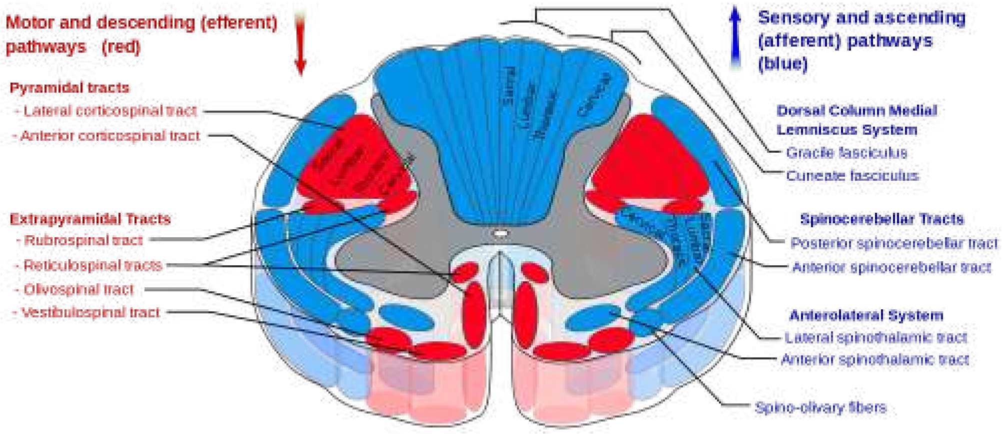

Motor and descending (efferent) pathways (red)
Pyramidal tracts
- Lateral corticospinal tract
- Anterior corticospinal tract
Extrapyramidal Tracts
- Rubrospinal tract
- Reticulospinal tracts
- Olivospinal tract
- Vestibulospinal tract
Sacral
Lumbar
Thoracic
Cervical
Sensory and ascending (afferent) pathways (blue)
Dorsal Column Medial Lemniscus System
Gracile fasciculus
Cuneate fasciculus
Spinocerebellar Tracts
Posterior spinocerebellar tract
Anterior spinocerebellar tract
Anterolateral System
Lateral spinothalamic tract
Anterior spinothalamic tract
Spino-olivary fibers

Image sourced from Wikipedia

Motor lesions

Amyotrophic lateral sclerosis (motor neuron disease)
- affects both upper (corticospinal tracts) and lower motor neurons
- results in a combination of upper and lower motor neuron signs

Poliomyelitis
- affects anterior horns resulting in lower motor neuron signs

Combined motor and sensory lesions

Disorder	Tracts affected	Clinical notes
Brown-Sequard syndrome (spinal cord hemisection)	1. Lateral corticospinal tract 2. Dorsal columns 3. Lateral spinothalamic tract	1. Ipsilateral spastic paresis below lesion 2. Ipsilateral loss of proprioception and vibration sensation 3. Contralateral loss of pain and temperature sensation
Subacute combined degeneration of the spinal cord (vitamin B12 & E deficiency)	1. Lateral corticospinal tracts 2. Dorsal columns 3. Spinocerebellar tracts	1. Bilateral spastic paresis 2. Bilateral loss of proprioception and vibration sensation 3. Bilateral limb ataxia
Friedrich's ataxia	Same as subacute combined degeneration of the spinal cord (see above)	Same as subacute combined degeneration of the spinal cord (see above)

Disorder	Tracts affected	Clinical notes
		In addition cerebellar ataxia → other features e.g. intention tremor
Anterior spinal artery occlusion	1. Lateral corticospinal tracts 2. Lateral spinothalamic tracts	1. Bilateral spastic paresis 2. Bilateral loss of pain and temperature sensation
Syringomyelia	1. Ventral horns 2. Lateral spinothalamic tract	1. Flacid paresis (typically affecting the intrinsic hand muscles) 2. Loss of pain and temperature sensation
Multiple sclerosis	Asymmetrical, varying spinal tracts involved	Combination of motor, sensory and ataxia symptoms

Sensory lesions

Disorder	Tracts affected	Clinical notes
Neurosyphilis (tabes dorsalis)	1. Dorsal columns	1. Loss of proprioception and vibration sensation

AI + Q 123

Next question ›

Textbooks

High-yield textbook

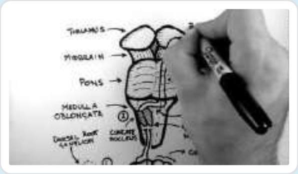
Extended textbook

Media

ANATOMY
SPINAL CORD
CLINICAL ANATOMY

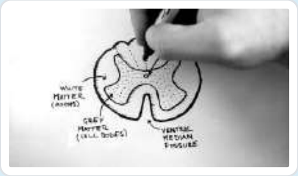
Spinal Cord - Clinical Anatomy and Physiology (dermatomes, blood supply, shingles, lumbar puncture)

Armando Hasudungan - YouTube 👍 7 👎 0


THALAMUS
MIDBRAIN
PONS
MEDULLA OBLONGATA
DORSAL ROOT GANGLION
CUNEATE NUCLEUS

Spinal Pathways 2 - Dorsal Column - Medial Lemniscus Pathway

Handwritten Tutorials - YouTube 👍 4 👎 0


WHITE MATTER (AXONS)
GREY MATTER (CELL BODIES)
VENTRAL MEDIAN FISSURE

Spinal Pathways 1 - Spinal Cord Anatomy and Organisation

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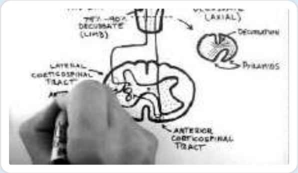
DECUSSATE (LIMB)
(AXIAL)
DECUSSATION
PYRAMIDS
LATERAL CORTICOSPINAL TRACT
ANTERIOR CORTICOSPINAL TRACT

Spinal Pathways 4 - Corticospinal Tract

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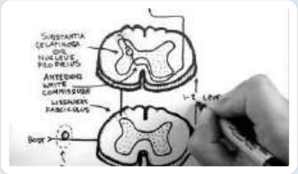
SUBSTANTIA GELATINOSA OR NUCLEUS PROPRIUS
ANTERIOR WHITE COMMISSURE
LISSAUER'S FASCICULUS
BODY

Spinal Pathways 3 - Spinothalamic Tract

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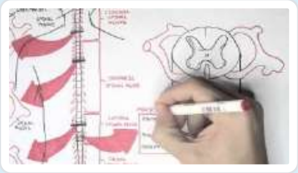

Neurology - Spinal Cord Introduction

Armando Hasudungan - YouTube 👍 0 👎 0

Report broken media

Score: **22%**

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A 67-year-old male presents to your neurology clinic with his wife, reporting a 3-month history of worsening vision in his left eye. He is very reluctant to seek any medical help but says that a number of factors have finally convinced him to attend.

Over the past 2 months, he reports a constant and worsening headache, worse at night and with coughing. He has occasionally felt extremely nauseated and vomited on a number of occasions, at least twice waking him from sleep. His wife also notes significant personality change: her husband is a retired farmer who prides him on being a stoic man. She notes that he has become increasingly emotional and occasionally aggressive, which she puts down however to struggling with his symptoms. Prior to these symptoms starting 3 months ago, he has always been fit and well with no past medical or drug history.

On examination, you note a left relative afferent papillary defect with equally sized pupils. Visual acuity in the left eye is 6/60 and 6/9 on the right. Testing of colour vision with Ishihara plates demonstrates 0/17 on the left and 17/17 on the right. A central scotoma is found in the left eye. Fundoscopy of the left eye reveals a pale optic disc with poor vasculature while the right appears swollen. A full range of painless eye movements is demonstrated. The remaining examination of the cranial nerves, upper and lower limbs are unremarkable. Drops of blood are unremarkable, a MRI head is awaited.

What is the most likely diagnosis?

- ○ Foster-Kennedy syndrome
- ○ Idiopathic intracranial hypertension (IIH)
- ○ Multiple sclerosis (MS)
- ○ Frontotemporal dementia
- ○ Age related macular degeneration (ARMD)

Submit answer

Reference ranges

Score: **24.1%**

1	✗
2	✗
3	✗
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6	✗
7	✗
8	✗
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A 67-year-old male presents to your neurology clinic with his wife, reporting a 3-month history of worsening vision in his left eye. He is very reluctant to seek any medical help but says that a number of factors have finally convinced him to attend.

Over the past 2 months, he reports a constant and worsening headache, worse at night and with coughing. He has occasionally felt extremely nauseated and vomited on a number of occasions, at least twice waking him from sleep. His wife also notes significant personality change: her husband is a retired farmer who prides him on being a stoic man. She notes that he has become increasingly emotional and occasionally aggressive, which she puts down however to struggling with his symptoms. Prior to these symptoms starting 3 months ago, he has always been fit and well with no past medical or drug history.

On examination, you note a left relative afferent papillary defect with equally sized pupils. Visual acuity in the left eye is 6/60 and 6/9 on the right. Testing of colour vision with Ishihara plates demonstrates 0/17 on the left and 17/17 on the right. A central scotoma is found in the left eye. Fundoscopy of the left eye reveals a pale optic disc with poor vasculature while the right appears swollen. A full range of painless eye movements is demonstrated. The remaining examination of the cranial nerves, upper and lower limbs are unremarkable. Drops of blood are unremarkable, a MRI head is awaited.

What is the most likely diagnosis?

Foster-Kennedy syndrome	69%
Idiopathic intracranial hypertension (IIH)	11%
Multiple sclerosis (MS)	9%
Frontotemporal dementia	7%
Age related macular degeneration (ARMD)	4%

This is a difficult question requiring recognition of a high-pressure headache, left optic atrophy and contralateral swollen disc secondary to papilloedema. The combination of an ipsilateral optic atrophy and contralateral papilloedema is an MRCP favourite: Foster-Kennedy syndrome, typically caused by a frontal lobe mass, normally a meningioma, which compressing on the ipsilateral optic nerve and olfactory nerve, while increasing intracranial pressure for the contralateral optic nerve sheath.

Idiopathic intracranial hypertension may present asymmetrically but typically produces bilateral disc swelling. It is conceivable the patient has multiple sclerosis with optic atrophy as a sequelae of left optic neuritis, followed by a second episode of contralateral optic neuritis presenting as papillitis, inflammation of the optic nerve head. This is unlikely with only one episode of visual dysfunction described with an insidious progressive onset.

Frontotemporal dementia may explain the behavioural changes but does not account for visual symptoms.

Lastly, age-related macular degeneration can mimic papillitis on fundoscopy, with deposition of drusen making the optic disc 'look' swollen. However, the deposition of these retinal cell byproducts is generally more diffuse across the retina, with wet ARMD also displaying abnormal retinal vasculature. While retinal atrophy may be present, it does not typically produce optic atrophy.

Discuss (6) | Improve

Next question ›

Foster-Kennedy syndrome ★

Foster-Kennedy syndrome describes a series of symptoms and signs associated with frontal lobe lesions.

Features
- optic atrophy in the ipsilateral eye
- papilloedema in the contralateral eye
- central scotoma in the ipsilateral eye
- anosmia

AI + Q 123

Next question ›

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Score: **22%**

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Back to top

A 45 year old previously fit and well man presents to the emergency department with worsening leg weakness. He is in full time employment as a brick layer and is normally very active. Over the last 24 hours he has started dragging his feet and feels unsteady when walking, describing his gait 'like a drunk man'. On questioning he also describes increasing difficulty passing urine and has not had the sensation to empty his bladder for the past eight hours. He denies any preceding trauma, recent viral illness or similar previous symptoms in the past.

On examination he has normal muscle bulk and no fasciculations. There is symmetrical lower limb flaccid paralysis to the hips, with symmetrical hyporeflexia. He has a sensory level to T10 and is in urinary retention. Examination of the upper limbs and cranial nerves is entirely normal.
Which of the following would be the most useful initial investigation?

- ○ Aquaporin 4 antibodies ×
- ○ Lumbar puncture ×
- ○ MRI spinal cord ×
- ○ MRI brain ×
- ○ HIV serology ×

Submit answer

Reference ranges

Score: **24.1%**

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On examination he has normal muscle bulk and no fasciculations. There is symmetrical lower limb flaccid paralysis to the hips, with symmetrical hyporeflexia. He has a sensory level to T10 and is in urinary retention. Examination of the upper limbs and cranial nerves is entirely normal.
Which of the following would be the most useful initial investigation?

Aquaporin 4 antibodies	8%
Lumbar puncture	7%
MRI spinal cord	79%
MRI brain	3%
HIV serology	2%

This man presents with signs and symptoms of acute transverse myelitis. The underlying aetiological process can be diverse; it can be associated with underlying autoimmune systemic response, infectious cause or demyelinating process such as neuromyelitis optica.

The presence of a sensory level suggests a spinal cord lesion, as such a CT or MRI Brain would not be the first investigation. The most important initial approach in a patient presenting with acute disturbance of motor, sensory or autonomic function (and a sensory level) is to rule out a compressive cord lesion; an MRI spine is therefore an essential first step. Although HIV serology would be important in this patient, as aforementioned, the most important step is to exclude a compressive lesion.

Discuss (5) Improve

Next question

Transverse myelitis ★

Causes of transverse myelitis

- viral infections: varicella-zoster, herpes simplex, cytomegalovirus, Epstein-Barr, influenza, echovirus, human immunodeficiency virus
- bacterial infections: syphilis, Lyme disease
- post-infectious (immune mediated)
- first symptom of multiple sclerosis (MS) or neuromyelitis optica (NMO)

＋ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

TRANSVERSE
MYELITIS
Osmosis.org

Transverse myelitis

Osmosis - YouTube 👍 9 👎 0

Report broken media

Score: **22%**

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Question 39 of 409

You are called to see a 67-year-old lady by psychiatry who is asking for your opinion on management. Over the past 3 weeks, her husband has become increasingly concerned by her behaviour. She has become suspicious of him and accused him of stealing 'my true husband'. Initially, the lady would avoid her husband and refuse to eat food that he had prepared. However, today she threatened him with a knife and the police had to be called.

The psychiatry doctor is concerned as he notes that she was admitted to hospital 5 weeks ago and was treated for a suspected urinary tract infection with intravenous antibiotics. Subsequent testing showed the pathogen to be an extended-spectrum beta-lactamase producing bacteria. She currently has a temperature of 38.7 degrees heart rate 105bpm regular RR 18 Sats 99% on room air. Her husband notes that she has been spending more time in the toilet over the past 3 weeks but is unsure if this is due to her paranoia.

Her husband describes an episode 30 years ago where she required antidepressants, antipsychotics and ECT after a close family bereavement. She is otherwise well and has no history of cognitive problems. Her husband states that there is a strong history of mental health problems in her family but he is unable to be more specific.

When you speak to her she appears to be confused and scores 19/30 on the Mini Mental State Examination. She can point to, name and recognise her husband and can also pick him out from pictures. However, she tells you that the man standing next to her is not her husband but a lookalike who has replaced him. Despite all your best efforts to show evidence to the contrary, she cannot be persuaded to change her opinion.

Which of the following below would best describe her presentation?

- ○ Delirium causing Cotard syndrome
- ○ Delirium causing Fregoli syndrome
- ○ Delirium causing Capgras syndrome
- ○ Late onset schozphrenia causing Capgras syndrome
- ○ Atypical severe depression

Submit answer

Reference ranges

Score: **24.1%**

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Which of the following below would best describe her presentation?

Delirium causing Cotard syndrome	12%
Delirium causing Fregoli syndrome	12%
Delirium causing Capgras syndrome	66%
Late onset schozphrenia causing Capgras syndrome	9%
Atypical severe depression	1%

Firstly, this woman has features that suggest delirium having a factor to play in her presentation: fever, acute cognitive impairment, infective symptoms, recent treatment for a highly resistant infection. Late-onset schizophrenia is very rare and when it does occur has no association with family history of mental health problems or personal history of depression. In addition, the duration of symptoms is probably too rapid for late onset schizophrenia.

Secondly, the woman is describing a delusion where she believes her husband to be replaced by an imposter. This is known as Capgras syndrome and can be seen in organic states such as delirium as well as in schizophrenia. It is rare but is most commonly seen in older women. The delusion most commonly relates to a life partner and can sometimes lead to serious violence being perpetrated against the supposed imposter.

Cotard syndrome is a nihilistic delusion seen in severely depressed people where they believe that they, or a part of their body is dead.

In Fregoli syndrome, the patient believes that an individual (who is almost always a persecutory figure and someone close to them) has taken on many different guises. This syndrome is named after an artist called Leopoldo Fregoli renowned for his ability to change costumes very quickly. A person with Fregoli syndrome will identify several different strangers as being the persecutor in disguise.

👍 👎 Discuss (6) Improve

Next question ›

Capgras syndrome ★

Capgras syndrome refers to a disorder in which a person holds a delusion that a friend or partner has been replaced by an identical-looking impostor.

+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Media

Capgras syndrome

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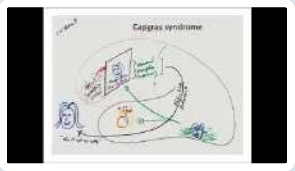
Capgras syndrome

Capgras syndrome

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Score: **22%**

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Question 40 of 409

A 28-year-old male presents to the acute medical unit with confusion. His girlfriend states that over the past couple of days he has become progressively more confused. His past medical history includes epilepsy. His drug history includes sodium valproate. On examination, you note myoclonus and his Glasgow coma score is reduced at 13 (M6, V4, E3).

Blood results are as follows:

Hb	142 g/l	Na^+	138 mmol/l	Bilirubin	14 µmol/l
Platelets	388 * 10^9/l	K^+	4.4 mmol/l	ALP	82 u/l
WBC	10.8 * 10^9/l	Urea	5.8 mmol/l	ALT	24 u/l
Neuts	8.4 * 10^9/l	Creatinine	72 µmol/l	γGT	36 u/l
Lymphs	1.2 * 10^9/l	Albumin	42 g/l	Plasma ammonia	65 µmol/L (normal range 11-32 µmol/L)

What treatment is indicated?

- ○ L-carnitine
- ○ Lactulose
- ○ Rifaximin
- ○ Sodium phenylbutyrate
- ○ Sodium benzoate

Submit answer

Reference ranges

Score: **24.1%**

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Question 40 of 409 ✗

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Lymphs	1.2 * 10^9/l	Albumin	42 g/l	Plasma ammonia	65 µmol/L (normal range 11-32 µmol/L)

What treatment is indicated?

L-carnitine	47%
Lactulose	32%
Rifaximin	11%
Sodium phenylbutyrate	5%
Sodium benzoate	5%

L-carnitine can be used in the treatment of hepatotoxicity or hyperammonemic encephalopathy in the context of an acute or chronic overdose of sodium valproate

Important for me Less important

Valproate-induced hyperammonaemic encephalopathy (VHE) is an unusual complication characterised by a decreasing level of consciousness, focal neurological deficits, cognitive slowing, vomiting, drowsiness, and lethargy. Asymptomatic hyperammonaemia occurs in 15-50% of patients on valproate.

Treatment with L-carnitine may be beneficial in reducing ammonia levels.

Lactulose and rifaximin are used in the prevention of hepatic encephalopathy.

Intravenous sodium phenylbutyrate and sodium benzoate are used to treat hyperammonaemia in patients with urea cycle enzyme deficiencies.

Discuss (5) | Improve

Next question

Sodium valproate

Sodium valproate is used in the management of epilepsy and is first-line therapy for generalised seizures. It works by increasing GABA activity.

Adverse effects

- teratogenic
    - neural tube defects
    - maternal use of sodium valproate is associated with a significant risk of neurodevelopmental delay in children
    - guidance is now clear that sodium valproate should not be used during pregnancy and in women of childbearing age unless clearly necessary. Women of childbearing age should not start treatment without specialist neurological or psychiatric advice.
- P450 inhibitor
- gastrointestinal: nausea
- increased appetite and weight gain
- alopecia: regrowth may be curly
- ataxia
- tremor
- hepatotoxicity
- pancreatitis
- thrombocytopaenia
- hyponatraemia
- hyperammonemic encephalopathy: L-carnitine may be used as treatment if this develops

+ Q 123

Next question

Textbooks

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Extended textbook

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A 52-year-old male presents to the emergency department with a 5-week history of constipation not relieved by over the counter laxatives. His past medical history was significant for hypertension, COPD and schizophrenia. His current medications include ramipril, clozapine and tiotropium. He denied weight loss, poor appetite or other alarming symptoms. His family history was significant for bowel cancer with his father and uncle having died of colon cancer aged 78 and 82 respectively. His sister had a hysterectomy at the age of 72 but he was not sure why. On examination, his blood pressure was 124/78mmHg lying and 115/80mmHg standing. Pulse was 63/min. Clinical examination did not reveal any significant findings.

Investigations:

Hb	137 g/l
MCV	82 fl
Platelets	420 * 10^9/l
WBC	7 * 10^9/l
Creatinine	89 umol/L
Urea	4.7 umol/L
Na+	143 mmol/L
K+	3.9 mmol/L
Corrected Calcium	2.3mmol/L
FOB	negative
Abdominal X-ray	faecal loading

What is the most likely cause of constipation in this case?

○ Clozapine

○ Colon cancer

○ Illicit drugs

○ Dehydration

○ Malingering

Submit answer

Reference ranges

Score: **24.1%**

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Na+	143 mmol/L
K+	3.9 mmol/L
Corrected Calcium	2.3mmol/L
FOB	negative
Abdominal X-ray	faecal loading

What is the most likely cause of constipation in this case?

Clozapine	86%
Colon cancer	4%
Illicit drugs	3%
Dehydration	4%
Malingering	3%

One of the most common side effects of clozapine is constipation/intestinal obstruction

Important for me Less important

This is an interesting case and the differential is between colon cancer and clozapine-induced constipation. Clozapine is reserved for drug-resistant schizophrenia and rightfully so as it comes with serious side effects one of which is constipation.

Constipation occurs in 14% of people on clozapine and it is believed to be an anticholinergic effect. There are papers in literature which report more serious implications like bowel infarction and death from longstanding clozapine-induced constipation.

Cancer is unlikely here as this patient has no other alarming symptoms or signs (no weight loss, normal haemoglobin, negative FOB) to suggest this and also the cancer cases in his family history are unlikely due to a hereditary condition as it has appeared in elderly relatives with no evidence of anticipation. In practice though cancer should be excluded.

There is no indication of illicit drug use and dehydration is unlikely to cause such long history of constipation without physical findings. Here we are not told of any finding suggesting dehydration on clinical examination. Malingering usually indicates personal gains of which there are not mentioned.

Clozapine is a common cause of constipation and should not be ignored. The above case is a good example.

Death From Clozapine-Induced Constipation Case Report and Literature Review
http://psychrights.org/research/digest/nlps/DeathbyClozapineConstipation.pdf

Clozapine-induced Bowel Infarction A Case Report Nicholas D. McKinnon, MD, CAPT, USAF, MC,corresponding author Alvi Azad, DO, CAPT, USAF, MC, Brian M Waters, MD, CAPT, USAF, MC, and Kaustubh G. Joshi, MD, MAJ, USAF, MC, FS
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2719458/

Pharmacy update: Clozapine-induced constipation http://www.bsmhft.nhs.uk/gps-and-commissioners/gpmatters/issue-27-october-2013/pharmacy/

Death from clozapine-induced constipation prompts warning http://www.pharmaceutical-journal.com/news-and-analysis/news/death-from-clozapine-induced-constipation-prompts-warning/11100557.article

Discuss (7) Improve

Next question

Clozapine ★

Clozapine, one of the first atypical agents to be developed, carries a significant risk of agranulocytosis and full blood count monitoring is therefore essential during treatment. For this reason, clozapine should only be used in patients resistant to other antipsychotic medication. The BNF states:

> *Clozapine should be introduced if schizophrenia is not controlled despite the sequential use of two or more antipsychotic drugs (one of which should be a second-generation antipsychotic drug), each for at least 6-8 weeks.*

Adverse effects of clozapine
- agranulocytosis (1%), neutropaenia (3%)
- reduced seizure threshold - can induce seizures in up to 3% of patients
- constipation
- myocarditis: a baseline ECG should be taken before starting treatment
- hypersalivation

Dose adjustment of clozapine might be necessary if smoking is started or stopped during treatment.

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

BNF 👍 4 👎 2

Clozapine

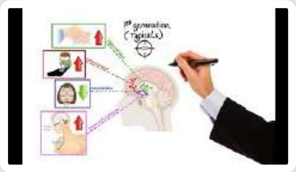
Clinical Knowledge Summaries 👍 3 👎 0

Schizophrenia > Prescribing Information > Adverse effects

Suggest link

Report broken link

Media



Antipsychotics

Speed Pharmacology - YouTube 👍 2 👎 0

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A 74-year-old man is brought to the emergency by ambulance with limb weakness and speech disturbance. He awoke at 8 am with the symptoms and was last seen by his wife at 10 pm the previous night, at which point he was well. His past medical history includes hypertension and hypercholesterolemia. He takes ramipril and atorvastatin. He does not smoke or drink alcohol. He is functionally independent at baseline and is a non-executive director of a major multinational organization.

His observations are heart rate 88 beats per minute, blood pressure 128/67 mmHg, respiratory rate 17/minute, oxygen saturation 96% on room air and temperature 37ºC.

Neurological examination reveals an expressive dysphasia and 3+/5 power in the right upper and lower limbs with correspondingly reduced sensation.

Blood glucose testing is normal.

An ECG demonstrates atrial fibrillation.

Urgent CT head and CT angiography are organized which demonstrate a proximal occlusion of the anterior circulation and no haemorrhage.

The patient proceeds to a diffusion-weighted MRI, which demonstrates a limited core infarct volume.

What is the best choice of management?

- ○ Apixaban
- ○ Clopidogrel
- ○ Intravenous thrombolysis
- ○ Intravenous thrombolysis + thrombectomy
- ○ Thrombectomy

Submit answer

Reference ranges

Score: **24.1%**

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Blood glucose testing is normal.

An ECG demonstrates atrial fibrillation.

Urgent CT head and CT angiography are organized which demonstrate a proximal occlusion of the anterior circulation and no haemorrhage.

The patient proceeds to a diffusion-weighted MRI, which demonstrates a limited core infarct volume.

What is the best choice of management?

Apixaban	2%
Clopidogrel	5%
Intravenous thrombolysis	3%
Intravenous thrombolysis + thrombectomy	22%
Thrombectomy	67%

For thrombectomy in acute ischaemic stroke, an extended target time of 6-24 hours may be considered if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Important for me   Less important

Thrombectomy is the correct answer. This treatment can be offered to those patients who were last known to be well 6-24 hours previously (including wake-up strokes) and who have confirmed occlusion of the proximal anterior circulation but limited core infarct volume on diffusion-weighted MRI. This patient fulfils all of those criteria.

Apixaban is incorrect. While this patient has new atrial fibrillation, anticoagulation is typically delayed for two weeks after an ischaemic stroke to prevent haemorrhagic complications.

Clopidogrel is incorrect. This is used for secondary prevention of ischaemic stroke (not caused by atrial fibrillation) and typically follows treatment in the initial period with high dose aspirin.

Intravenous thrombolysis is incorrect. This patient presents outside the thrombolysis window (4.5 hours) and so this treatment is unsuitable.

Intravenous thrombolysis + thrombectomy is incorrect. This patient presents outside the thrombolysis window and so this treatment would not be offered in combination with thrombectomy.

👍 👎 Discuss (4) Improve

Next question ›

Stroke: management of acute ischaemic stroke ★

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
    - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
    - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'

- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:
- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.
- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
    - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
    - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Absolute	Relative
- Oesophageal varices - Uncontrolled hypertension >200/120mmHg	

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke

- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled
- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%
- it should be performed as soon as possible within 7 days

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 11 👎 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 16

Stroke and TIA guidelines

Age and Ageing 👍 21 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

2023 National Clinical Guideline for Stroke

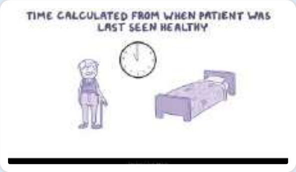
NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link

Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia
Acute ischaemic Stroke

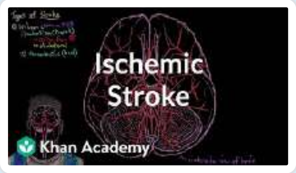
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

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Score: **22%**

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An 85-year-old woman was referred by her General Practitioner to the stroke team on an urgent outpatient basis after her rest home staff reported an unusual episode the previous week. The patient normally suffered from mild dementia but was independent with activities of daily life with minimal assistance from care staff. One week previously, she had been found unusually drowsy in an armchair at her home. Staff recalled that she had been inconsistent with following commands and in particular was unable to raise her left arm in the air and was unable to stand. The drowsiness and arm weakness had resolved within 30 minutes. However, the patient had subsequently been more confused than normal with reduced mobility. On direct questioning, rest home staff report that a few days before the above episode, the patient had lost her balance and had sat down heavily on the ground. The care worker with her at the time was certain that the patient had not hit her head during this incident.

Past medical history included mild dementia, myocardial infarction and atrial fibrillation. Regular medications included aspirin 75 mg daily, ramipril 2.5 mg daily, bisoprolol 2.5 mg daily, simvastatin 20 mg daily and warfarin (target INR 2-3). Review of recent blood test monitoring showed that the patients INR had been well controlled over recent months with no results outside of the therapeutic range. The patient did not drink and was a long-term ex-smoker. As mentioned above, the patient normally needed minimal assistance with self-care and was fully continent.

Examination in clinic demonstrated a significant cognitive impairment (abbreviated mental test score 4/10) but without evidence of dysphasia. Pupils were equal and reactive to light with no papilloedema on fundoscopy. There was a full range of conjugate eye movements. No facial weakness or sensory loss was demonstrated. Tongue and palate function was unremarkable with no weakness of trapezius muscle. Examination of the peripheral nerves was unremarkable except for possible slight pronator drift in the left arm and an up going left plantar response. Cardiovascular examination was unremarkable except for an irregular pulse. The respiratory and abdominal examination was unremarkable.

Results of investigations performed at the time of clinic assessment are given below.

Electrocardiogram: atrial fibrillation at rate 80 bpm, normal axis, inferior T wave inversion

Chest x-ray: clear lung fields

Carotid doppler: no significant stenosis in right internal carotid artery; 50 % stenosis in the left internal carotid

Transthoracic echocardiogram: mild left ventricular systolic impairment, normal valvular function, no mural thrombus

What is most likely finding on CT brain scan performed after clinic review?

○ Sub-arachnoid haemorrhage ×

○ Cerebral infarction ×

○ Intracerebral haemorrhage ×

○ Normal pressure hydrocephalus ×

○ Sub-dural haematoma ×

Submit answer

Reference ranges ⌄

Score: **24.1%**

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What is most likely finding on CT brain scan performed after clinic review?

Sub-arachnoid haemorrhage	3%
Cerebral infarction	28%
Intracerebral haemorrhage	5%
Normal pressure hydrocephalus	13%
Sub-dural haematoma	50%

The diagnosis of sub-dural haematoma can be challenging, especially in those individuals with underlying dementia. In particular, a high index of suspicion is required in elderly patients treated with anticoagulant drugs. Sub-dural haemorrhage commonly occurs after minor injury, with up to 50 % of cases preceded by a fall without head injury. Transient focal neurological deficit can occur in up to 20 % of cases, leading to the potential for diagnostic confusion with transient ischaemic attack.

In this case the patient's history of a fall, anticoagulation and worsening confusion all point towards sub-dural haematoma as the most likely underlying pathology. The lack of risk factors for cerebral infarction other than appropriately treated atrial fibrillation make this less likely. The history is less likely to be consistent with the other possible answers.

Teale E, Lliffe S, Young J. Subdural haematoma in the elderly. BMJ 2014;348:g1682.

Discuss (10) | Improve

Next question >

Subdural haemorrhage

A subdural haematoma is a collection of blood deep to the dural layer of the meninges. The blood is not within the substance of the brain and is therefore called an 'extra-axial' or 'extrinsic' lesion. They can be unilateral or bilateral.

Subdural haematomas can be classified in terms of their age:

- **Acute**: Symptoms usually develop within 48 hours of injury, characterised by rapid neurological deterioration

- **Subacute**: Symptoms manifest within days to weeks post-injury, with a more gradual progression.
- **Chronic**: Common in the elderly, developing over weeks to months. Patients may not recall a specific head injury.

The typical presentation of a subdural haematoma (SDH) often involves a history of head trauma, which may range from minor to severe. Patients frequently exhibit a lucid interval followed by a gradual decline in consciousness. This pattern is particularly common in chronic SDH. Other hallmark features include headache, confusion, and lethargy.

Clinical Features

Neurological Symptoms:

- Altered Mental Status: Ranging from mild confusion to deep coma. Fluctuations in the level of consciousness are common.
- Focal Neurological Deficits: Weakness on one side of the body, aphasia, or visual field defects, depending on the haematoma's location.
- Headache: Often localised to one side, worsening over time.
- Seizures: May occur, particularly in acute or expanding hematomas.

Physical Examination Findings:

- Papilloedema: Indicates raised intracranial pressure.
- Pupil Changes: Unilateral dilated pupil, especially on the side of the haematoma, indicating compression of the third cranial nerve.
- Gait Abnormalities: Including ataxia or weakness in one leg.
- Hemiparesis or Hemiplegia: Reflecting the mass effect and midline shift.

Behavioural and Cognitive Changes:

- Memory Loss: Especially in chronic SDH.
- Personality Changes: Irritability, apathy, or depression.
- Cognitive Impairment: Difficulty with attention, problem-solving, and other executive functions.

Other Associated Features:

- Nausea and Vomiting: Secondary to increased intracranial pressure.
- Drowsiness: Progressing to stupor and coma in severe cases.
- Signs of Increased Intracranial Pressure: Such as bradycardia, hypertension, and respiratory irregularities (Cushing's triad).

Although the collection of blood is within the same anatomical compartment, acute and chronic subdurals have important differences in terms of their mechanisms, associated clinical features and management:

Acute subdural haematoma

An acute subdural haematoma is a collection of fresh blood within the subdural space and is most commonly caused by high-impact trauma. Since it is associated with high-impact injuries, there is often other brain underlying brain injuries.

There is a spectrum of severity of symptoms and presentation depending on the size of the compressive acute subdural haematoma and the associated injuries. Presentation ranges from an incidental finding in trauma to severe coma and coning due to herniation.

CT imaging is the first-line investigation and will show a crescentic collection, not limited by suture lines. They will appear hyperdense (bright) in comparison to the brain. Large acute subdural haematomas will push on the brain ('mass effect') and cause midline shift or herniation.

Small or incidental acute subdurals can be observed conservatively. Surgical options include monitoring of intracranial pressure and decompressive craniectomy.

Chronic subdural haematoma

A chronic subdural haematoma is a collection of blood within the subdural space that has been present for weeks to months.

Rupture of the small bridging veins within the subdural space rupture and cause slow bleeding. Elderly and alcoholic patients are particularly at risk of subdural haematomas since they have brain atrophy and therefore fragile or taut bridging veins.

Presentation is typically a several week to month progressive history of either confusion, reduced consciousness or neurological deficit.

Infants also have fragile bridging veins and can rupture in shaken baby syndrome.

On CT imaging they similarly are crescentic in shape, not restricted by suture lines and compress the brain ('mass effect'). In contrast to acute subdurals, chronic subdurals are hypodense (dark) compared to the substance of the brain.

If the chronic subdural is an incidental finding or if it is small in size with no associated neurological deficit then it can be managed conservatively with the hope that it will dissolve with time. If the patient is confused, has an associated neurological deficit or has severe imaging findings then surgical decompression with burr holes is required.

+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

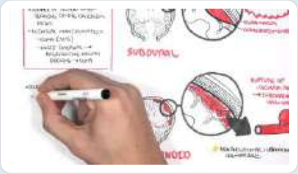
Links

Radiopaedia 👍 3 👎 1

Subdural haemorrhage

Suggest link Report broken link

Media



Intracranial Haemorrhage Types

Armando Hasudungan - YouTube 👍 1 👎 0

Intracranial
Bleeds
subdural
&
extradural

Intracranial bleeds - Subdural and Extradural haematoma

Soton Brain Hub - YouTube 👍 0 👎 0

TRAUMATIC
BRAIN INJURY
~ PATHOLOGY REVIEW ~
Osmosis.org

Traumatic brain injury: pathology review

Osmosis - YouTube 👍 0 👎 0

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Score: **22%**

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An urgent medical consult was requested regarding a 52-year-old female who had been admitted to the neurology ward a few hours previously and who had developed hypotension. She had presented to the Emergency Department with new weakness in her legs that had occurred over a course of three days. She also complained of continual lower back pain and pain in the top of her thighs, and as her illness progressed she was no longer able to weight bear. She had a past medical history of hypothyroidism, for which she was prescribed levothyroxine 125mcg OD, and other than an episode of viral gastroenteritis diagnosed by her GP two weeks prior to her current admission she was fit and well. Upon transfer to the neurology ward blood investigations, a chest x-ray, CT head scan and lumbar puncture were performed, the results of which are as follows:

Hb	132 g/l
Platelets	248 * 10^9/l
WBC	8.2 * 10^9/l

Na^+	138 mmol/l
K^+	5.2 mmol/l
Urea	6.4 mmol/l
Creatinine	77 µmol/l
CRP	15 mg/l
Glucose	7.2 mmol/l
TSH 0.26	(NR 04-3.6 mu/l)
Free T4 10.6	(NR 10 - 20 pmol/L)

Autoantibody screen: awaiting results

Urinalysis: negative
Urine MCS: awaiting results
Blood MCS: awaiting results

Chest x-ray: normal appearances of heart and lung fields
CT head: normal intracranial appearances

Lumbar puncture:

Protein	0.8g (NR 0.2-0.4 g/L)
Glucose	6.3 (blood glucose 7.2)
WCC 8	(<5/mm^3)

Opening pressure	15 (NR 10-20 cmH2O)
Microscopy	nil seen, awaiting culture and sensitivity result

Whilst awaiting definitive measurement her blood pressure was noted to fluctuate between 55/30 mmHg and 162/84 mmHg within a matter of minutes; at present her blood pressure was 62/42 mmHg. Upon examination of her cardiovascular system, she was clinically well perfused with a normal and palpable radial pulse, a JVP of 3cm and a peripheral and central capillary refill time of less than two seconds. He heart rate was 82 bpm. Examination of her respiratory system was unremarkable with no respiratory distress, a respiratory rate of 16/min and oxygen saturations of 99% on air. Examination of her gastrointestinal system was unremarkable. Examination of her peripheral nervous system revealed a power deficit of grade 2/5 in all muscle groups of the lower limbs, power 5/5 in the upper limbs and normal sensation and coordination throughout. Knee and ankle reflexes were absent bilaterally. Cranial nerve examination was unremarkable.

What is single next best step with regards to management of her blood pressure?

- ○ Commence dobutamine infusion ×
- ○ Commence noradrenaline infusion ×
- ○ Commence intravenous fluid resuscitation with 500ml of colloid stat ×
- ○ Commence intravenous immunoglobulin ×
- ○ Arrange urgent plasmapheresis ×

Submit answer

Reference ranges ⌵

Score: **24.1%**

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An urgent medical consult was requested regarding a 52-year-old female who had been admitted to the neurology ward a few hours previously and who had developed hypotension. She had presented to the Emergency Department with new weakness in her legs that had occurred over a course of three days. She also complained of continual lower back pain and pain in the top of her thighs, and as her illness progressed she was no longer able to weight bear. She had a past medical history of hypothyroidism, for which she was prescribed levothyroxine 125mcg OD, and other than an episode of viral gastroenteritis diagnosed by her GP two weeks prior to her current admission she was fit and well. Upon transfer to the neurology ward blood investigations, a chest x-ray, CT head scan and lumbar puncture were performed, the results of which are as follows:

Hb	132 g/l
Platelets	248 * 10^9/l
WBC	8.2 * 10^9/l

Na^+	138 mmol/l
K^+	5.2 mmol/l
Urea	6.4 mmol/l
Creatinine	77 µmol/l
CRP	15 mg/l
Glucose	7.2 mmol/l
TSH 0.26	(NR 04-3.6 mu/l)
Free T4 10.6	(NR 10 - 20 pmol/L)

Autoantibody screen: awaiting results

Urinalysis: negative
Urine MCS: awaiting results
Blood MCS: awaiting results

Chest x-ray: normal appearances of heart and lung fields
CT head: normal intracranial appearances

Lumbar puncture:

Protein	0.8g (NR 0.2-0.4 g/L)
Glucose	6.3 (blood glucose 7.2)
WCC 8	($<5/mm^3$)

Opening pressure	15 (NR 10-20 cmH2O)
Microscopy	nil seen, awaiting culture and sensitivity result

Whilst awaiting definitive measurement her blood pressure was noted to fluctuate between 55/30 mmHg and 162/84 mmHg within a matter of minutes; at present her blood pressure was 62/42 mmHg. Upon examination of her cardiovascular system, she was clinically well perfused with a normal and palpable radial pulse, a JVP of 3cm and a peripheral and central capillary refill time of less than two seconds. He heart rate was 82 bpm. Examination of her respiratory system was unremarkable with no respiratory distress, a respiratory rate of 16/min and oxygen saturations of 99% on air. Examination of her gastrointestinal system was unremarkable. Examination of her peripheral nervous system revealed a power deficit of grade 2/5 in all muscle groups of the lower limbs, power 5/5 in the upper limbs and normal sensation and coordination throughout. Knee and ankle reflexes were absent bilaterally. Cranial nerve examination was unremarkable.

What is single next best step with regards to management of her blood pressure?

Commence dobutamine infusion	1%
Commence noradrenaline infusion	4%
Commence intravenous fluid resuscitation with 500ml of colloid stat	9%
Commence intravenous immunoglobulin	67%
Arrange urgent plasmapheresis	18%

IVIG is generally used first-line for Guillain-Barre syndrome

Important for me Less important

This lady has developed Guillain Barre Syndrome (GBS) with evidence of a temporary autonomic neuropathy, as manifested by her highly labile blood pressure. The treatment of this is therefore to treat the underlying disorder, which in this instance is with intravenous immunoglobulin. In the presence of GBS complicated by autonomic neuropathy blood pressure is not a reliable indicator and any thus any decisions regarding fluid status must be in concordance with clinical examination findings. In this question, she is clearly euvolaemic clinically and so there is no indication for fluid or inotropic support.

Discuss (6) Improve

Next question

Guillain-Barre syndrome: management ★

Guillain-Barre syndrome describes an immune-mediated demyelination of the peripheral nervous system often triggered by an infection (classically *Campylobacter jejuni*).

Management

- IV immunoglobulins (IVIG) or plasma exchange can be used
  - IVIG is as effective as plasma exchange better tolerated and easier to administer
  - therefore IVIG is generally used first-line for Guillain-Barre syndrome where patients require immunomodulatory therapy
  - no benefit in combining both treatments
- steroids and immunosuppressants have not been shown to be beneficial
- FVC regularly to monitor respiratory function

Prognosis

- severe motor problems persist in about 15%
- around 5% die

AI + Q 123

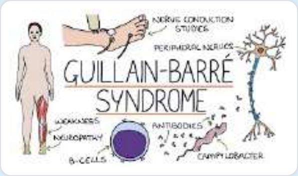
Next question >

Textbooks

High-yield textbook

Extended textbook

Media


NERVE CONDUCTION STUDIES
PERIPHERAL NERVES
GUILLAIN-BARRÉ SYNDROME
WEAKNESS
NEUROPATHY
B-CELLS
ANTIBODIES
CAMPYLOBACTER

[Understanding Guillain-Barre Syndrome]

Zero To Finals - YouTube 👍 3 👎 1

[Guillain-Barre syndrome]

Osmosis - YouTube 👍 1 👎 1

Report broken media

Score: **22%**

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A 60-year-old man presents to the neurology clinic with progressive weakness in the upper and lower limbs bilaterally. Symptoms initially came on gradually over 3 years and he is now bedbound. His past medical history is significant for hypertension, which is well controlled on amlodipine 10 mg once daily. On examination, there is no sensory abnormality and deep tendon reflexes are intact. Power is significantly reduced to 1/5 in the quadriceps and wrist flexors. Relevant investigations are shown below:

Calcium	2.5 mmol/L	(2.1-2.6)
Phosphate	1.2 mmol/L	(0.8-1.4)
Magnesium	0.9 mmol/L	(0.7-1.0)
Thyroid stimulating hormone (TSH)	4.0 mU/L	(0.5-5.5)
Free thyroxine (T4)	13 pmol/L	(9.0 - 18)
Amylase	80 U/L	(70 - 300)
Uric acid	0.53 mmol/L	(0.18 - 0.48)
Creatine kinase	1020 U/L	(35 - 250)
Electromyography (EMG)	Suggestive of inflammatory myopathy	

What is the most likely **specific** finding on muscle biopsy?

○ Absent dystrophin protein

○ CD8+ T-cell infiltrates

○ Cytoplasmic inclusions

○ Muscle necrosis

○ Ragged red fibers

Submit answer

Reference ranges

Score: **24.1%**

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Amylase	80 U/L	(70 - 300)
Uric acid	0.53 mmol/L	(0.18 - 0.48)
Creatine kinase	1020 U/L	(35 - 250)
Electromyography (EMG)	Suggestive of inflammatory myopathy	

What is the most likely **specific** finding on muscle biopsy?

Absent dystrophin protein	5%
CD8+ T-cell infiltrates	11%
Cytoplasmic inclusions	59%
Muscle necrosis	6%
Ragged red fibers	19%

Older man + quadriceps and finger/wrist flexors weakness + raised CK → inclusion body myositis

Important for me Less important

The scenario describes a patient with inclusion body myositis. Weak flexors and extensors and involvement of both proximal and distal muscles, as described in this scenario, are characteristic of inclusion body myositis. It typically occurs in older males and CK is raised, as with all inflammatory myopathies. Normal sensations and intact deep tendon reflexes also point towards inflammatory myopathy. Muscle biopsy findings of **cytoplasmic inclusions** are the distinguishing feature of inclusion body myositis. The cytoplasmic inclusions are ovoid, electron-dense bodies that are surrounded by clear haloes.

The age and onset of this patient's disease do not favour a diagnosis of Duchenne muscle dystrophy, which would arise in childhood. A biopsy would instead show **absent dystrophin proteins**.

Polymyositis is the most common AIDS-related myopathy, characterised by the presence of **CD8+ T-cell infiltrates** in the muscles. The patient in this scenario denies any significant medical problems and has no history of repeated infections, weight loss, fever or any HIV-related features.

Muscle necrosis, muscle fibre regeneration, and inflammatory infiltrates are the biopsy findings of all inflammatory myopathies. These are not specific to inclusion body myositis.

Ragged red fibres on muscle biopsy are a feature of mitochondrial myopathies. These rare diseases are unlikely in this patient, as they would present at a much younger age.

Discuss Improve

Next question

Inclusion body myositis

Overview
- cause of myopathy
- associated with cytoplasmic inclusions on muscle biopsy

Features
- typically affects older males
- can affect both proximal and distal muscles
- characteristically affects quadriceps and finger/wrist flexors are usually more severely affected than extensor counterparts

+ Q 123

Next question

Textbooks

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Extended textbook

Media

INCLUSION BODY MYOPATHY

Inclusion body myositis

Osmosis - YouTube 👍 7 👎 2

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Score: **22%**

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Question 46 of 409

A 55-year-old male is found confused on street by a policeman. Hospital records document that he is a known to be a type 2 diabetic with previously DNAs to clinic and known poor compliance with medications. He also had two previous admissions to hospital, 2 and 10 years ago, for alcohol withdrawal. On examination, the patient is not orientated in time or place, scoring 0 out of 10 on abbreviated mental. He is able to follow your commands in lifting his upper and lower limbs during his neurological exams. All reflexes were present. He fails to follow your finger with his eyes on cranial nerve examination. His gait is grossly ataxic, as you noted when he tried to walk to the toilet against medical and nursing advice. His blood sugar is awaited. What is the diagnosis?

○ Wernicke's encephalopathy

○ Delirium

○ Miller-Fisher syndrome

○ Hypoglycaemia secondary to anti-glycaemic overdose

○ Acute ischaemic stroke

Submit answer

Reference ranges

Score: **24.1%**

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Question 46 of 409 ✗

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Wernicke's encephalopathy	90%
Delirium	3%
Miller-Fisher syndrome	4%
Hypoglycaemia secondary to anti-glycaemic overdose	2%
Acute ischaemic stroke	1%

The patient presents with a triad of ophthalmoplegia, ataxia and encephalopathy, suggestive of Wernicke's encephalopathy. The pathophysiology relies on an underlying thiamine deficiency, which is a key co-factor in energy metabolism and is often deficient in alcoholics. Miller-Fisher syndrome is a key differential in this case: this is a variant of Guillain-Barre syndrome presenting with ophthalmoplegia, ataxia and areflexia. Delirium and hypoglycaemia should not present with ophthalmoplegia.

Discuss (2) | Improve

Next question >

Wernicke's encephalopathy

Wernicke's encephalopathy is a neuropsychiatric disorder caused by thiamine deficiency which is most commonly seen in alcoholics. Rarer causes include persistent vomiting, stomach cancer, and dietary deficiency. A classic triad of ophthalmoplegia/nystagmus, ataxia and encephalopathy may occur.

In Wernicke's encephalopathy, petechial haemorrhages occur in a variety of structures in the brain including the mamillary bodies and ventricle walls.

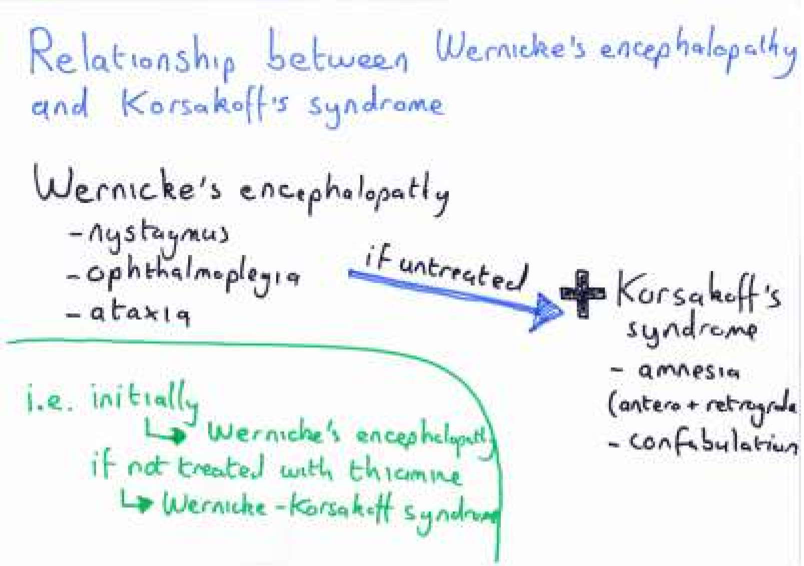
Features
- oculomotor dysfunction
  - nystagmus (the most common ocular sign)
  - ophthalmoplegia: lateral rectus palsy, conjugate gaze palsy
- gait ataxia
- encephalopathy: confusion, disorientation, indifference, and inattentiveness
- peripheral sensory neuropathy

Investigations
- decreased red cell transketolase
- MRI

Treatment is with urgent replacement of thiamine

Relationship with Korsakoff syndrome

If not treated Korsakoff's syndrome may develop as well. This is termed Wernicke-Korsakoff syndrome and is characterised by the addition of antero- and retrograde amnesia and confabulation in addition to the above symptoms.


Relationship between Wernicke's encephalopathy and Korsakoff's syndrome
Wernicke's encephalopathy
- nystagmus
- ophthalmoplegia
- ataxia
if untreated
+ Korsakoff's syndrome
- amnesia (antero + retrograde)
- confabulation
i.e. initially
→ Wernicke's encephalopathy
if not treated with thiamine
→ Wernicke-Korsakoff syndrome

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

WERNICKE-KORSAKOFF SYNDROME
~ YEAR of the ZEBRA ~
OSMOSIS

Wernicke-Korsakoff syndrome

Osmosis - YouTube 👍 2 👎 0

Thiamine Deficiency

Thiamine Deficiency (Vitamin B1)

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Report broken media

Score: **22%**

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You have been referred a 45-year-old man by the Accident and Emergency doctors with a severe headache. The headache woke him up at 3am, and he describes it as the worst headache he's ever had, (although he admits he's not a regular headache sufferer). He has had seven episodes like this over the past two weeks that have followed a very similar pattern, with the other two headaches lasting around 60 minutes before going. The pain is mainly around the left eye and temple and is sharp in nature. You have to ask him to sit down to examine him because he is up and pacing around his room, clearly very agitated. On examining him you note that his left eye is watering and swollen, and there's some redness and mild bruising just above the eye. When you ask him about this bruising he says that the pain was so bad he bashed his head against the fridge door to try and help take it away.

On examination he has normal power, sensation and reflexes in all four limbs.

On examining his cranial nerves you notice that he has a mild left sided ptosis and miosis, and there is conjunctival injection and lacrimation on that side too.

His neck movements are slow but complete, and he has no obvious stiffness or photophobia

What is the most likely cause of his headache?

- ○ Cluster headache ×
- ○ Subarachnoid haemorrhage ×
- ○ Paroxysmal hemicrania ×
- ○ Migraine ×
- ○ Tension headache ×

Submit answer

Reference ranges

Score: **24.1%**

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His neck movements are slow but complete, and he has no obvious stiffness or photophobia

What is the most likely cause of his headache?

Cluster headache	81%
Subarachnoid haemorrhage	3%
Paroxysmal hemicrania	14%
Migraine	1%
Tension headache	0%

What is described above is the classical cluster headache. NICE guidelines characterise cluster headaches as one-sided headache in or around the eye or temporal region associated with signs of autonomic dysfunction on the same side.

Attacks of pain:
- Usually last for 15-180 minutes
- Almost always described as the most severe pain known
- Tend to recur at the same time each day

- Often wake the person shortly after falling asleep

Signs of symptoms of autonomic dysfunction include:
- Rhinorrhoea or nasal congestion
- Red eye and/or lacrimation
- Facial or forehead sweating or flushing
- Constriction of the pupil and/or ptosis
- Eyelid oedema
- A sense of aural fullness

Tension headaches tend to be bilateral, pressing, with a pain intensity of mild to moderate and without other associated signs or symptoms.

Migraines can be both unilateral or bilateral and tend to be throbbing or banging in nature. While they can be severe in nature, they tend not to be described as the worst pain the patient has ever had. Unlike the agitation that comes with cluster headaches, patients with migraines tend to avoid activities of daily living shutting themselves in quiet dark rooms to provide relief. In addition the headaches tend to be longer, most commonly being between 4 hours and 3 days in adults.

Paroxysmal hemicrania attacks then to be much shorter (2-30 minutes), happen with much higher frequency (>5 times/day), and are more common in women. In an exam situation they will also tend to mention that the patient has found complete relief with indometacin (or this will be the subject of the question).

Subarachnoid haemorrhage headaches do not have a preceding history of previous attacks.

👍 👎 Discuss (5) Improve

Next question >

Cluster headache ★

Cluster headaches are known to be one of the most painful conditions that patients can have the misfortune to suffer. The name relates to the pattern of the headaches - they typically occur in clusters lasting several weeks, with the clusters themselves typically once a year.

Cluster headaches are more common in men (3:1) and smokers. Alcohol may trigger an attack and there also appears to be a relation to nocturnal sleep.

Features
-intense sharp, stabbing pain around one eye
- pain typical occurs once or twice a day, each episode lasting 15 mins - 2 hours

  - the patient is restless and agitated during an attack due to the severity
- clusters typically last 4-12 weeks
- accompanied by redness, lacrimation, lid swelling
- nasal stuffiness
- miosis and ptosis in a minority

Investigations
- most patients will have neuroimaging - underlying brain lesions are sometimes found even if the clinical symptoms are typical for cluster headache
- MRI with gadolinium contrast is the investigation of choice

Management
- NICE recommend seeking specialist advice from a neurologist if a patient develops cluster headaches
- acute
  - 100% oxygen (80% response rate within 15 minutes)
  - subcutaneous triptan (75% response rate within 15 minutes)
- prophylaxis
  - verapamil is the drug of choice
  - there is also some evidence to support a tapering dose of prednisolone

Some neurologists use the term trigeminal autonomic cephalgia to group a number of conditions including cluster headache, paroxysmal hemicrania and short-lived unilateral neuralgiform headache with conjunctival injection and tearing (SUNCT). It is recommended such patients are referred for specialist assessment as specific treatment may be required, for example it is known paroxysmal hemicrania responds very well to indomethacin

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 17 👎 10

2012 Headache guidelines

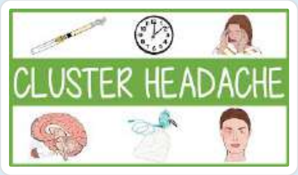
Suggest link Report broken link

Media

SEVERE SYMPTOMS
HIGH FLOW 100% OXYGEN
CLUSTER HEADACHES
PREVENTION
SUBCUT TRIPTANS
HOME OXYGEN

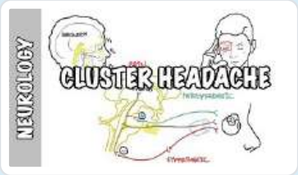
Cluster headaches

Zero To Finals - YouTube 👍 0 👎 0


CLUSTER HEADACHE

Cluster headache

Townsend Teaching - YouTube 👍 0 👎 0


NEUROLOGY
CLUSTER HEADACHE

Cluster Headaches - symptoms, pathophysiology, treatment

Armando Hasudungan - YouTube 👍 1 👎 1

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Score: **22%**

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A 54-year-old gentleman is referred to Neurology clinic with a 2 month history of nocturnal headache and difficulty walking.

On examination he has an ataxic gait and impaired co-ordination affecting the left arm and leg.

Blood results show the following

Hb	190 $x10^{12}/L$	130-168 $x10^{12}/L$
WCC	5.4 $x10^{9}/L$	4.2-10.6 $x10^{9}/L$
Platelets	180 $x10^{9}/L$	130-370 $x10^{9}/L$
CRP	4 mg/L	0-5 mg/L
Creatinine	90 µmol/L	60-125 µmol/L
eGFR	>90 mL/min/1.73 m^2	>90 mL/min/1.73 m^2
Na	140 mmol/L	133-146 mmol/L
K	4.1 mmol/L	3.5-5.3 mmol/L
ALT	31 IU/L	0-45 U/L
ALP	54 IU/L	30-130 IU/L
Bilirubin	10 µmol/L	0-21 µmol/L

A CT (computed tomography) brain scan is done which shows shows as a well-defined, low attenuation region in the posterior fossa with an enhancing nodule on the wall

What is the most likely underlying lesion?

○ Meningioma

○ Cerebellar metastasis

○ Astrocytoma

○ Haemangioblastoma

○ Medulloblastoma

Submit answer

Reference ranges ⌵

Score: **24.1%**

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ALT	31 IU/L	0-45 U/L
ALP	54 IU/L	30-130 IU/L
Bilirubin	10 µmol/L	0-21 µmol/L

A CT (computed tomography) brain scan is done which shows shows as a well-defined, low attenuation region in the posterior fossa with an enhancing nodule on the wall

What is the most likely underlying lesion?

Meningioma	45%
Cerebellar metastasis	8%
Astrocytoma	10%
Haemangioblastoma	27%
Medulloblastoma	11%

Cerebellar haemangioblastomas release erythropoietin so can cause a rise in haemoglobin

Important for me Less important

Cerebellar haemangioblastomas release erythropoietin as a paraneoplastic syndrome causing an elevated haemoglobin. None of the other tumours mentioned are associated with this phenomenon.

Haemangioblastoma is an uncommon tumour, but it is the most common primary intra-axial tumours in the posterior fossa in adults.

Astrocytomas and medulloblastomas are encountered in the posterior fossa of younger adults but are rare in older adults, accounting for <1% all tumours

The outcome for haemangioblastomas is very good, if surgical extraction of the tumor can be achieved

Haemangioblastomas are commonly seen in von- hippel Lindau (VHL) syndrome. These individuals have a worse prognosis than those who have sporadic tumors since those with VHL syndrome usually have more than one lesion

Discuss (4) Improve

Next question

Brain tumours: features

Brain tumours may cause symptoms either due to taking up space within the skull (a fixed box) or specific symptoms due to their position in the brain.

Brain tumours can be divided into groups depending on whether they are:

- Intrinsic (within the brain parenchyma) vs. extrinsic (outside the parenchyma)
- Primary vs. secondary (metastatic)
- Benign vs cancerous

Important epidemiology / demographic points:

- Metastatic brain tumours are more common than primary brain tumours
- Glioblastoma (grade IV, aggressive and invasive) is the most common adult primary brain tumour
- Whilst supratentorial tumours are more common in adults, infratentorial tumours are more common in children

Generic/classical presentation of brain tumours includes a progressive history of:

- Headache (typically morning headaches, worse on lying down)
- Nausea and vomiting
- Neurological deficit
- Lethargy

Brain tumours, especially low-grade gliomas, may present with as a seizure.

Investigations

- CT head is a reasonable screening tool for patients in which a tumour is included in the differential
- An MRI brain with contrast is the gold-standard for imaging brain tumours since it offers excellent resolution and can be used to distinguish between other pathologies (such as an abscess)
- CT chest, abdomen and pelvis to exclude an extracranial primary cancer

Management

- Referral to neurosurgery if the patient is acutely neurologically unwell
- Referral to a neuro-oncology multidisciplinary team (neurologists, oncologists, neurosurgeons, neuroradiologists) for recommendation for surgery, chemotherapy and/or radiotherapy
- Dexamethasone is used to treat vasogenic oedema (if present) with a PPI for gastric protection

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

ADULT
BRAIN TUMORS
Osmosis.org

Adult brain tumours

Osmosis - YouTube 👍 2 👎 0

PEDIATRIC
BRAIN TUMORS
Osmosis.org

Paediatric brain tumours

Osmosis - YouTube 👍 0 👎 0

Report broken media

Score: **22%**

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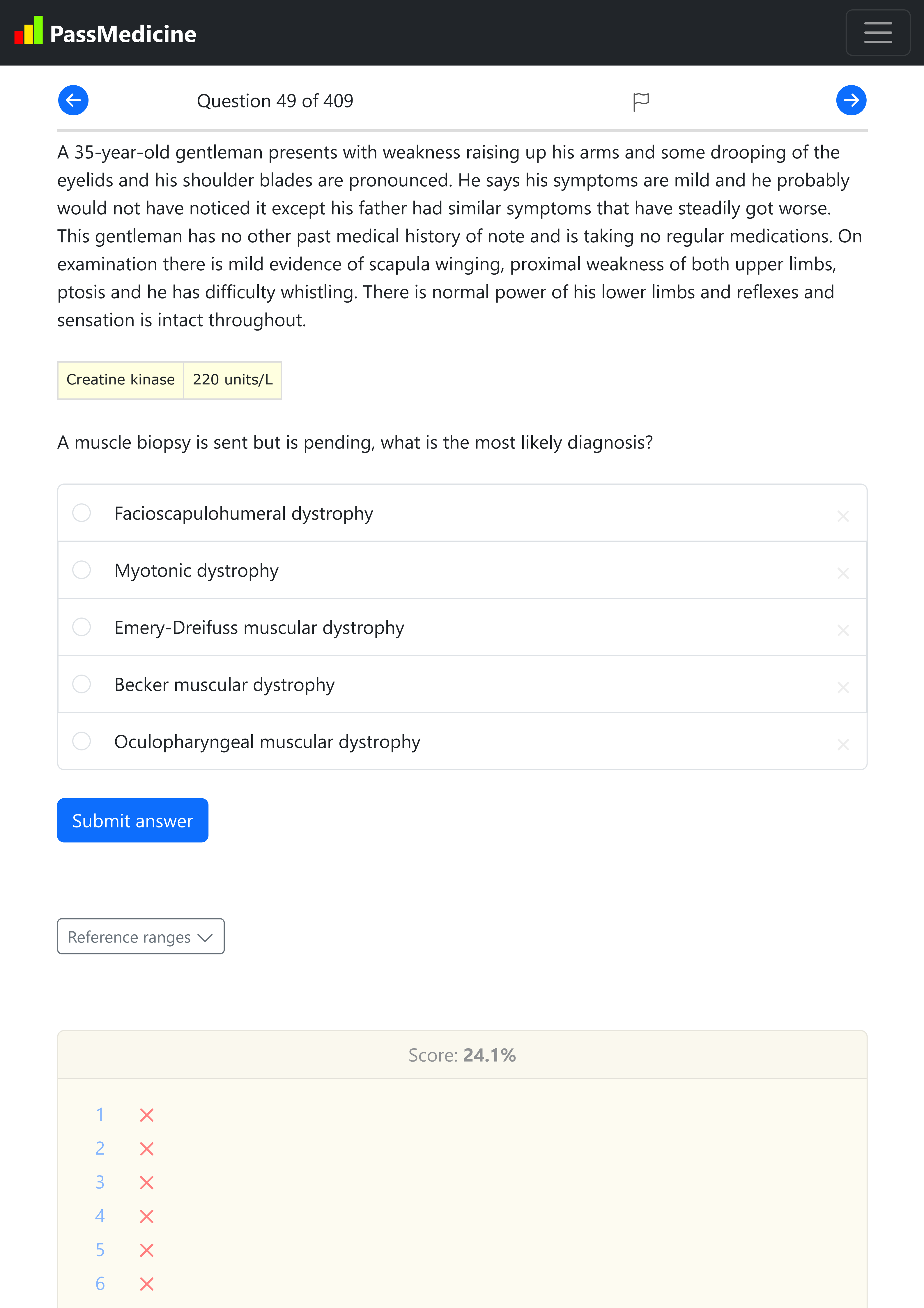

Question 49 of 409

A 35-year-old gentleman presents with weakness raising up his arms and some drooping of the eyelids and his shoulder blades are pronounced. He says his symptoms are mild and he probably would not have noticed it except his father had similar symptoms that have steadily got worse. This gentleman has no other past medical history of note and is taking no regular medications. On examination there is mild evidence of scapula winging, proximal weakness of both upper limbs, ptosis and he has difficulty whistling. There is normal power of his lower limbs and reflexes and sensation is intact throughout.

Creatine kinase	220 units/L

A muscle biopsy is sent but is pending, what is the most likely diagnosis?

- ○ Facioscapulohumeral dystrophy
- ○ Myotonic dystrophy
- ○ Emery-Dreifuss muscular dystrophy
- ○ Becker muscular dystrophy
- ○ Oculopharyngeal muscular dystrophy

Submit answer

Reference ranges

Score: **24.1%**

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Question 49 of 409

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Creatine kinase	220 units/L

A muscle biopsy is sent but is pending, what is the most likely diagnosis?

Facioscapulohumeral dystrophy	87%
Myotonic dystrophy	7%
Emery-Dreifuss muscular dystrophy	1%
Becker muscular dystrophy	3%
Oculopharyngeal muscular dystrophy	2%

Facioscapulohumeral muscular dystrophy: facial weakness (e.g. difficulty closing eyes, smiling, blowing), winged scapula and weakness of the shoulderm upper arm and hip girdle muscles

Important for me Less important

Facioscapulohumeral dystrophy is usually an autosomal dominant condition and although it affects men and women equally, men may be more symptomatic. Shoulder, upper arm and facial muscles are the most often implicated and lower limb involvement is less common. Winging of the scapulae are one characteristic finding which may necessitate surgical fixation. It is a progressive condition however prognosis is usually good with minor disability and lifespan unaffected.

Myotonic dystrophy is the most common muscular dystrophy of which symptoms include delayed relaxation of muscles wasting and weakness as well as cardiac conduction deficits, cataracts and characteristic facies. Becker muscular dystrophy usually presents in childhood with predominantly lower limb and girdle weakness. Patients with Emery-Dreifuss muscular dystrophy also usually present in childhood, characteristically with contractures and oculopharyngeal muscular dystrophy presents usually over the age of 40 with eyelid, facial and throat muscle weakness initially followed by pelvic and shoulder weakness.

Discuss (3) Improve

Next question

Facioscapulohumeral muscular dystrophy ★

Facioscapulohumeral muscular dystrophy (FSHMD) is an autosomal dominant form of muscular dystrophy. As the name suggests it typically affects the face, scapula and upper arms first. Symptoms typically present by the age of 20 years.

Features

- facial muscles are involved first - difficulty closing eyes, smiling, blowing etc
- weakness of the shoulder and upper arm muscles
- abnormal prominence of the borders of the shoulder blades - 'winging'
- lower limb: hip girdle weakness, foot drop

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **22%**

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A 45-year-old man complains of lower limb numbness associated with weakness. He also noted some recent problems with urinary incontinence and has recently attended the opticians due to some blurring of vision

On examination he has bilateral lower limb weakness (grade 3+/5). His tone was spastic on both sides with exaggerated reflexes. The planter reflexes were up-going bilaterally. The sensory level was at the tenth thoracic vertebra (T10). Eye examination revealed a normal visual acuity with a normal fundal examination.

He has a history of asthma and is currently well controlled with just a salbutamol inhaler.

Magnetic resonance imaging (MRI) of the brain is normal. MRI of the spine shows a hyper intense lesion spanning from (T7-T12). The following investigations had been ordered:

Na+	135 mmol/l
K+	4 mmol/l
Creatinine	80 µmol/l
Urea	4.5 mmol/l
CRP	5 mg/l
ESR	10 mm/hr
Urine analysis	Normal
ANA	Negative

Which one of the following investigations would be most useful to reach a diagnosis?

- ○ Auditory evoked response potentials
- ○ CSF analysis for oligoclonal bands
- ○ Anti-aquaporin 4 antibodies
- ○ Serum B12
- ○ Visual evoked response potentials

Submit answer

Reference ranges

Score: **24.1%**

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K+	4 mmol/l
Creatinine	80 µmol/l
Urea	4.5 mmol/l
CRP	5 mg/l
ESR	10 mm/hr
Urine analysis	Normal
ANA	Negative

Which one of the following investigations would be most useful to reach a diagnosis?

Auditory evoked response potentials	1%
CSF analysis for oligoclonal bands	23%
Anti-aquaporin 4 antibodies	61%
Serum B12	6%
Visual evoked response potentials	9%

Neuromyelitis optica is associated with aquaporin 4 positive serum antibody

Important for me Less important

Neuromyelitis optica (Devic disease) should be suspected when the following is noted:

- quadriparesis or paraparesis within days to weeks
- vision changes
- normal brain MRI
- presence of anti-NMO antibodies (anti-aquaporin 4 antibodies)
- responsive to immunosuppressants

Discuss (3) Improve

Next question

Neuromyelitis optica ★

Neuromyelitis optica (NMO) is a monophasic or relapsing-remitting demyelinating CNS disorder Although previously thought to be a variant of multiple sclerosis, it is now recognised to be a distinct disease, particularly prevalent in Asian populations. It typically involves the optic nerves and cervical spine, with imaging of the brain frequently normal. Vomiting is also a common presenting complaint.

Diagnosis requires bilateral optic neuritis, myelitis and 2 of the following 3 criteria:

- 1. Spinal cord lesion involving 3 or more spinal levels
- 2. Initially normal MRI brain
- 3. Aquaporin 4 positive serum antibody

Management

- immunosuppressant e.g. with anti-CD20 agent rituximab)

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **22%**

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Question 51 of 409

A 62-year-old man is being reviewed in the neurology clinic for 6 months of progressive difficulty walking and memory issues. He has difficulty initiating movements and has become increasingly forgetful. He also suffers from increased urinary frequency, with a few instances of urinary incontinence. The patient has a history of subarachnoid haemorrhage 8 months prior, for which a craniotomy and clipping were performed.

A CT scan is requested and performed.

What abnormality is most likely to be relevant to his presentation?

○ Frontal & temporal lobe atrophy

○ Midbrain atrophy & enlargement of third ventricle

○ Normal

○ Ventriculomegaly with enlarged sulci and shrunken gyri

○ Ventriculomegaly with normal-sized sulci and gyri

Submit answer

Reference ranges

Score: **22%**

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A CT scan is requested and performed.

What abnormality is most likely to be relevant to his presentation?

Frontal & temporal lobe atrophy	4%
Midbrain atrophy & enlargement of third ventricle	6%
Normal	4%
Ventriculomegaly with enlarged sulci and shrunken gyri	35%
Ventriculomegaly with normal-sized sulci and gyri	51%

Urinary incontinence + gait abnormality + dementia = normal pressure hydrocephalus

Important for me Less important

Dementia, urinary incontinence, and gait abnormality, as described in this scenario, are the classic triad of normal pressure hydrocephalus (NPH). The patient's symptoms started 2 months after a subarachnoid haemorrhage, most likely due to reduced cerebrospinal fluid (CSF) absorption by the arachnoid villi. Neuroimaging in NPH shows **ventriculomegaly with normal-sized sulci and gyri**. There is an enlargement of the frontal, temporal, and occipital horns of the lateral ventricles. This enlargement is disproportionate to the sulci, which are mostly normal in size. The definitive treatment for NPH is ventriculoperitoneal (VP) shunting. The dementia, gait disturbance, and incontinence associated with NPH are reversible and patient improves significantly after a successful VP shunt.

Frontal and temporal lobe atrophy may be seen in a patient with frontotemporal dementia (FTD). Behavioural changes are the most common presenting feature of FTD, which is absent in this scenario and therefore FTD is a less likely diagnosis here. A history of urinary incontinence along with dementia and gait abnormality is more in favour of normal pressure hydrocephalus

(NPH) than FTD.

Midbrain atrophy and enlargement of the third ventricle is a characteristic imaging feature of progressive supranuclear palsy (PSP). Gait disorder in PSP is also associated with a typical supranuclear gaze which is absent in this scenario.

NPH is diagnosed by ventricular enlargement on neuroimaging and so a **normal study** is unlikely to be seen in such a scenario.

Alzheimer's disease may show **ventriculomegaly with enlarged sulci and shrunken gyri** on imaging. The acute onset of symptoms and urinary incontinence in this scenario makes Alzheimer's disease much less likely.

👍 | 👎 | Discuss (1) | Improve

Next question >

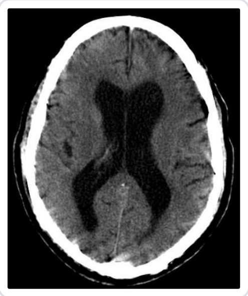
Normal pressure hydrocephalus ★

Normal pressure hydrocephalus is a reversible cause of dementia seen in elderly patients. It is thought to be secondary to reduced CSF absorption at the arachnoid villi. These changes may be secondary to head injury, subarachnoid haemorrhage or meningitis.

A classical triad of features is seen
- urinary incontinence
- dementia and bradyphrenia
- gait abnormality (may be similar to Parkinson's disease)

It is thought around 60% of patients will have all 3 features at the time of diagnosis. Symptoms typically develop over a few months.

Imaging
- hydrocephalus with ventriculomegaly in the absence of, or out of proportion to, sulcal enlargement



Management

- ventriculoperitoneal shunting
- around 10% of patients who have shunts experience significant complications such as seizures, infection and intracerebral haemorrhages

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

YouTube 👍 26 👎 2

Typical NPH Gait

Suggest link

Report broken link

Score: **20%**

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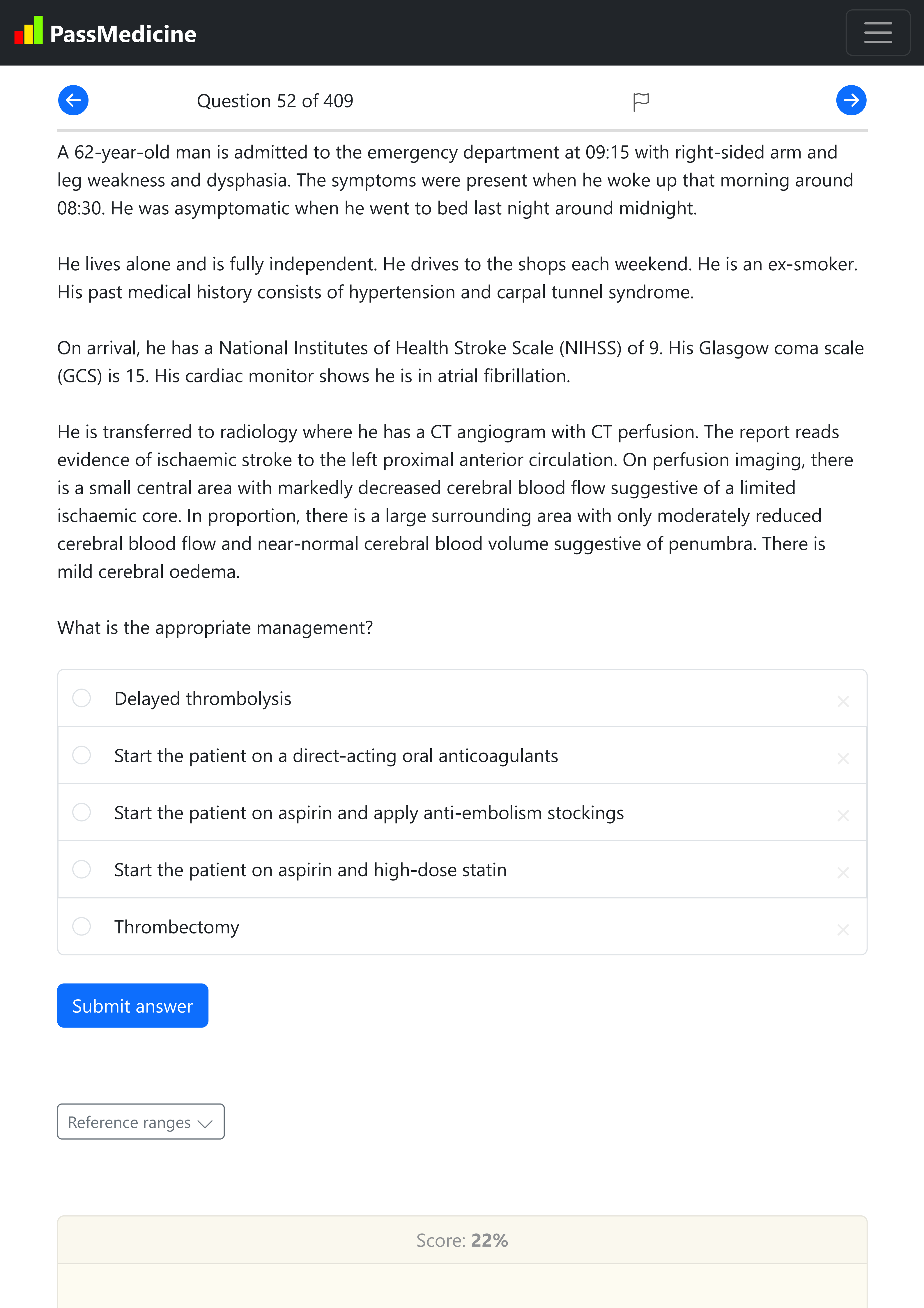

A 62-year-old man is admitted to the emergency department at 09:15 with right-sided arm and leg weakness and dysphasia. The symptoms were present when he woke up that morning around 08:30. He was asymptomatic when he went to bed last night around midnight.

He lives alone and is fully independent. He drives to the shops each weekend. He is an ex-smoker. His past medical history consists of hypertension and carpal tunnel syndrome.

On arrival, he has a National Institutes of Health Stroke Scale (NIHSS) of 9. His Glasgow coma scale (GCS) is 15. His cardiac monitor shows he is in atrial fibrillation.

He is transferred to radiology where he has a CT angiogram with CT perfusion. The report reads evidence of ischaemic stroke to the left proximal anterior circulation. On perfusion imaging, there is a small central area with markedly decreased cerebral blood flow suggestive of a limited ischaemic core. In proportion, there is a large surrounding area with only moderately reduced cerebral blood flow and near-normal cerebral blood volume suggestive of penumbra. There is mild cerebral oedema.

What is the appropriate management?

○ Delayed thrombolysis

○ Start the patient on a direct-acting oral anticoagulants

○ Start the patient on aspirin and apply anti-embolism stockings

○ Start the patient on aspirin and high-dose statin

○ Thrombectomy

Score: **22%**

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Question 52 of 409 ✓

A 62-year-old man is admitted to the emergency department at 09:15 with right-sided arm and leg weakness and dysphasia. The symptoms were present when he woke up that morning around 08:30. He was asymptomatic when he went to bed last night around midnight.

He lives alone and is fully independent. He drives to the shops each weekend. He is an ex-smoker. His past medical history consists of hypertension and carpal tunnel syndrome.

On arrival, he has a National Institutes of Health Stroke Scale (NIHSS) of 9. His Glasgow coma scale (GCS) is 15. His cardiac monitor shows he is in atrial fibrillation.

He is transferred to radiology where he has a CT angiogram with CT perfusion. The report reads evidence of ischaemic stroke to the left proximal anterior circulation. On perfusion imaging, there is a small central area with markedly decreased cerebral blood flow suggestive of a limited ischaemic core. In proportion, there is a large surrounding area with only moderately reduced cerebral blood flow and near-normal cerebral blood volume suggestive of penumbra. There is mild cerebral oedema.

What is the appropriate management?

Delayed thrombolysis	8%
Start the patient on a direct-acting oral anticoagulants	2%
Start the patient on aspirin and apply anti-embolism stockings	5%
Start the patient on aspirin and high-dose statin	4%
Thrombectomy	81%

For thrombectomy in acute ischaemic stroke, an extended target time of 6-24 hours may be considered if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Important for me   Less important

As the patient was well going he went to sleep, the longest time from onset to presentation is 9 and a half hours. He has a good baseline function (functional status would be less than 3 on the modified Rankin scale) and his NIHSS is greater than 5 making him a potential candidate for thrombectomy. This is usually used within 6 hours of onset but if there is potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume, then it can be considered up to 24 hours from onset (including wake-up strokes).

Thrombolysis with alteplase should only be given if it is administered within 4.5 hours of the onset of stroke symptoms. In this case, the stroke onset is likely outside this window and therefore thrombolysis should not be considered.

NICE has written guidance for 'Interventions for people with acute coronary syndromes or acute stroke or for acutely ill patients' where it is advised 'do not offer anti-embolism stockings for VTE prophylaxis to people who are admitted for acute stroke.'

Atrial fibrillation and stroke is a common combination. NICE recommends 'that people with disabling ischaemic stroke who are in atrial fibrillation are treated with aspirin 300 mg for the first 2 weeks before anticoagulation treatment is considered'.

Immediate initiation of statin therapy isn't recommended in acute stroke. There is an association with an increased risk of haemorrhagic transformation. Treatment should be delayed by 48 hours after stroke symptom onset (as per BNF and NICE).

👍 👎 Discuss (5) Improve

Next question >

Stroke: management of acute ischaemic stroke ★

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:
- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
    - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
    - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the

onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:
- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.
- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
    - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
    - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Absolute	Relative
- Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke

- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 11 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 16 16

Stroke and TIA guidelines

Age and Ageing 21 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

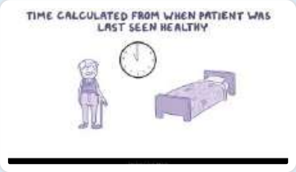
2023 National Clinical Guideline for Stroke

NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia
Acute ischaemic Stroke

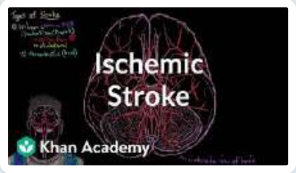
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

Khan Academy - YouTube 👍 1 👎 1

Report broken media

Score: **20%**

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Question 53 of 409

A 66-year-old male is brought to the emergency department by ambulance complaining of an abrupt onset of bilateral weakness progressing over the last 4 hours. He has a past medical hypertension and hypercholesterolaemia. His medications include amlodipine, ramipril and atorvastatin. He does not smoke cigarettes.

On examination, there is bilateral flaccid weakness of the lower limbs, with power 0/5 in all muscle groups. The patient is areflexic. There is loss of pain and temperature sensation from the level of the umbilicus to the feet. Proprioception and vibration sense is preserved.

Over the course of the next few weeks, the weakness becomes spastic in nature. The lower limbs develop hyperreflexia and the plantar reflexes are now noted to be upgoing.

What is the likely diagnosis?

- ○ Anterior spinal artery occlusion
- ○ Basilar artery occlusion
- ○ Cauda equina syndrome
- ○ Posterior spinal artery occlusion
- ○ Vitamin B 12 deficiency

Submit answer

Reference ranges

Score: **22%**

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On examination, there is bilateral flaccid weakness of the lower limbs, with power 0/5 in all muscle groups. The patient is areflexic. There is loss of pain and temperature sensation from the level of the umbilicus to the feet. Proprioception and vibration sense is preserved.

Over the course of the next few weeks, the weakness becomes spastic in nature. The lower limbs develop hyperreflexia and the plantar reflexes are now noted to be upgoing.

What is the likely diagnosis?

Anterior spinal artery occlusion	77%
Basilar artery occlusion	2%
Cauda equina syndrome	7%
Posterior spinal artery occlusion	8%
Vitamin B 12 deficiency	5%

Bilateral spastic paresis and loss of pain and temperature sensation - anterior spinal artery occlusion

Important for me Less important

Anterior spinal artery occlusion is the correct answer. The patient has risk factors for atherosclerosis (hypertension, hypercholesterolaemia) and this is a common cause of anterior spinal artery occlusion. An infarct of this artery causes bilateral weakness below the level of the lesion (in this case, the umbilicus suggests T10 as the level) associated with loss of pain and temperature sensation. This anterior cord syndrome is caused by injury to the spinothalamic and corticospinal tracts. Initially there were will be a flaccid paralysis but this will become more reflective of an upper motor neuron lesion with development of spastic paraparesis over the following days to weeks.

Basilar artery occlusion is incorrect. This causes 'locked-in syndrome' which causes complete paralysis of voluntary muscles, except for the muscles that control the eyes.

Posterior spinal artery occlusion is incorrect. This causes a dorsal cord syndrome characterized by loss of proprioception and vibration.

Vitamin B 12 deficiency is incorrect. This causes subacute combined degeneration of the spinal cord if severe. However, the onset would be more insidious and there would be mixed upper motor and lower motor neuron signs in the lower limbs that would occur concurrently e.g. absent ankle reflexes but upgoing plantar reflexes.

Cauda equina syndrome is incorrect. This causes lower motor neuron weakness. It would not progress to a spastic paraparesis.

Discuss (4) Improve

Next question

Spinal cord lesions

The diagram belows shows cross-section view of the spinal cord:

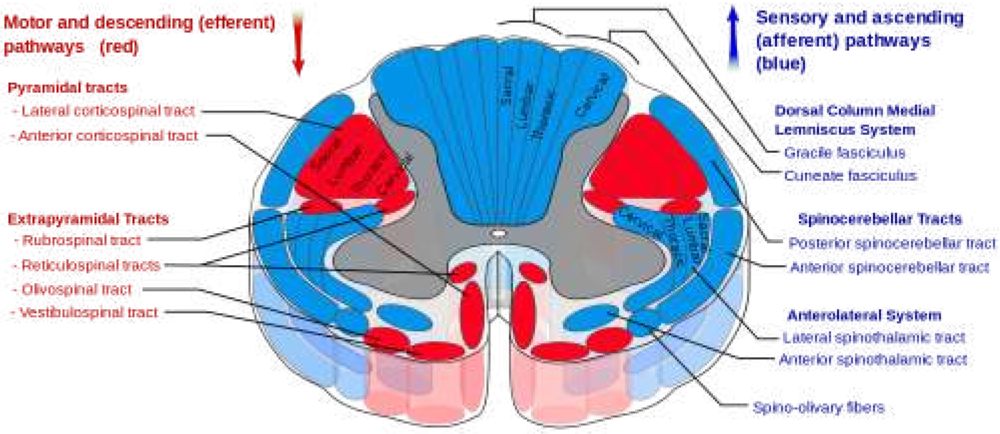

Motor and descending (efferent) pathways (red)
Pyramidal tracts
- Lateral corticospinal tract
- Anterior corticospinal tract
Extrapyramidal Tracts
- Rubrospinal tract
- Reticulospinal tracts
- Olivospinal tract
- Vestibulospinal tract
Sensory and ascending (afferent) pathways (blue)
Dorsal Column Medial Lemniscus System
Gracile fasciculus
Cuneate fasciculus
Spinocerebellar Tracts
Posterior spinocerebellar tract
Anterior spinocerebellar tract
Anterolateral System
Lateral spinothalamic tract
Anterior spinothalamic tract
Spino-olivary fibers
Sacral
Lumbar
Thoracic
Cervical

Image sourced from Wikipedia

Motor lesions

Amyotrophic lateral sclerosis (motor neuron disease)

- affects both upper (corticospinal tracts) and lower motor neurons
- results in a combination of upper and lower motor neuron signs

Poliomyelitis

- affects anterior horns resulting in lower motor neuron signs

Combined motor and sensory lesions

Disorder	Tracts affected	Clinical notes
Brown-Sequard syndrome (spinal cord hemisection)	1. Lateral corticospinal tract 2. Dorsal columns 3. Lateral spinothalamic tract	1. Ipsilateral spastic paresis below lesion 2. Ipsilateral loss of proprioception and vibration sensation 3. Contralateral loss of pain and temperature sensation
Subacute combined degeneration of the spinal cord (vitamin B12 & E deficiency)	1. Lateral corticospinal tracts 2. Dorsal columns 3. Spinocerebellar tracts	1. Bilateral spastic paresis 2. Bilateral loss of proprioception and vibration sensation 3. Bilateral limb ataxia
Friedrich's ataxia	Same as subacute combined degeneration of the spinal cord (see above)	Same as subacute combined degeneration of the spinal cord (see above) In addition cerebellar ataxia → other features e.g. intention tremor
Anterior spinal artery occlusion	1. Lateral corticospinal tracts 2. Lateral spinothalamic tracts	1. Bilateral spastic paresis 2. Bilateral loss of pain and temperature sensation
Syringomyelia	1. Ventral horns 2. Lateral spinothalamic tract	1. Flacid paresis (typically affecting the intrinsic hand muscles) 2. Loss of pain and temperature sensation
Multiple sclerosis	Asymmetrical, varying spinal tracts involved	Combination of motor, sensory and ataxia symptoms

Sensory lesions

Disorder	Tracts affected	Clinical notes
Neurosyphilis (tabes dorsalis)	1. Dorsal columns	1. Loss of proprioception and vibration sensation

+ Q 123

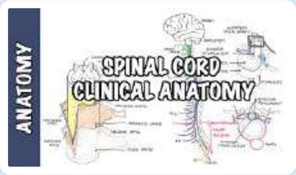
Next question >

Textbooks

High-yield textbook

Extended textbook

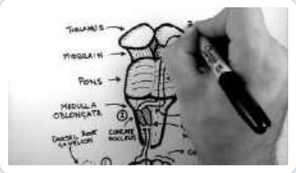
Media


ANATOMY
SPINAL CORD
CLINICAL ANATOMY

Spinal Cord - Clinical Anatomy and Physiology (dermatomes, blood supply, shingles, lumbar puncture)

Armando Hasudungan - YouTube

👍 7 👎 0


THALAMUS
MIDBRAIN
PONS
MEDULLA OBLONGATA

Spinal Pathways 2 - Dorsal Column - Medial Lemniscus Pathway

Handwritten Tutorials - YouTube

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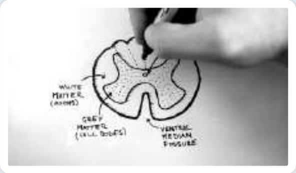
WHITE MATTER (AXONS)
GREY MATTER (CELL BODIES)
VENTRAL MEDIAN FISSURE

Spinal Pathways 1 - Spinal Cord Anatomy and Organisation

Handwritten Tutorials - YouTube

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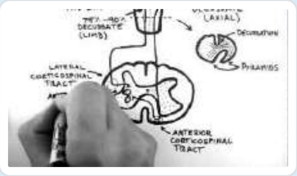

[Spinal Pathways 4 - Corticospinal Tract]

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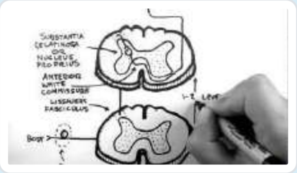

[Spinal Pathways 3 - Spinothalamic Tract]

Handwritten Tutorials - YouTube 👍 1 👎 0

[Neurology - Spinal Cord Introduction]

Armando Hasudungan - YouTube 👍 0 👎 0

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Question 54 of 409

A 78-year-old male with a background of atrial fibrillation, ischaemic heart disease, hypertension is brought to hospital by his wife who is concerned about a recent deterioration in his health. For the last few weeks his wife has noticed that the patient has been more unsteady on his feet which has resulted in falls around the house. His wife also informs you that the patient has been more forgetful and less interested in his hobbies than usual. When you ask the patient about his symptoms, his main concern is that he has recently developed problems with urinary incontinence which he finds embarrassing.

As part of the investigation into this patient's condition, a CT scan of the patient's head is requested. What is the most likely finding?

- ○ Diffuse cerebral atrophy with enlargement of cortical sulci and ventricles ×
- ○ A hyper-dense bi-convex extra-axial collection beneath the squamous part of the temporal bone ×
- ○ Small areas of low density in the distribution of the middle cerebral artery, suggestive of old lacunar infarcts ×
- ○ A concave-shaped extra-axial collection with increased attenuation ×
- ○ Enlarged third and lateral ventricles, disproportionate to the enlargement of the cortical sulci ×

Submit answer

Reference ranges

Score: **22%**

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Question 54 of 409 ✓

A 78-year-old male with a background of atrial fibrillation, ischaemic heart disease, hypertension is brought to hospital by his wife who is concerned about a recent deterioration in his health. For the last few weeks his wife has noticed that the patient has been more unsteady on his feet which has resulted in falls around the house. His wife also informs you that the patient has been more forgetful and less interested in his hobbies than usual. When you ask the patient about his symptoms, his main concern is that he has recently developed problems with urinary incontinence which he finds embarrassing.

As part of the investigation into this patient's condition, a CT scan of the patient's head is requested. What is the most likely finding?

Diffuse cerebral atrophy with enlargement of cortical sulci and ventricles	11%
A hyper-dense bi-convex extra-axial collection beneath the squamous part of the temporal bone	1%
Small areas of low density in the distribution of the middle cerebral artery, suggestive of old lacunar infarcts	2%
A concave-shaped extra-axial collection with increased attenuation	2%
Enlarged third and lateral ventricles, disproportionate to the enlargement of the cortical sulci	84%

Normal pressure hydrocephalus neuroimaging findings: ventriculomegaly in the absence of, or out of proportion to, sulcal enlargement

Important for me Less important

The patient's history of atrial fibrillation places him at increased risk of stroke and so it may be tempting to attribute his deterioration to cerebrovascular disease. Another pitfall with this question is the report of falls at home, which may lead to a suspicion that the patient has developed a subdural haematoma (SDH). However, it is important to appreciate that his symptom of ataxia pre-dates his tendency to fall. The critical part of this question is that this patient's presenting complaints of ataxia, dementia and urinary incontinence are the classical triad of clinical findings associated with normal pressure hydrocephalus. As such, a CT scan of his head would be expected to show ventricles which are dilated out of proportion to the enlargement of his sulci.

Normal pressure hydrocephalus

Normal pressure hydrocephalus is a reversible cause of dementia seen in elderly patients. It is thought to be secondary to reduced CSF absorption at the arachnoid villi. These changes may be secondary to head injury, subarachnoid haemorrhage or meningitis.

A classical triad of features is seen
- urinary incontinence
- dementia and bradyphrenia
- gait abnormality (may be similar to Parkinson's disease)

It is thought around 60% of patients will have all 3 features at the time of diagnosis. Symptoms typically develop over a few months.

Imaging
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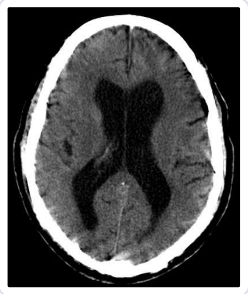

Management
- ventriculoperitoneal shunting
- around 10% of patients who have shunts experience significant complications such as seizures, infection and intracerebral haemorrhages

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YouTube 👍 26 👎 2

Typical NPH Gait

Suggest link Report broken link

Score: **20%**

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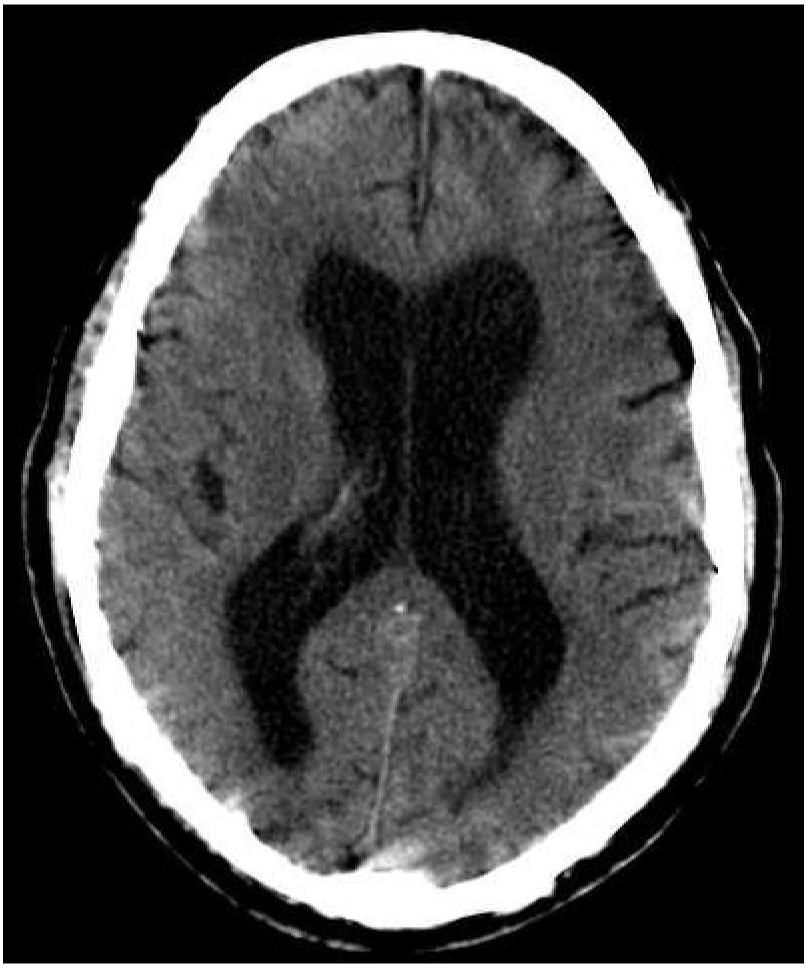
A 62-year-old man is reviewed in the neurology clinic due to a gait disorder for the last 2 months. His wife tells that he has become forgetful and has difficulty continuing his job. The patient has a history of hospital admission 4 months back due to bacterial meningitis requiring intravenous antibiotics. Past medical history is significant for hypertension well-controlled on amlodipine 10 mg once daily.

A lumbar puncture is performed and an interpretation of the CSF analysis is shown below:

Appearance	Clear, colourless	(Clear, colourless)
Opening pressure	6 mmHg	(8-16)
Cells	4 cells /mm ^{3}	(0-4)
Glucose	3.9 mmol/L	(2.5-4.4)
Protein	50 mg/100 mL	(20-60)

Blood glucose	5.6 mmol/L

A CT head is performed, as shown below:



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What is the most appropriate treatment option for this patient?

- ○ Intravenous corticosteroids ×
- ○ Intravenous mannitol ×
- ○ Oral memantine ×
- ○ Therapeutic lumbar puncture ×
- ○ Ventriculoperitoneal (VP) shunting ×

Submit answer

Reference ranges ⌄

Score: **22%**

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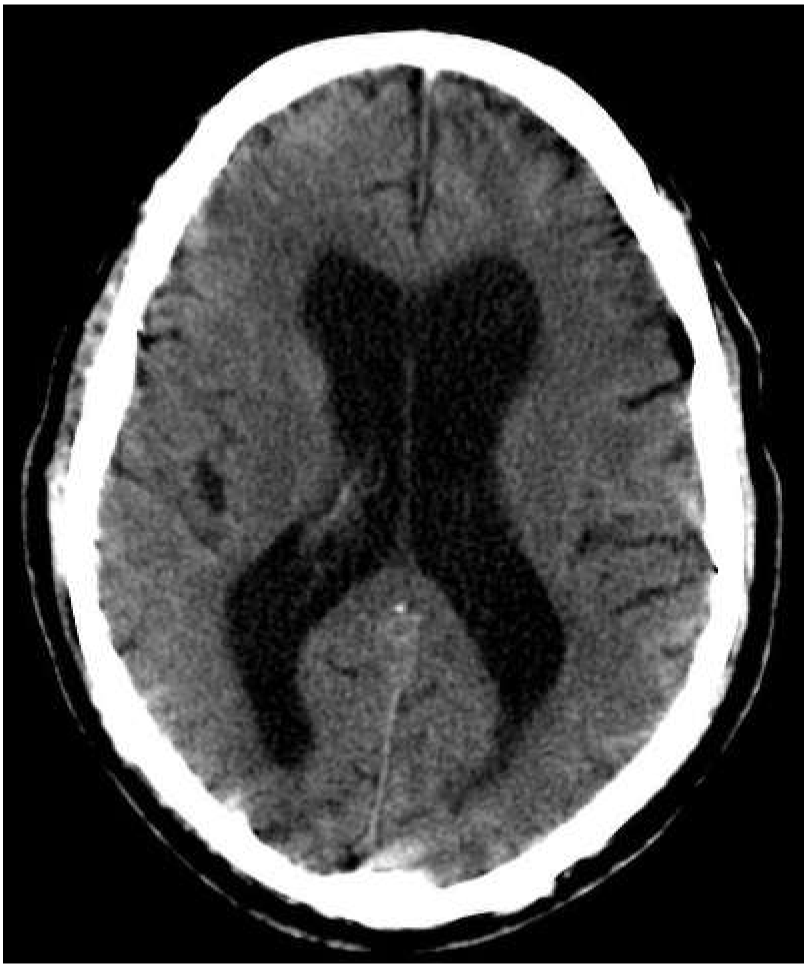

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What is the most appropriate treatment option for this patient?

Intravenous corticosteroids	3%
Intravenous mannitol	3%
Oral memantine	3%
Therapeutic lumbar puncture	13%
Ventriculoperitoneal (VP) shunting	79%

The scenario describes a patient with normal pressure hydrocephalus, for which the definitive treatment is **ventriculoperitoneal shunting** . The patient has gait apraxia and dementia that started 2 months after meningitis. The CT scan shows enlarged frontal, temporal, and occipital horns of the lateral ventricles disproportionate to the sulci, which appear normal in size. These CT findings when combined with the patient's history suggest the diagnosis of normal pressure hydrocephalus (NPH) in this scenario. NPH can occur following meningitis or subarachnoid haemorrhage due to reduced CSF absorption by arachnoid villi. The classical triad of NPH includes gait apraxia, dementia, and urinary incontinence. LP reveals a normal or low opening pressure which is also hinted at in this question. The definitive treatment for NPH is VP shunting. Symptoms of NPH including dementia are reversed with successful VP shunting in 60-90 % of the patients. 10% develop complications like postural headache, subdural effusion, shunt infection, and bacterial meningitis.

The history of gait problems and dementia in this scenario may suggest Alzheimer's disease for which **memantine** is the most appropriate treatment option. However, the acute onset of symptoms and normal sulci on CT scan in this scenario rule out Alzheimer's disease. Neuroimaging in Alzheimer's reveals dilated ventricles with enlarged sulci and decreased volume gyri.

Intravenous mannitol is unlikely to benefit this patient with NPH. It is an osmotic diuretic and helps in decreasing intracranial pressure.

There is no role of **intravenous corticosteroids** in treating NPH. Corticosteroids are used to reduce hearing loss and neurological sequelae in meningitis.

Therapeutic lumbar puncture (LP) treats conditions with increased intracranial pressure without a space-occupying lesion. It is an important treatment option for idiopathic intracranial hypertension. This patient with NPH requires continuous drainage of CSF which is not possible via LP. A lumbar peritoneal (LP) shunt may be used as an alternative to VP shunt to treat NPH.

👍 👎 Discuss (6) Improve

Next question >

Normal pressure hydrocephalus ★

Normal pressure hydrocephalus is a reversible cause of dementia seen in elderly patients. It is thought to be secondary to reduced CSF absorption at the arachnoid villi. These changes may be secondary to head injury, subarachnoid haemorrhage or meningitis.

A classical triad of features is seen
- urinary incontinence
- dementia and bradyphrenia
- gait abnormality (may be similar to Parkinson's disease)

It is thought around 60% of patients will have all 3 features at the time of diagnosis. Symptoms typically develop over a few months.

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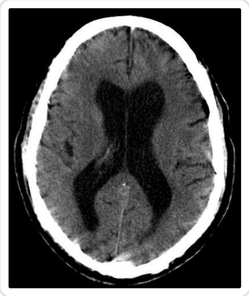

Management
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AI + Q 123

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Typical NPH Gait

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A 76-year-old gentleman with diabetes mellitus was referred to a specialist memory clinic by his family doctor. His wife was concerned as he had been more forgetful recently and had developed urinary incontinence. His family doctor had treated him with a course of trimethoprim for a urinary tract infection but his symptoms had persisted.

On examination this gentleman smelt strongly of urine. He had a broad-based, unsteady gait. Examination of his cardiovascular and respiratory systems was unremarkable but on abdominal examination there was suprapubic tenderness. He was orientated to place but scored only 22/30 on a Mini Mental State Examination (MMSE).

Urinalysis:

Blood -
Protein Trace
Nitrites -
White cells +
Microscopy 50 white cells seen, bacteria +
Cultures Mixed growth, please send repeat sample

Magnetic Resonance Imaging (MRI) of the brain: 'Disproportionately enlarged ventricular system compared with the degree of sulcal atrophy.'

Lumbar Puncture (LP):

Opening pressure	18 cmH20 (12 20)
Appearance	Clear and colourless
Red cells	4
White cells	1 (0 5)
Gram stain	No organisms seen
Culture	No organisms seen
Protein	30 mg/100ml (15 60)
Glucose	98mg/100ml (50 80)
Virology	No viruses detected on PCR
Cytology	No abnormal cells seen
Timed 10 metre walk pre-LP	15 seconds
Timed 10 metre walk post-40 mL CSF drainage	12 seconds

Given the underlying diagnosis what is the definitive treatment of choice?

○ Repeated therapeutic lumbar punctures

○ Acetazolamide

○ Referral for a ventriculo-peritoneal shunt

○ Vitamin B12 and folate replacement

○ Referral for lumbar drain insertion

Submit answer

Reference ranges ∨

Score: **22%**

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Timed 10 metre walk pre-LP	15 seconds
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Given the underlying diagnosis what is the definitive treatment of choice?

Repeated therapeutic lumbar punctures	5%
Acetazolamide	8%
Referral for a ventriculo-peritoneal shunt	84%
Vitamin B12 and folate replacement	1%
Referral for lumbar drain insertion	2%

This gentleman presents with all three components of the classic triad of Normal Pressure Hydrocephalus (NPH): urinary incontinence, ataxia and memory impairment. He may also have a co-existent urinary tract infection but this alone cannot explain his presentation. Further evidence to support a diagnosis of NPH includes the MRI scan, which shows ventricular enlargement, and the improvement in his 10 metre walk time following therapeutic CSF drainage.

This gentleman needs a referral to the neurosurgical team for consideration of a definitive surgical procedure. There are alternative surgical options and the specifics of each particular case will dictate which approach is used. The most commonly performed procedure is ventriculo-peritoneal shunting as this has a lower failure rate than alternative procedures.

Repeated therapeutic lumbar punctures (LP) can be performed but this approach has fallen out of favour as it is unpopular with patients and exposes them repeatedly to an invasive procedure with associated risks.

Acetazolamide is a diuretic used to treat Idiopathic Intracranial Hypertension and does not have a role in NPH.

Vitamin B12 and folate replacement is not indicated in this case although vitamin B12 deficiency can cause memory impairment and walking problems (subacute combined degeneration of the cord).

A lumbar drains diverts CSF from the lumbar subarachnoid space to the outside world. It can be useful in determining whether or not a patient may respond well to a shunting procedure but due to the high risk of infection it is not in itself a definitive treatment for NPH.

👍 👎 Discuss (5) Improve

Next question ›

Normal pressure hydrocephalus ★

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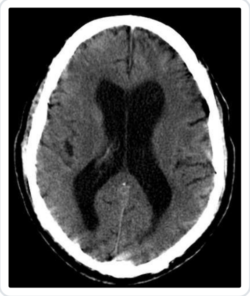

Management
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+ Q 123

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Typical NPH Gait

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Question 57 of 409

Vincent, 28, has treatment resistant schizophrenia, with his usual symptoms being auditory hallucinations and persecutory delusions. He was recently prescribed clozapine, fluoxetine and lactulose. He has been complaining of constipation recently, but now presents to the emergency department with acute abdominal pain and vomiting. On examination abdomen is distended. What is the most likely cause?

- ○ Intestinal obstruction
- ○ Appendicitis
- ○ Constipation
- ○ A bezoar
- ○ Ingestion of foreign object

Submit answer

Reference ranges

Score: **22%**

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Question 57 of 409 ✗

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Intestinal obstruction	58%
Appendicitis	1%
Constipation	21%
A bezoar	17%
Ingestion of foreign object	3%

One of the most common side effects of clozapine is constipation/intestinal obstruction

Important for me Less important

The most likely cause of this patients presentation is intestinal obstruction. Intestinal obstruction is one of the more under-recognised complications of clozapine therapy, yet one of the more serious. Gastric hypomotility is common in patients who are treated with clozapine, and its presentation can range from a simple constipation to more severe conditions such as intestinal obstruction, bowel ischaemia and necrosis.

The important pieces of information to consider when answering this question are the recent prescription of clozapine, the presence of constipation previous to current presentation, and his physical presentation of acute abdominal pain and vomiting with distension. When answering a question such as this, recent prescriptions of medications prior to the deterioration of their physical condition should raise the thought that maybe one of the medications is causing the symptoms. In this scenario, the patient has recently been prescribed clozapine, fluoxetine and lactulose. Therefore, considering each of these medications and their side effects is a must.

The patients presentation, and recent past medical history, provides a good indication of what could be the underlying issue. The patient has presented with acute abdominal pain and vomiting. When you consider this with the recently experienced constipation, it becomes clear that this patient is likely suffering from an obstruction, as opposed to the other options on offer.

Constipation would be an inappropriate answer to this question. Although he has had constipation recently, and abdominal pain could be a presentation of constipation, vomiting is not generally observed in constipation. This would means that this diagnosis is less likely.

Bezoars are indigestible masses that become trapped in the gastrointestinal tract. They can occur when individuals consume a variety of items, including hair, soil, chewing gum etc. These items form a mass, which ultimately becomes lodged in the gastrointestinal tract, often requiring surgical intervention to relieve the obstruction. The symptoms that the patient in this scenario has experienced could be explained by a bezoar, however, we do not have any evidence that he consumes any items that may lead to a bezoar. Furthermore, the recent commencement of clozapine means that we cannot select a bezoar as the most likely cause of this patients symptoms above intestinal obstruction. Similarly, we do not have any evidence that the patient ingests foreign objects, meaning that this cannot be classed as a likely cause in this question.

Appendicitis could explain the acute presentation of this patient. However, the fact that the patient experienced constipation prior to him developing acute abdominal pain and vomiting means that appendicitis is less likely.

👍 👎 Discuss (5) Improve

Next question >

Clozapine ★

Clozapine, one of the first atypical agents to be developed, carries a significant risk of agranulocytosis and full blood count monitoring is therefore essential during treatment. For this reason, clozapine should only be used in patients resistant to other antipsychotic medication. The BNF states:

> *Clozapine should be introduced if schizophrenia is not controlled despite the sequential use of two or more antipsychotic drugs (one of which should be a second-generation antipsychotic drug), each for at least 6-8 weeks.*

Adverse effects of clozapine

- agranulocytosis (1%), neutropaenia (3%)
- reduced seizure threshold - can induce seizures in up to 3% of patients
- constipation
- myocarditis: a baseline ECG should be taken before starting treatment

- hypersalivation

Dose adjustment of clozapine might be necessary if smoking is started or stopped during treatment.

123

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BNF 4 2

Clozapine

Clinical Knowledge Summaries 3 0

Schizophrenia > Prescribing Information > Adverse effects

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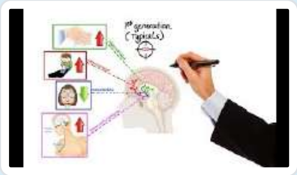
Media



Antipsychotics

Speed Pharmacology - YouTube 2 0

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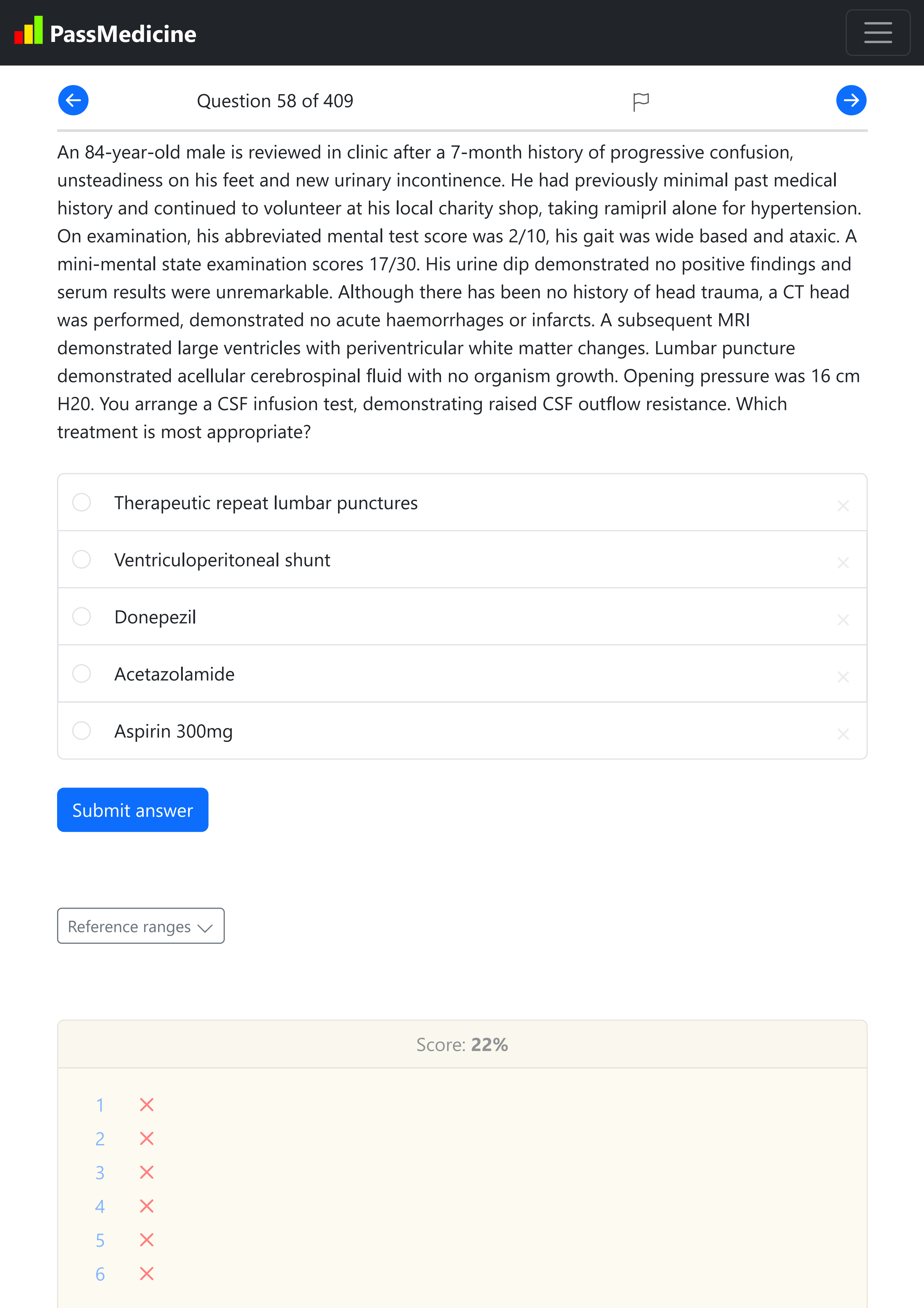

An 84-year-old male is reviewed in clinic after a 7-month history of progressive confusion, unsteadiness on his feet and new urinary incontinence. He had previously minimal past medical history and continued to volunteer at his local charity shop, taking ramipril alone for hypertension. On examination, his abbreviated mental test score was 2/10, his gait was wide based and ataxic. A mini-mental state examination scores 17/30. His urine dip demonstrated no positive findings and serum results were unremarkable. Although there has been no history of head trauma, a CT head was performed, demonstrated no acute haemorrhages or infarcts. A subsequent MRI demonstrated large ventricles with periventricular white matter changes. Lumbar puncture demonstrated acellular cerebrospinal fluid with no organism growth. Opening pressure was 16 cm H20. You arrange a CSF infusion test, demonstrating raised CSF outflow resistance. Which treatment is most appropriate?

○ Therapeutic repeat lumbar punctures ×

○ Ventriculoperitoneal shunt ×

○ Donepezil ×

○ Acetazolamide ×

○ Aspirin 300mg ×

Score: **22%**

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Therapeutic repeat lumbar punctures	8%
Ventriculoperitoneal shunt	80%
Donepezil	2%
Acetazolamide	10%
Aspirin 300mg	0%

The history and examination are consistent with normal pressure hydrocephalus, with the triad of new onset dementia, urinary incontinence and gait disturbance. The key points in the investigations indicate the suitability of the patient for a ventriculoperitoneal shunt: the raised CSF resistance on CSF infusion test indicates impaired CSF absorption and correlates towards surgical improvement. In addition, prominent gait disturbance also points towards an increased likelihood of surgical success.

Discuss (6) | Improve

Next question

Normal pressure hydrocephalus

Normal pressure hydrocephalus is a reversible cause of dementia seen in elderly patients. It is thought to be secondary to reduced CSF absorption at the arachnoid villi. These changes may be secondary to head injury, subarachnoid haemorrhage or meningitis.

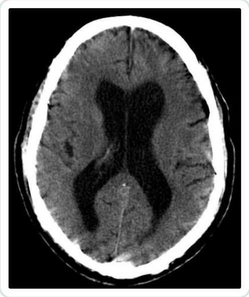
A classical triad of features is seen

- urinary incontinence
- dementia and bradyphrenia
- gait abnormality (may be similar to Parkinson's disease)

It is thought around 60% of patients will have all 3 features at the time of diagnosis. Symptoms typically develop over a few months.

Imaging

- hydrocephalus with ventriculomegaly in the absence of, or out of proportion to, sulcal enlargement



Management

- ventriculoperitoneal shunting
- around 10% of patients who have shunts experience significant complications such as seizures, infection and intracerebral haemorrhages

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

YouTube 👍 26 👎 2

Typical NPH Gait

Suggest link

Report broken link

Score: **20%**

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A 78-year-old male presents with a one-year history of progressive unsteadiness on walking. He had previously been extremely fit and healthy, walking around 2 miles to the shops every day and only retired as a publicist 3 years ago. He underwent radiotherapy for localised squamous cell carcinoma of his vocal cords 18 months ago but otherwise had no other past medical history. He admits to having drunk 'more than he should have' while working in the city but says he has since cut down to moderate levels. He stopped smoking 5 years ago, with a 30 pack year history. Over the past 3 months, he has become incontinent of urine and has to rely on pads, which he is greatly embarrassed by.

On examination, he has a shuffling gait in his lower limbs with good arm swing. He is markedly slowed and takes 120 seconds to walk 20 metres. He turns around 180 degrees in 6 steps with no resting tremor, rigidity or bradykinesia. Examination of his tone, power, sensation, coordination and reflexes are all unremarkable. His voice is quiet and whispering. His cranial nerves are normal with a full range of eye movements. An abbreviated mental test scores 9/10, a Montreal cognitive assessment (MOCA) scored 29/30. His initial blood tests are as follows:

Hb	94 g/l
MCV	103.3 fl
Platelets	232 * 10^9/l
WBC	6.3 * 10^9/l

Na^+	139 mmol/l
K^+	4.9 mmol/l
Urea	7.5 mmol/l
Creatinine	98 µmol/l
CRP	2 mg/l
Vitamin B12	327ng/l
Folate	5.2 nmol/l

An MRI head is performed, demonstrating diffuse mild microangiopathic changes with prominently dilated lateral and third ventricles. No intracranial masses are noted. You perform a lumbar puncture, with the patient lying in the left lateral position using a 22G spinal needle and obtain cerebrospinal fluid with the first pass. His opening pressure is 16.6 cmH2O. What is the most appropriate course of action?

○ Remove 30mls cerebrospinal fluid and refer to neurosurgeons ✕

○ Prescribe urgent intravenous pabrinex ×

○ Urgent urological referral for urodynamic studies +/- flexible cystoscopy ×

○ Prescribe Sinemet 250 with anti-emetic and re-measure walking time ×

○ Urgent CT angiography and prescribe aspirin 300mg ×

Submit answer

Reference ranges ⌄

Score: **22%**

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Remove 30mls cerebrospinal fluid and refer to neurosurgeons 82%

Prescribe urgent intravenous pabrinex	7%
Urgent urological referral for urodynamic studies +/- flexible cystoscopy	3%
Prescribe Sinemet 250 with anti-emetic and re-measure walking time	6%
Urgent CT angiography and prescribe aspirin 300mg	2%

There are two of three features of the classic triad of gait disturbance, urinary incontinence and cognitive impairment, suggestive of normal pressure hydrocephalus (NPH). The normal opening pressure and dilated prominent ventricles lend further support to this diagnosis. Mild microangiopathic disease is not uncommon in this age group and potentially could cause gait disturbances; there is no indication of an acute ischaemic stroke secondary to embolic events however, urgent vascular imaging and aspirin loading would thus be inappropriate. Despite the previous alcohol history, the patient's vitamin B12 and folate are within normal range with no neuroimaging features of degeneration, intravenous Pabrinex is unlikely to resolve these symptoms. Although there may be a primary urological cause for the patient's urological symptoms, his gait features are not easily explained. The patient does not display any 'hard signs' of Parkinsonism to warrant a Sinemet trial.

The definitive treatment for normal pressure hydrocephalus is neurosurgical implantation of a ventricular shunt, most commonly into the peritoneum. Of the three main features of NPH, gait apraxia is the most commonly improved feature with shunting, while dementia and cognitive impairment rarely improve after surgery. Despite the normal pressure, removal of a relatively significant amount of CSF and repeat of the patients walking time is a useful clinical predictor of whether the patient will improve after surgery.

👍 👎 Discuss (6) Improve

Next question >

Normal pressure hydrocephalus ★

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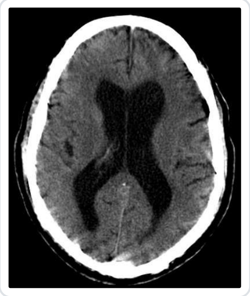

Management
- ventriculoperitoneal shunting
- around 10% of patients who have shunts experience significant complications such as seizures, infection and intracerebral haemorrhages

＋ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

YouTube 26 2

Typical NPH Gait

Suggest link Report broken link

Score: **20%**

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A 35-year-old woman is referred to a headache clinic with frequent migraines over the last 9 months. They occur unpredictably, around 3 times a month. Her past medical history includes eczema, brittle asthma and depression. She is not sexually active.

She is keen on something to reduce the frequency of her migraines. In the past, she has tried topiramate but stopped as she felt it caused her to get eye discomfort.

What is the most appropriate management option?

- ○ Candesartan ×
- ○ Pizotifen ×
- ○ Propranolol ×
- ○ Re-trial topiramate ×
- ○ Zolmitriptan ×

Submit answer

Reference ranges

Score: **22%**

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She is keen on something to reduce the frequency of her migraines. In the past, she has tried topiramate but stopped as she felt it caused her to get eye discomfort.

What is the most appropriate management option?

Candesartan	41%
Pizotifen	14%
Propranolol	29%
Re-trial topiramate	3%
Zolmitriptan	14%

Candesartan may be considered for migraine prophylaxis if other first-line options either don't work or are contradicted

Important for me   Less important

Either topiramate or propranolol is usually used first-line for prophylaxis of migraine. When standard treatments are not appropriate then other treatment options may be considered by specialists, but these are outside the remit of NICE guidelines. **Candesartan** is recommended by the British Association for the Study of Headache guidelines.

Pizotifen is no longer recommended as adverse effects such as weight gain and drowsiness are common.

In this case, **topiramate** has caused side effects on previous use. Eye discomfort is a recognised uncommon side effect and can rarely cause eye inflammation. Hence, this should be avoided.

Propranolol is best avoided given the risk of bronchospasm, listed as an uncommon side effect in the BNF. This is because this patient is asthmatic and particularly given that it is brittle asthma.

Zolmitriptan can be used as a type of 'mini-prophylaxis' for women with predictable menstrual migraine. However, this patient suffers from unpredictable migraines so this is not likely to be effective.

👍 👎 Discuss (2) Improve

Next question >

Migraine: management ★

It should be noted that as a general rule 5-HT receptor agonists are used in the acute treatment of migraine whilst 5-HT receptor antagonists are used in prophylaxis. NICE produced guidelines in 2012 on the management of headache, including migraines.

Acute treatment

- first-line: offer combination therapy with
  - an oral triptan and an NSAID, or
  - an oral triptan and paracetamol
- for young people aged 12-17 years consider a nasal triptan in preference to an oral triptan
- if the above measures are not effective or not tolerated offer a non-oral preparation of metoclopramide or prochlorperazine and consider adding a non-oral NSAID or triptan
  - caution should be exercised when prescribing metoclopramide to young patients as acute dystonic reactions may develop

Prophylaxis

- prophylaxis should generally be given if '*Migraine attacks are having a significant impact on quality of life and daily function, for example they occur frequently (more than once a week on average) or are prolonged and severe despite optimal acute treatment*'
- options:
  - propranolol
  - topiramate: should be avoided in women of childbearing age as it may be teratogenic and it can reduce the effectiveness of hormonal contraceptives
  - amitriptyline
- if these measures fail NICE recommends 'a course of up to 10 sessions of acupuncture over 5-8 weeks'
- NICE recommend: '*Advise people with migraine that riboflavin (400 mg once a day) may be effective in reducing migraine frequency and intensity for some people*'
- for women with predictable menstrual migraine treatment NICE recommend either frovatriptan (2.5 mg twice a day) or zolmitriptan (2.5 mg twice or three times a day) as a type of 'mini-prophylaxis'
- treatment options that may be considered by specialists, but fall outside the NICE guidelines:
  - candesartan: recommended by the British Association for the Study of Headache guidelines

  - monoclonal antibodies directed against the calcitonin gene-related peptide (CGRP) receptor: examples include erenumab
- pizotifen is no longer recommended. Adverse effects such as weight gain & drowsiness are common

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Association for the Study of Headache 14 21

Headache guidelines

Clinical Knowledge Summaries 14 7

Migraine guidelines

NICE 16 4

2012 Headache guidelines

Suggest link

Report broken link

Media

Management of Migraine
Podmedics

Migraine

Podmedics - YouTube 👍 1 👎 1

Report broken media

Score: **20%**

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A 38-year-old man with severe ulcerative colitis presents to his GP with a red right eye that has developed over the last 24 hours. He is finding it uncomfortable to move his eye in any direction but feels his vision is unchanged. On examination his left eye is normal, but his right eye is erythematous, watery, and there is asymmetric miosis. During the examination, he asks you to turn down the lights as he finds them uncomfortable.

Which of the following is the most appropriate treatment for this man's red-eye?

- ○ Supportive management
- ○ Topical atropine
- ○ Topical corticosteroids
- ○ Topical cyclopentolate and topical corticosteroids
- ○ Topical pilocarpine

Submit answer

Reference ranges

Score: **22%**

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Which of the following is the most appropriate treatment for this man's red-eye?

Supportive management	10%
Topical atropine	4%
Topical corticosteroids	31%
Topical cyclopentolate and topical corticosteroids	41%
Topical pilocarpine	14%

Anterior uveitis is most likely to be treated with a steroid + cycloplegic (mydriatic) drops

Important for me Less important

The painful eye movements, lacrimation and irregular small pupil are strongly indicative of anterior uveitis. Uveitis may be idiopathic but is often associated with HLA-B27-related diseases, such as inflammatory bowel disease (IBD). Uveitis occurs in 0.5%-3% of patients with IBD and is often bilateral, but not in this case. In terms of extraintestinal manifestations of IBD, uveitis is less common than episcleritis. However, episcleritis is not typically painful and does not present with photophobia. If episcleritis were the diagnosis, then supportive management would be appropriate.

Anterior uveitis describes inflammation of the anterior portion of the uvea - the iris and ciliary body. Uveitis does not affect visual acuity unless it involves the posterior segment - the posterior vitreous, retina, choroid, retinal vasculature and optic nerve. Uveitis can lead to blindness and should be referred to an experienced ophthalmologist. Treatment is with topical corticosteroids and cycloplegic (mydriatic) drops. Topical corticosteroids are appropriate in anterior uveitis but note that intermediate and posterior uveitis usually require locally injected or systemic corticosteroids.

Cyclopentolate is a muscarinic antagonist, and cycloplegic. It relieves the pain associated with uveitis as it dilates the pupil and prevents iridospasm. Atropine is another cycloplegic medication that can be used in anterior uveitis. Used alone it is less appropriate than the combination of cyclopentolate drops and steroids, which is the single best answer here.

Topical pilocarpine is a muscarinic receptor agonist and would cause miosis, worsening the situation here. It may be used in acute-angle glaucoma, another cause of acute red eye which would instead present with increased pain in the dark.

👍 👎 Discuss (3) Improve

Next question >

Anterior uveitis ★

Anterior uveitis is one of the important differentials of a red eye. It is also referred to as iritis. Anterior uveitis describes inflammation of the anterior portion of the uvea - iris and ciliary body. It is associated with HLA-B27 and may be seen in association with other HLA-B27 linked conditions (see below).

Features
- acute onset
- ocular discomfort & pain (may increase with use)
- pupil may be small +/- irregular due to sphincter muscle contraction
- photophobia (often intense)
- blurred vision
- red eye
- lacrimation
- ciliary flush: a ring of red spreading outwards
- hypopyon; describes pus and inflammatory cells in the anterior chamber, often resulting in a visible fluid level
- visual acuity initially normal → impaired

Associated conditions
- ankylosing spondylitis
- reactive arthritis
- ulcerative colitis, Crohn's disease
- Behcet's disease
- sarcoidosis: bilateral disease may be seen

Management
- urgent review by ophthalmology

- cycloplegics (dilates the pupil which helps to relieve pain and photophobia) e.g. Atropine, cyclopentolate
- steroid eye drops

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **20%**

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Question 62 of 409

A 70-year-old male presents with a 3-month history of left foot drop. He complains of having to lift his thighs higher than normal to accommodate this pathology. On examination, he has a high stepping gait. Power is normal in all movements except left ankle dorsiflexion (2/5) and eversion (2/5). Ankle inversion is intact (5/5), ankle jerks are present and plantars are downgoing. He reports reduced sensation on the dorsum of his foot. What is the most likely diagnosis?

- ○ L5 radiculopathy
- ○ Common peroneal palsy
- ○ Functional neurology
- ○ Sciatic nerve compression
- ○ Lumbar plexopathy

Submit answer

Reference ranges

Score: **22%**

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Question 62 of 409 ✗

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L5 radiculopathy	20%
Common peroneal palsy	75%
Functional neurology	1%
Sciatic nerve compression	2%
Lumbar plexopathy	1%

'Foot drop' - ?common peroneal nerve lesion

Important for me Less important

The differential diagnosis of foot drop is a MRCP favourite. In this case, a number of features favour a common peroneal nerve palsy instead of a L5 radiculopathy: common peroneal nerve palsy patients normally have an intact ankle inversion and flexion of the big toe. Sensory loss is usually around the lateral aspect of the lower leg and the dorsum of the foot while L5 appears as a thin strip down the middle of the anterior lower limb, usually not affect the lateral lower leg. Ankle jerks are present in both, as the tibial nerve is branched from S1 and the sciatic nerve. There is little to suggest the involvement of multiple lumbar levels in this scenario. The most common causes of common peroneal nerve palsy are trauma or compression at the fibula head, classically by tight plaster casts.

Discuss (3) Improve

Next question >

Common peroneal nerve lesion

The sciatic nerve divides into the tibial and common peroneal nerves. Injury often occurs at the neck of the fibula

The most characteristic feature of a common peroneal nerve lesion is foot drop.

Other features include:

- weakness of foot dorsiflexion
- weakness of foot eversion
- weakness of extensor hallucis longus
- sensory loss over the dorsum of the foot and the lower lateral part of the leg
- wasting of the anterior tibial and peroneal muscles

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20%**

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Question 63 of 409

A 46-year-old woman presents to her GP with a neck lump. After examination and initial investigations, she is urgently referred to the local endocrine multidisciplinary team, which perform further scans and diagnose cancer of the thyroid.

Ultimately, she undergoes a thyroidectomy. The procedure itself is uneventful.

Post-thyroidectomy, she notices that her voice has become very hoarse, which was not a symptom prior to the surgery.

What is most appropriate next?

- ○ Acoustic analysis
- ○ Chest X-ray
- ○ Laryngeal electromyography
- ○ Laryngoscopy
- ○ Reassure and monitor

Submit answer

Reference ranges

Score: **22%**

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A 46-year-old woman presents to her GP with a neck lump. After examination and initial investigations, she is urgently referred to the local endocrine multidisciplinary team, which perform further scans and diagnose cancer of the thyroid.

Ultimately, she undergoes a thyroidectomy. The procedure itself is uneventful.

Post-thyroidectomy, she notices that her voice has become very hoarse, which was not a symptom prior to the surgery.

What is most appropriate next?

Acoustic analysis	1%
Chest X-ray	2%
Laryngeal electromyography	27%
Laryngoscopy	41%
Reassure and monitor	30%

Hoarseness following total thyroidectomy should be investigated with laryngoscope

Important for me Less important

Hoarseness following thyroidectomy indicates damage to the recurrent laryngeal nerve. This is a relatively common complication of thyroidectomy surgery. The correct answer is **laryngoscopy** - this should be performed if hoarseness develops, to assess for vocal cord paralysis and evaluate motility.

Acoustic analysis - that is, assessment of the change in voice itself - is insufficient. This may help to give an idea of the extent to which the hoarseness has affected speech, but more objective assessment is required via laryngoscopy.

Chest X-ray is inappropriate. This would not allow for assessment of the vocal cords. Laryngoscopy is required.

Laryngeal electromyography can be used to assess vocal cord paralysis and estimate prognosis.

However, first-line, laryngoscopy is generally performed before any further investigation.

Reassurance and monitoring would be inappropriate. Further assessment is required here, given the new onset of hoarseness following thyroidectomy.

Discuss (4) Improve

Next question

Subtotal thyroidectomy

Surgery complications
- hypothyroidism: most common
- immediate haematoma (airway compression)
- hypoparathyroidism → hypocalcaemia (paraesthesia, spasm)
- recurrent laryngeal nerve palsy (hoarseness, bovine cough)
- superior laryngeal nerve injury (loss of pitch)
- thyroid crisis (fast AF, pulmonary oedema)

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **20%**

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Question 64 of 409

A 40-year-old lady with myasthenia gravis is admitted under the medical team with pneumonia. She is also experiencing a flare of her myasthenia symptoms. She is treated with antibiotics, pyridostigmine and methylprednisolone begins to improve clinically. She is also given IV magnesium replacement to correct hypomagnesemia.

The next day her symptoms suddenly worsen and she develops profound weakness on exertion and new dysphagia requiring insertion of a nasogastric (NG) tube.

Which of the following drugs administered by the medical team is the most likely cause?

- ○ IV magnesium ×
- ○ Co-amoxiclav ×
- ○ Low molecular weight heparin ×
- ○ Cyclizine ×
- ○ Naproxen ×

Submit answer

Reference ranges

Score: **22%**

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The next day her symptoms suddenly worsen and she develops profound weakness on exertion and new dysphagia requiring insertion of a nasogastric (NG) tube.

Which of the following drugs administered by the medical team is the most likely cause?

IV magnesium	40%
Co-amoxiclav	24%
Low molecular weight heparin	2%
Cyclizine	22%
Naproxen	11%

Don't given IV magnesium in myasthenia as can precipitate a crisis

Important for me Less important

The following drugs may exacerbate myasthenia:

- Magnesium
- Penicillamine
- Quinidine
- Procainamide
- Beta-blockers
- Lithium
- Phenytoin
- Antibiotics: gentamicin, macrolides, quinolones, tetracyclines

Discuss (10) Improve

Next question

Myasthenia gravis: exacerbating factors

The most common exacerbating factor is exertion resulting in fatigability, which is the hallmark feature of myasthenia gravis.. Symptoms become more marked during the day

The following drugs may exacerbate myasthenia:

- penicillamine
- quinidine, procainamide
- beta-blockers
- lithium
- phenytoin
- antibiotics: gentamicin, macrolides, quinolones, tetracyclines

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians (Edin) 👍 4 👎 2

Myasthenia gravis review

Myasthenia Gravis Association 👍 9 👎 3

Drugs which may aggravate myasthenia gravis

Suggest link

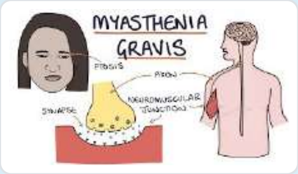
Report broken link

Media

MYASTHENIA
GRAVIS

[Myasthenia gravis - causes, symptoms, treatment, pathology]

Osmosis - YouTube 👍 1 👎 0


MYASTHENIA
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Myasthenia gravis

Zero to Finals - YouTube 👍 2 👎 1

Report broken media

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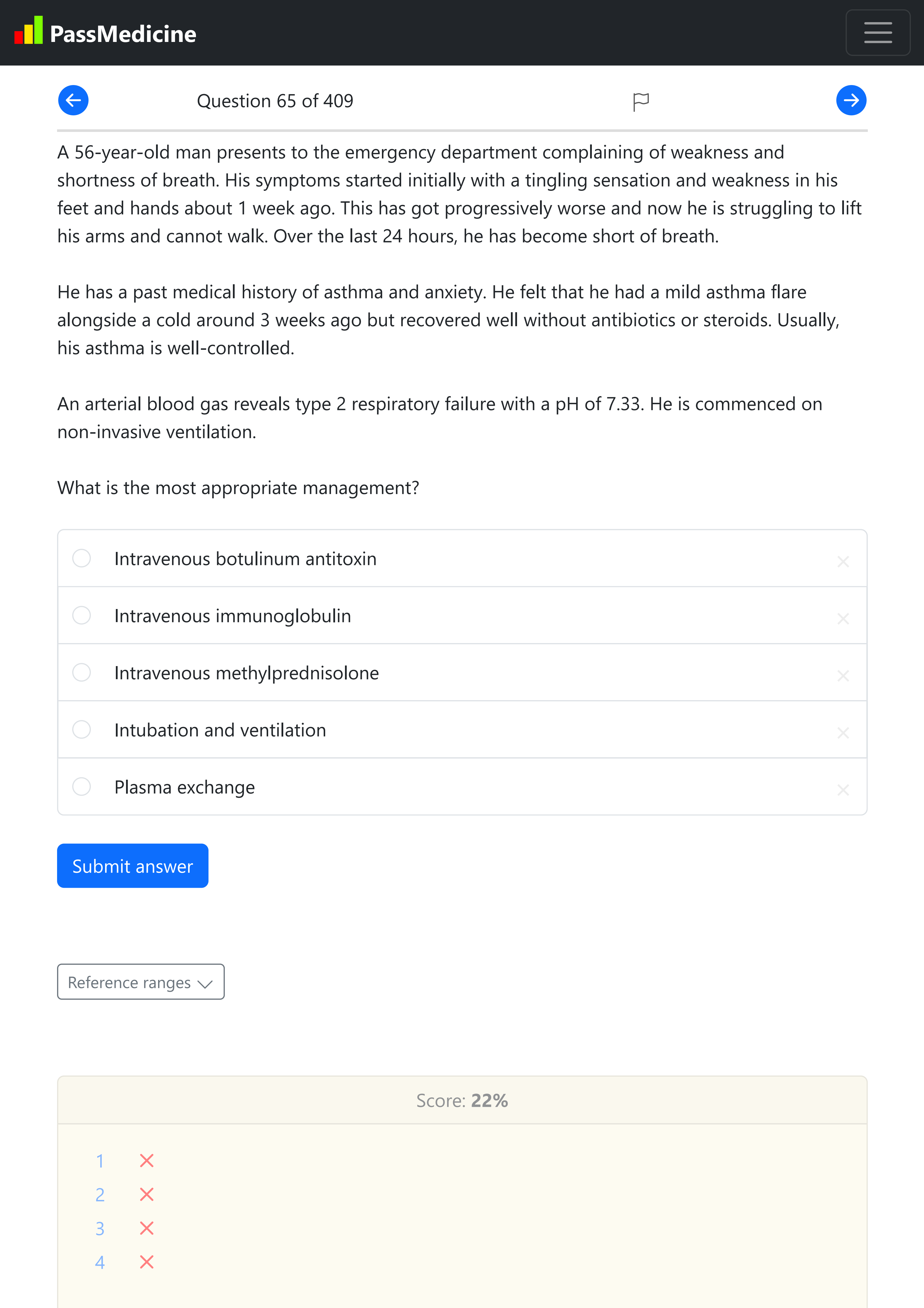

A 56-year-old man presents to the emergency department complaining of weakness and shortness of breath. His symptoms started initially with a tingling sensation and weakness in his feet and hands about 1 week ago. This has got progressively worse and now he is struggling to lift his arms and cannot walk. Over the last 24 hours, he has become short of breath.

He has a past medical history of asthma and anxiety. He felt that he had a mild asthma flare alongside a cold around 3 weeks ago but recovered well without antibiotics or steroids. Usually, his asthma is well-controlled.

An arterial blood gas reveals type 2 respiratory failure with a pH of 7.33. He is commenced on non-invasive ventilation.

What is the most appropriate management?

- ○ Intravenous botulinum antitoxin
- ○ Intravenous immunoglobulin
- ○ Intravenous methylprednisolone
- ○ Intubation and ventilation
- ○ Plasma exchange

Submit answer

Reference ranges

Score: **22%**

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An arterial blood gas reveals type 2 respiratory failure with a pH of 7.33. He is commenced on non-invasive ventilation.

What is the most appropriate management?

Intravenous botulinum antitoxin	3%
🟩 Intravenous immunoglobulin	69%
Intravenous methylprednisolone	4%
Intubation and ventilation	14%
🟥 Plasma exchange	10%

IVIG is generally used first-line for Guillain-Barre syndrome

Important for me Less important

The ascending neurological impairment coupled with a recent upper respiratory tract infection is highly suggestive of Guillain-Barre syndrome. In this case, this has progressed to the extent of affecting the respiratory muscles. **Intravenous immunoglobulin** or plasma exchange can be used in treatment. Immunoglobulin is as effective as plasma exchange, is better tolerated, and is easier to administer. Therefore, immunoglobulin is generally used first-line for Guillain-Barre syndrome where patients require immunomodulatory therapy.

Steroids and immunosuppressants have not been shown to be beneficial in Guillain-Barre syndrome. Therefore, **intravenous methylprednisolone** is incorrect.

Immunoglobulin is better tolerated and easier to administer than **plasma exchange**. Therefore plasma exchange would not be the first-line management choice.

Intravenous botulinum antitoxin is the treatment for botulism. This typically presents with a descending weakness, starting in the cranial nerves. Given this patient has ascending weakness this diagnosis is less likely and so the treatment is not correct.

For patients with severe respiratory failure, **intubation and ventilation** may be required. Given that this patient is only mildly acidotic it can be inferred that type 2 respiratory failure is not too severe, especially given it is acute so there will not be significant metabolic compensation. Intubation and ventilation carry risks and are not warranted at present. If there was poor response to non-invasive ventilation then this would need to be re-considered.

Discuss (1) Improve

Next question

Guillain-Barre syndrome: management

Guillain-Barre syndrome describes an immune-mediated demyelination of the peripheral nervous system often triggered by an infection (classically *Campylobacter jejuni*).

Management
- IV immunoglobulins (IVIG) or plasma exchange can be used
  - IVIG is as effective as plasma exchange better tolerated and easier to administer
  - therefore IVIG is generally used first-line for Guillain-Barre syndrome where patients require immunomodulatory therapy
  - no benefit in combining both treatments
- steroids and immunosuppressants have not been shown to be beneficial
- FVC regularly to monitor respiratory function

Prognosis
- severe motor problems persist in about 15%
- around 5% die

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

NERVE CONDUCTION STUDIES
PERIPHERAL NERVES
GUILLAIN-BARRÉ SYNDROME
WEAKNESS
NEUROPATHY
ANTIBODIES
B-CELLS
CAMPYLOBACTER

Understanding Guillain-Barre Syndrome

Zero To Finals - YouTube 👍 3 👎 1

Guillain-Barre syndrome

Osmosis - YouTube 👍 1 👎 1

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Question 66 of 409

A 54-year-old man presents to the emergency department with reduced mobility. He describes an absence of feeling in his lower limbs bilaterally that makes him feel very unsteady on his feet. His past medical history includes type 2 diabetes, hypercholesterolaemia and hypertension, for which he takes metformin, sitagliptin, atorvastatin and ramipril.

On examination, he has 5/5 power in his upper limbs. There is increased tone and rigidity in his lower limbs, although he is able to weight bear. His proprioception is intact but there is an absence of pain and temperature sensation.

What is the most likely diagnosis?

- ○ Anterior spinal artery occlusion
- ○ Brown-Sequard syndrome
- ○ Central cord syndrome
- ○ Primary lateral sclerosis
- ○ Subacute combined degeneration of the spinal cord

Submit answer

Reference ranges

Score: **22%**

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A 54-year-old man presents to the emergency department with reduced mobility. He describes an absence of feeling in his lower limbs bilaterally that makes him feel very unsteady on his feet. His past medical history includes type 2 diabetes, hypercholesterolaemia and hypertension, for which he takes metformin, sitagliptin, atorvastatin and ramipril.

On examination, he has 5/5 power in his upper limbs. There is increased tone and rigidity in his lower limbs, although he is able to weight bear. His proprioception is intact but there is an absence of pain and temperature sensation.

What is the most likely diagnosis?

Anterior spinal artery occlusion	62%
Brown-Sequard syndrome	8%
Central cord syndrome	11%
Primary lateral sclerosis	5%
Subacute combined degeneration of the spinal cord	14%

Bilateral spastic paresis and loss of pain and temperature sensation - anterior spinal artery occlusion

Important for me Less important

This patient has anterior spinal artery occlusion, affecting the lateral corticospinal and spinothalamic tracts. Symptoms of anterior spinal artery occlusion include bilateral spastic paresis and loss of pain and temperature sensation, as described in this scenario. This patient has significant risk factors for arterial occlusion including diabetes, hypertension and hypercholesterolaemia.

Brown-Sequard syndrome refers to hemisection of the spinal cord, usually secondary to traumatic injury. Symptoms include ipsilateral spastic paresis below the level of the lesion along with ipsilateral loss of proprioception and vibration sensation and contralateral loss of pain and temperature sensation. The absence of these symptoms and history of trauma make Brown-Sequard syndrome less likely.

Central cord syndrome is a very common type of incomplete spinal cord injury, usually secondary to trauma (e.g. following a hyperextension injury in a patient with cervical spondylosis). Rather than a complete loss of movement or sensation, there tends to be a partial pattern of motor and sensory loss with greater motor impairment seen in the upper limbs and variable sensory loss below the level of the injury.

Primary lateral sclerosis, or motor neuron disease, is a neurodegenerative condition affecting the upper and lower motor neurons, causing a combination of upper motor neuron signs (increased tone) and lower motor neuron signs (muscle fasciculations and weakness). The combination of symptoms in primary lateral sclerosis is purely motor, unlike this patient's presentation that also has a sensory deficit, making primary lateral sclerosis unlikely.

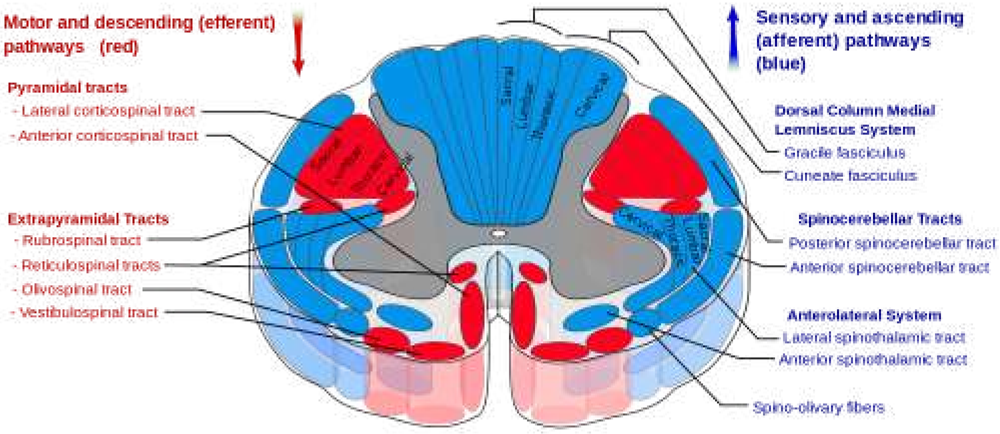
Subacute combined degeneration of the spinal cord is commonly related to vitamin B12 deficiency, causing degeneration of the lateral corticospinal tracts and the dorsal columns. Symptoms include bilateral spastic paresis similarly to this patient. However, sensory symptoms in subacute combined degeneration of the spinal cord are secondary to dorsal column loss and include the absence of proprioception and vibration sensation rather than pain and temperature sensation.

Discuss (4) | Improve

Next question >

Spinal cord lesions

The diagram belows shows cross-section view of the spinal cord:



Motor and descending (efferent) pathways (red)
Pyramidal tracts
- Lateral corticospinal tract
- Anterior corticospinal tract
Extrapyramidal Tracts
- Rubrospinal tract
- Reticulospinal tracts
- Olivospinal tract
- Vestibulospinal tract
Sensory and ascending (afferent) pathways (blue)
Dorsal Column Medial Lemniscus System
Gracile fasciculus
Cuneate fasciculus
Spinocerebellar Tracts
Posterior spinocerebellar tract
Anterior spinocerebellar tract
Anterolateral System
Lateral spinothalamic tract
Anterior spinothalamic tract
Spino-olivary fibers
Sacral
Lumbar
Thoracic
Cervical

Image sourced from Wikipedia

Motor lesions

Amyotrophic lateral sclerosis (motor neuron disease)

- affects both upper (corticospinal tracts) and lower motor neurons
- results in a combination of upper and lower motor neuron signs

Poliomyelitis

- affects anterior horns resulting in lower motor neuron signs

Combined motor and sensory lesions

Disorder	**Tracts affected**	**Clinical notes**
Brown-Sequard syndrome (spinal cord hemisection)	1. Lateral corticospinal tract 2. Dorsal columns 3. Lateral spinothalamic tract	1. Ipsilateral spastic paresis below lesion 2. Ipsilateral loss of proprioception and vibration sensation 3. Contralateral loss of pain and temperature sensation
Subacute combined degeneration of the spinal cord (vitamin B12 & E deficiency)	1. Lateral corticospinal tracts 2. Dorsal columns 3. Spinocerebellar tracts	1. Bilateral spastic paresis 2. Bilateral loss of proprioception and vibration sensation 3. Bilateral limb ataxia
Friedrich's ataxia	Same as subacute combined degeneration of the spinal cord (see above)	Same as subacute combined degeneration of the spinal cord (see above) In addition cerebellar ataxia → other features e.g. intention tremor
Anterior spinal artery occlusion	1. Lateral corticospinal tracts 2. Lateral spinothalamic tracts	1. Bilateral spastic paresis 2. Bilateral loss of pain and temperature sensation
Syringomyelia	1. Ventral horns 2. Lateral spinothalamic tract	1. Flacid paresis (typically affecting the intrinsic hand muscles) 2. Loss of pain and temperature sensation
Multiple sclerosis	Asymmetrical, varying spinal tracts involved	Combination of motor, sensory and ataxia symptoms

Sensory lesions

Disorder	**Tracts affected**	**Clinical notes**
Neurosyphilis (tabes dorsalis)	1. Dorsal columns	1. Loss of proprioception and vibration sensation

+ Q 123

Next question >

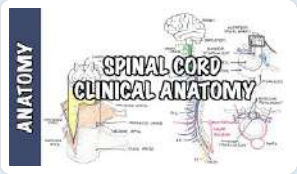
B *I*

Textbooks

High-yield textbook

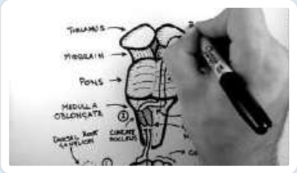
Extended textbook

Media


ANATOMY
SPINAL CORD
CLINICAL ANATOMY

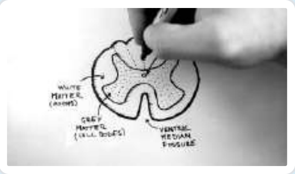
Spinal Cord - Clinical Anatomy and Physiology (dermatomes, blood supply, shingles, lumbar puncture)

Armando Hasudungan - YouTube 👍 7 👎 0


THALAMUS
MIDBRAIN
PONS
MEDULLA OBLONGATA

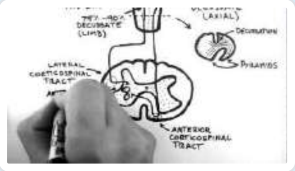
Spinal Pathways 2 - Dorsal Column - Medial Lemniscus Pathway

Handwritten Tutorials - YouTube 👍 4 👎 0


WHITE MATTER (AXONS)
GREY MATTER (CELL BODIES)
VENTRAL MEDIAN FISSURE

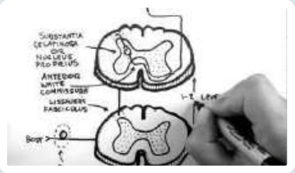
Spinal Pathways 1 - Spinal Cord Anatomy and Organisation

Handwritten Tutorials - YouTube 👍 2 👎 0


DECUSSATE
LATERAL CORTICOSPINAL TRACT
DECUSSATION
PYRAMIDS
ANTERIOR CORTICOSPINAL TRACT

Spinal Pathways 4 - Corticospinal Tract

Handwritten Tutorials - YouTube 👍 2 👎 0


SUBSTANTIA GELATINOSA
ANTERIOR WHITE COMMISSURE
LISSAUER'S FASCICULUS

Spinal Pathways 3 - Spinothalamic Tract

Handwritten Tutorials - YouTube 👍 1 👎 0

Neurology - Spinal Cord Introduction

Armando Hasudungan - YouTube 👍 0 👎 0

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A 42 year old caucasian man presents to his GP because of concerns about weight gain. He has put on 10kg in weight in the past 6 months but has been eating and exercising the same amounts for the past few years. The GP decides to do some further investigations and finds the following abnormalities. He tells you that he was on a medication for schizophrenia but this was changed one year ago due to abnormal movements. In the past year he has also been given medications to help with symptoms of nausea and has recently started medication for gynaecomastia.

Hb	13.8 g/dl
Platelets	154 * 10^9/l
WBC	3.8 * 10^9/l
Neuts	1.5 * 10^9/l
Lymphs	1.0 * 10^9/l
Eosinos	1.2 * 10^9/l

Fasting glucose	11.9 mmol/l
Prolactin	264 mu/l

ECG: sinus rhythm 84/min QTC 462ms

Which drug is most likely to have caused these abnormalities?

○ Haloperidol

○ Clozapine

○ Bromocriptine

○ Domperidone

○ Ondansetron

Submit answer

Reference ranges

Score: **22%**

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Question 67 of 409

A 42 year old caucasian man presents to his GP because of concerns about weight gain. He has put on 10kg in weight in the past 6 months but has been eating and exercising the same amounts for the past few years. The GP decides to do some further investigations and finds the following abnormalities. He tells you that he was on a medication for schizophrenia but this was changed one year ago due to abnormal movements. In the past year he has also been given medications to help with symptoms of nausea and has recently started medication for gynaecomastia.

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WBC	3.8 * 10^9/l
Neuts	1.5 * 10^9/l
Lymphs	1.0 * 10^9/l
Eosinos	1.2 * 10^9/l

Fasting glucose	11.9 mmol/l
Prolactin	264 mu/l

ECG: sinus rhythm 84/min QTC 462ms

Which drug is most likely to have caused these abnormalities?

Haloperidol	15%
Clozapine	54%
Bromocriptine	9%
Domperidone	10%
Ondansetron	12%

One of the most common side effects of clozapine is constipation/intestinal obstruction

Important for me Less important

Clozapine is an atypical antipsychotic that is used in treatment resistant schizophrenia and also if there has been tardive dyskinesia on a previous antipsychotic which is described in this question. Clozapine can cause neutropaenia, eosinophilia and prolongation of the QTc as well as increased weight gain and reduced insulin tolerance which are all seen in this individual

Haloperidol can also cause QTc prolongation. It is less likely to cause weight gain and reduced glucose tolerance than clozapine and it is not associated with neutropaenia or eosinophilia. Haloperidol more commonly causes tardive dyskinesia than Clozapine and it is possible that he was on this before switching due to hyperprolactinaemia and tardive dyskinesia

Bromocriptine is a treatment designed to reduce prolactin levels and does not cause reduced glucose tolerance or neutropaenia.

Domperidone can cause prolonged QTc but would not explain any of the other abnormalities.

Ondansetron can cause prolongation of the QTc but does not cause any of the other abnormalities described here

👍 | 👎 | 💬 Discuss (8) | Improve

Next question >

Clozapine ★

Clozapine, one of the first atypical agents to be developed, carries a significant risk of agranulocytosis and full blood count monitoring is therefore essential during treatment. For this reason, clozapine should only be used in patients resistant to other antipsychotic medication. The BNF states:

> *Clozapine should be introduced if schizophrenia is not controlled despite the sequential use of two or more antipsychotic drugs (one of which should be a second-generation antipsychotic drug), each for at least 6-8 weeks.*

Adverse effects of clozapine

- agranulocytosis (1%), neutropaenia (3%)
- reduced seizure threshold - can induce seizures in up to 3% of patients
- constipation
- myocarditis: a baseline ECG should be taken before starting treatment

- hypersalivation

Dose adjustment of clozapine might be necessary if smoking is started or stopped during treatment.

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

BNF 👍 4 👎 2

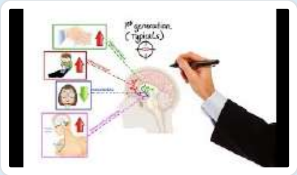
Clozapine

Clinical Knowledge Summaries 👍 3 👎 0

Schizophrenia > Prescribing Information > Adverse effects

Suggest link Report broken link

Media



Antipsychotics

Speed Pharmacology - YouTube 👍 2 👎 0

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A 68-year-old man presents with back pain. He was recently diagnosed with peripheral neuropathy however no cause was identified. He has no other past medical history and takes no regular medicines. On examination he has tenderness of his lumbar spine. You also note hepatosplenomegaly and hyperpigmentation of his skin.

Blood results are as follows:

Hb	105 g/l	Na^+	136 mmol/l
Platelets	110 * 10^9/l	K^+	5.1 mmol/l
WBC	10.2 * 10^9/l	Urea	14.6 mmol/l
Neuts	8.1 * 10^9/l	Creatinine	162 µmol/l
Lymphs	1.1 * 10^9/l	CRP	24 mg/l
Eosin	0.2 * 10^9/l	Calcium (corrected)	2.85 mmol/l

Serum electrophoresis and immunofixation was also performed:

Monoclonal band	45 g/l
Immunofixation	IgG light chain spike

A whole body MRI scan is performed:

Whole body MRI	Osteoblastic lesion present in L4 vertebrae

What is the most likely diagnosis?

○ Osteosarcoma

○ Plasmacytoma

○ POEMS syndrome

○ Waldenstrom macroglobulinemia

○ Monoclonal gammopathy of unexplained significance

Submit answer

Reference ranges ⌵

Score: **22%**

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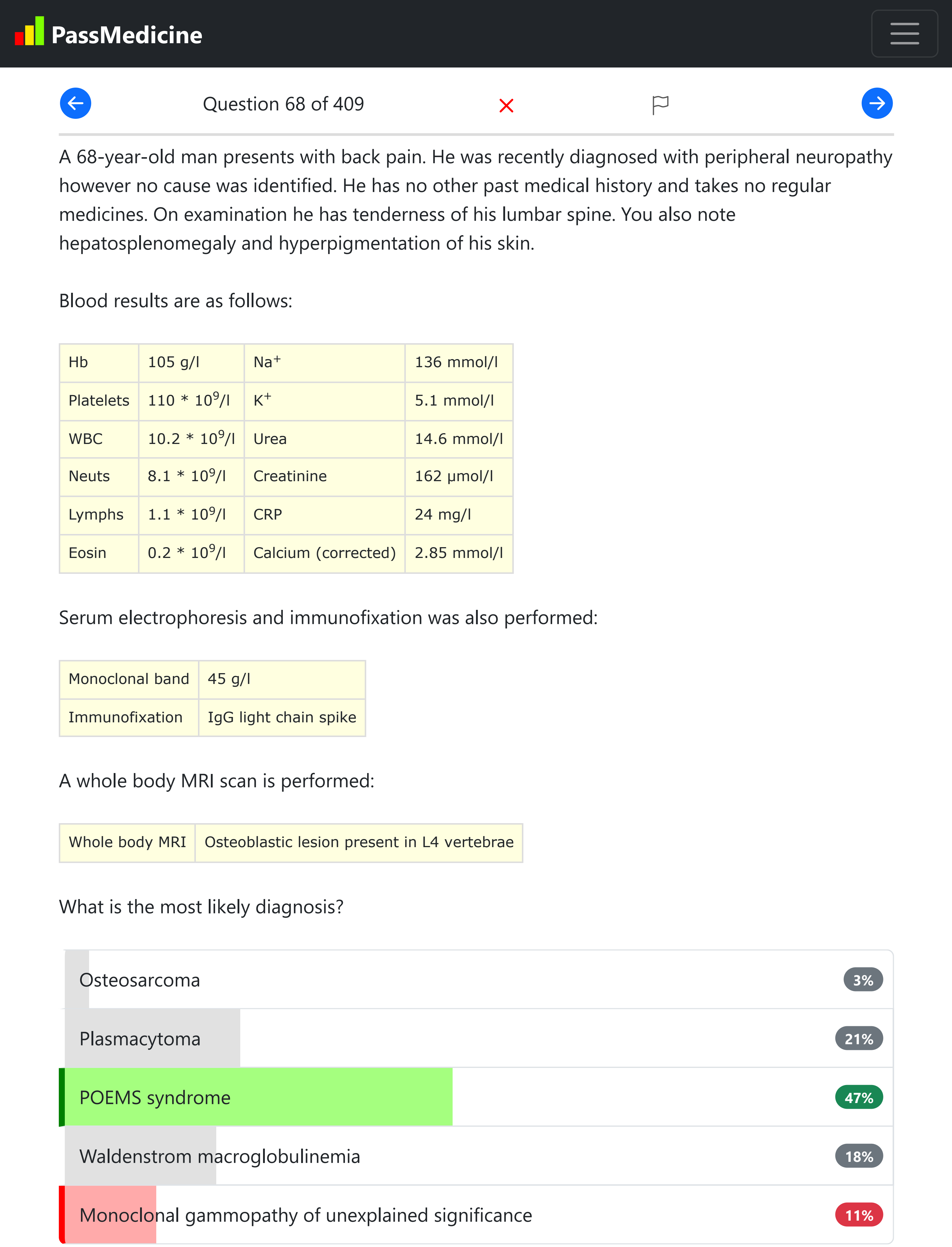

Question 68 of 409

A 68-year-old man presents with back pain. He was recently diagnosed with peripheral neuropathy however no cause was identified. He has no other past medical history and takes no regular medicines. On examination he has tenderness of his lumbar spine. You also note hepatosplenomegaly and hyperpigmentation of his skin.

Blood results are as follows:

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Eosin	0.2 * 10^9/l	Calcium (corrected)	2.85 mmol/l

Serum electrophoresis and immunofixation was also performed:

Monoclonal band	45 g/l
Immunofixation	IgG light chain spike

A whole body MRI scan is performed:

Whole body MRI	Osteoblastic lesion present in L4 vertebrae

What is the most likely diagnosis?

Osteosarcoma	3%
Plasmacytoma	21%
POEMS syndrome	47%
Waldenstrom macroglobulinemia	18%
Monoclonal gammopathy of unexplained significance	11%

POEMS syndrome is a rare paraneoplastic syndrome caused by a clone of abnormal plasma cells. It is associated with polyneuropathy, organomegaly, endocrinopathy, M-protein band from a plasmocytoma, and skin pigmentation

Important for me Less important

The patient has anaemia, renal impairment, and hypercalcaemia. This triad of features should raise suspicion of multiple myeloma, especially in elderly patients who present with back pain. The monoclonal band of plasma cells confirms a plasma cell dyscrasia. The differential for plasma cell dyscrasias include myeloma, monoclonal gammopathy of unexplained significance, plasmacytoma, POEMS syndrome and Waldenstrom macroglobulinemia.

The features of organomegaly, hyperpigmentation of the skin, and a single osteoblastic lesion in the spine should raise concern of POEMS syndrome. POEMS syndrome is a rare paraneoplastic syndrome caused by a clone of abnormal plasma cells. It is associated with polyneuropathy, organomegaly, endocrinopathy, M-protein band from a plasmacytoma, and skin pigmentation. It is therefore the most likely diagnosis in this patient.

Waldenstrom macroglobulinemia is a type of non-Hodgkin lymphoma (NHL) associated with very high levels immunoglobulin M (IgM). IgM is a very large molecule and when present in excess can result in hyperviscosity syndrome. This can also result in organomegaly, peripheral neuropathy and a variety of other features. In this case, the presence of an IgG monoclonal band suggest an alternative diagnosis.

The presence of end-organ damage rules out the diagnosis of monoclonal gammopathy of unexplained significance.

Discuss (10) Improve

Next question ›

POEMS ★

POEMS
- Polyneuropathy
- Organomegaly
- Endocrinopathy
- M-protein band from a plasmocytoma
- Skin pigmentation

AI + Q 123

Next question >

Textbooks

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Score: **20%**

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Question 69 of 409

A 62-year-old man presents with an episode of collapse. He states that the episode of the collapse was preceded by the worst headache of his life. He denies any recent head injuries. On examination, you note nuchal rigidity and photophobia. Observations are within normal limits.

A CT brain is arranged:

CT brain (non-contrast)	Hyperdense material filling the subarachnoid space

What is urgently required?

- ○ CT angiogram
- ○ Digital subtraction angiogram
- ○ Endovascular coiling and neurosurgical clipping
- ○ Lumbar puncture
- ○ MRI angiogram

Submit answer

Reference ranges

Score: **22%**

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A 62-year-old man presents with an episode of collapse. He states that the episode of the collapse was preceded by the worst headache of his life. He denies any recent head injuries. On examination, you note nuchal rigidity and photophobia. Observations are within normal limits.

A CT brain is arranged:

CT brain (non-contrast)	Hyperdense material filling the subarachnoid space

What is urgently required?

CT angiogram	49%
Digital subtraction angiogram	7%
Endovascular coiling and neurosurgical clipping	32%
Lumbar puncture	3%
MRI angiogram	9%

In non-traumatic subarachnoid haemorrhages, a CT angiogram should be performed to identify intracranial aneurysms

Important for me Less important

CT angiogram is correct. A non-contrast CT has confirmed a SAH. The patient does not have a history of trauma and as such an aneurysmal bleed is at the top of the differential diagnosis. Therefore an urgent CT angiogram is required to identify the causal pathology, define the anatomy of the aneurysm, and plan specific treatment.

Digital subtraction angiogram is incorrect. Digital subtraction catheter angiography remains the gold standard for diagnosis and characterisation of vascular abnormalities, and in many centres, even if the causative lesion is identified on MRA or CTA and it is thought to require surgical management, a catheter study is carried out. However, in the first instance, a CT angiogram is required.

Endovascular coiling and neurosurgical clipping is incorrect. Although this may well be required for definitive management of an aneurysmal bleed, in the first instance the lesion needs to be identified and characterised by a CT angiogram.

Lumbar puncture is incorrect. A lumbar puncture is only required if the initial CT brain is negative. In this case, the CT demonstrates clear evidence of a SAH.

MRI angiogram is incorrect. Although MRI is helpful to assess underlying brain parenchyma, MRI angiograms (MRA) are not as good as CT angiograms. Unlike CT angiography, MRA is not able to see and capture images of calcium deposits within the blood vessels. MRA evaluation of small vessels, in particular, may be difficult.

👍 👎 Discuss (3) Improve

Next question >

Subarachnoid haemorrhage ★

A subarachnoid haemorrhage (SAH) is an intracranial haemorrhage that is defined as the presence of blood within the subarachnoid space, i.e. deep to the subarachnoid layer of the meninges.

The most common cause of SAH is head injury and this is called *traumatic SAH* . In the absence of trauma, SAH is termed *spontaneous SAH* . The rest of this note focuses on spontaneous SAH.

Causes of spontaneous SAH include:

- intracranial aneurysm (saccular 'berry' aneurysms)
  - accounts for around 85% of cases
  - conditions associated with berry aneurysms include hypertension,adult polycystic kidney disease, Ehlers-Danlos syndrome and coarctation of the aorta
- arteriovenous malformation
- pituitary apoplexy
- mycotic (infective) aneurysms

Classical presenting features include:

- headache
  - usually of sudden-onset ('thunderclap' or 'hit with a baseball bat')
  - severe ('worst of my life')
  - occipital
  - typically peaking in intensity within 1 to 5 minutes
  - there may be a history of a less-severe 'sentinel' headache in the weeks prior to presentation
- nausea and vomiting
- meningism (photophobia, neck stiffness)
- coma
- seizures

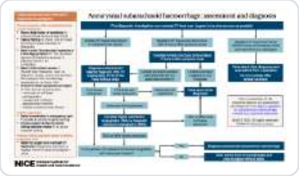
- ECG changes including ST elevation may be seen
  - this may be secondary to either autonomic neural stimulation from the hypothalamus or elevated levels of circulating catecholamines

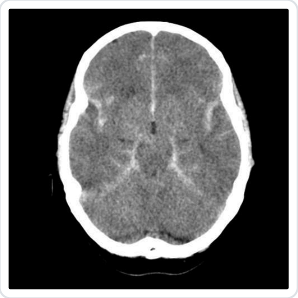
Investigation

- non-contrast CT head is the first-line investigation of choice
  - acute blood (hyperdense/bright on CT) is typically distributed in the basal cisterns, sulci and in severe cases the ventricular system.
- if CT head is done within 6 hours of symptom onset and is normal
  - new guidelines suggest **not** doing a lumbar puncture
  - consider an alternative diagnosis
- if CT head is done more than 6 hours after symptom onset and is normal
  - do a lumber puncture (LP)
  - timing wise the LP should be performed at least 12 hours following the onset of symptoms to allow the development of xanthochromia (the result of red blood cell breakdown).
  - xanthochromia helps to distinguish true SAH from a 'traumatic tap' (blood introduced by the LP procedure).
  - as well as xanthochromia, CSF findings consistent with subarachnoid haemorrhage include a normal or raised opening pressure
- if the CT shows evidence of a SAH
  - referral to neurosurgery to be made as soon as SAH is confirmed

After spontaneous SAH is confirmed, the aim of investigation is to identify a causative pathology that needs urgent treatment:

- CT intracranial angiogram (to identify a vascular lesion e.g. aneurysm or AVM)
- +/- digital subtraction angiogram (catheter angiogram)


Aneurysmal subarachnoid haemorrhage: assessment and diagnosis
NICE



Management of a confirmed aneurysmal subarachnoid haemorrhage

- supportive
  - bed rest
  - analgesia
  - venous thromboembolism prophylaxis
  - discontinuation of antithrombotics (reversal of anticoagulation if present)
- vasospasm is prevented using a course of oral nimodipine
- intracranial aneurysms are at risk of rebleeding and therefore require prompt intervention, preferably within 24 hours

    - most intracranial aneurysms are now treated with a coil by interventional neuroradiologists, but a minority require a craniotomy and clipping by a neurosurgeon

Complications of aneurysmal SAH:

- re-bleeding
    - happens in around 10% of cases and most common in the first 12 hours
    - if rebleeding is suspected (e.g. sudden worsening of neurological symptoms) then a repeat CT should be arranged
    - associated with a high mortality (up to 70%)
- hydrocephalus
    - hydrocephalus is temporarily treated with an external ventricular drain (CSF diverted into a bag at the bedside) or, if required, a long-term ventriculoperitoneal shunt
- vasospasm (also termed delayed cerebral ischaemia), typically 7-14 days after onset
    - ensure euvolaemia (normal blood volume)
    - consider treatment with a vasopressor if symptoms persist
- hyponatraemia (most typically due to syndrome inappropriate anti-diuretic hormone (SIADH))
- seizures

Important predictive factors in SAH:

- conscious level on admission
- age
- amount of blood visible on CT head

AI + Q 123

Next question >

B *I* A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia 👍 15 👎 15

Subarachnoid haemorrhage

Life in the Fast Lane 👍 19 👎 10

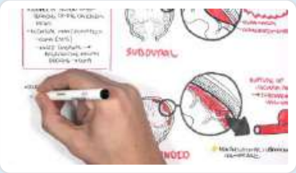
Example ECG changes in subarachnoid haemorrhage

NICE 👍 9 👎 4

2022 Subarachnoid haemorrhage caused by a ruptured aneurysm: diagnosis and management

Suggest link Report broken link

Media



Intracranial Haemorrhage Types

Armando Hasudungan - YouTube 👍 2 👎 0

Arteriovenous malformation

Osmosis - YouTube 👍 0 👎 0

NEUROLOGY
NEUROSURGERY
SUBARACHNOID HAEMORRHAGE

Subarachnoid Haemorrhage / pathophysiology, complications and management

Armando Hasudungan - YouTube 👍 0 👎 0

Report broken media

Score: **20%**

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An 82-year-old woman is brought to see you by her daughter. They are concerned as she has had a tremor for several years that has been worsening and is feeling slightly more forgetful and slowed down in the past 2 months.

On examination, there is a relatively large amplitude tremor on finger-nose testing which is worse on the left side and improves when moved passively. Gait is normal and there is no lower limb involvement. A 10-point abbreviated mental test score is 10/10.

Given the likely diagnosis, what would be the treatment of choice?

- ○ Dopamine agonists ×
- ○ Primidone ×
- ○ Propranolol ×
- ○ Quinine sulphate ×
- ○ Rivastigmine ×

Submit answer

Reference ranges

Score: **22%**

1 ×
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On examination, there is a relatively large amplitude tremor on finger-nose testing which is worse on the left side and improves when moved passively. Gait is normal and there is no lower limb involvement. A 10-point abbreviated mental test score is 10/10.

Given the likely diagnosis, what would be the treatment of choice?

Dopamine agonists	30%
Primidone	8%
Propranolol	50%
Quinine sulphate	1%
Rivastigmine	11%

Propranolol is the treatment of choice for essential tremor

Important for me Less important

The most likely diagnosis here is essential tremor given the worsening of the tremor on intentional movement and improvement when the muscles are relaxed and moved passively by the examiner. In essential tremors, the upper limbs and head/neck are most commonly affected. If there is lower limb involvement alternative pathology should be suspected.

Propranolol is the correct answer. This is the first-line treatment for essential tremors.

Primidone is incorrect. This would usually form second-line management, or treatment if propranolol was contraindicated.

Dopamine agonists is incorrect. These would form part of the management of Parkinson's disease. In this case, we have a tremor that is worsened by movement rather than a resting tremor, gait is normal, and the lower limbs are not affected. Parkinson's disease is therefore less likely.

Rivastigmine is incorrect. This is a cholinesterase inhibitor used in the treatment of Alzheimer's dementia, dementia associated with Parkinson's disease, and Lewy body dementia. In this case, the patient did notice some deterioration in her cognitive function, but the initial mental screening was normal. It may be reasonable to investigate this further with more in-depth mental testing, blood testing, and consideration of a CT head. An MMSE, for example, helps us grade dementia as being mild, or more severe. According to the ICD-10 classification, a decline in memory should have occurred over at least 6 months before a diagnosis of dementia is determined. Given that the symptoms of cognitive decline have been recent - over the past 2 months - it would be acceptable to take a 'watch and wait' approach after risk assessing the symptom severity.

Quinine sulphate is incorrect. This medication is used to treat malaria but also can be used in the management of leg cramps. Lower limb symptoms are not present here, and in fact, the most recent drug and safety guidance is that it should not be prescribed routinely for leg cramps unless very severe or frequent, where organic causes have been ruled out, and where non-pharmacological measures have been ruled out (e.g. exercises, passive stretching) as there is a risk of severe thrombocytopenia and impact on QT interval.

Discuss (2) Improve

Next question

Essential tremor

Essential tremor (previously called benign essential tremor) is an autosomal dominant condition which usually affects both upper limbs

Features
- postural tremor: worse if arms outstretched
- improved by alcohol and rest
- most common cause of titubation (head tremor)

Management
- propranolol is first-line
- primidone is sometimes used

+ Q 123

Next question

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Media

HEAD TREMOR
WORSE WITH CAFFEINE
DIFFERENTIALS?
ESSENTIAL TREMOR
BETTER WITH ALCOHOL
FINE TREMOR
SYMMETRICAL

Essential tremor

Zero To Finals - YouTube 👍 1 👎 0

Report broken media

Score: **20%**

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A 50-year-old woman attends an annual diabetic retinopathy screening appointment. She has a past history of type 1 diabetes mellitus. She reports a gradual decrease in vision in her left eye over the past year.

Examination reveals retinal neovascularisation and formation of fibrous tissue anterior to the retinal disc. The macula appears normal. Her recent HbA1c level is 48 mmol/mol. She is advised that she will need pan-retinal laser photocoagulation (PRP).

In addition to PRP, what is the most appropriate treatment option for this patient?

- ○ Intravenous aflibercept
- ○ Intravitreal ranibizumab
- ○ Optimise glycaemic control
- ○ Screening for cataract
- ○ Vitreoretinal surgery

Submit answer

Reference ranges

Score: **22%**

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In addition to PRP, what is the most appropriate treatment option for this patient?

Intravenous aflibercept	6%
Intravitreal ranibizumab	54%
Optimise glycaemic control	29%
Screening for cataract	6%
Vitreoretinal surgery	5%

Intravitreal VEGF inhibitors may be used in addition to panretinal laser photocoagulation to treat proliferative diabetic retinopathy

Important for me Less important

Intravitreal VEGF inhibitors should be used in combination with PRP for the treatment of patients with proliferative diabetic retinopathy. The scenario describes a patient with proliferative diabetic retinopathy (neovascularisation and fibrous tissue on the retina) for which she is already advised PRP. In addition to PRP, **intravitreal ranibizumab** slows the progression of diabetic retinopathy and improves visual acuity. The best effective treatment of proliferative diabetic retinopathy is PRP plus intravitreal VEGF inhibitors.

Intravitreal, not **intravenous, aflibercept** is used in treating proliferative diabetic retinopathy. Aflibercept is an anti-vascular endothelial growth factor (anti-VEGF) and its intravenous preparation has a role in chemotherapies.

The most important step in managing diabetic retinopathy is to **optimise glycaemic control**. However, the patient in this scenario has current HbA1c within the limits which implies good control already.

Screening for cataracts is an incorrect response because, although important, in a diabetic patient, cataract screening is irrelevant in this scenario.

The fundoscopy in this scenario does not show any vitreous haemorrhage - therefore, **vitreoretinal surgery** like vitrectomy is not a suitable treatment option here. Vitrectomy is for severe proliferative diabetic retinopathy with vitreous haemorrhage.

👍 👎 Discuss Improve

Next question >

Diabetic retinopathy ★

Diabetic retinopathy is the most common cause of blindness in adults aged 35-65 years-old.

Pathophysiology

Hyperglycaemia is thought to cause increased retinal blood flow and abnormal metabolism in the retinal vessel walls. This precipitates damage to endothelial cells and pericytes

Endothelial dysfunction leads to increased vascular permeability which causes the characteristic exudates seen on fundoscopy. Pericyte dysfunction predisposes to the formation of microaneurysms. Neovasculization is thought to be caused by the production of growth factors in response to retinal ischaemia

Classification

Patients are typically classified into those with non-proliferative diabetic retinopathy (NPDR), proliferative retinopathy (PDR) and maculopathy.

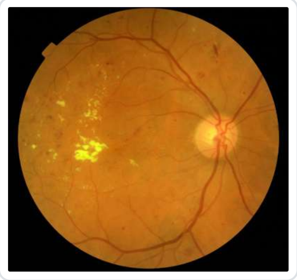
Non-proliferative diabetic retinopathy

Mild NPDR
- 1 or more microaneurysm

Moderate NPDR
- microaneurysms
- blot haemorrhages
- hard exudates

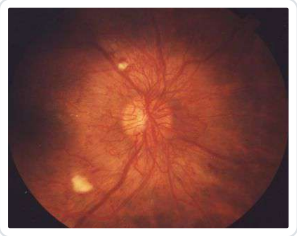
- cotton wool spots ('soft exudates' - represent areas of retinal infarction), venous beading/looping and intraretinal microvascular abnormalities (IRMA) less severe than in severe NPDR

Severe NPDR
- blot haemorrhages and microaneurysms in 4 quadrants
- venous beading in at least 2 quadrants
- IRMA in at least 1 quadrant



Proliferative diabetic retinopathy

Key features
- retinal neovascularisation - may lead to vitrous haemorrhage
- fibrous tissue forming anterior to retinal disc
- more common in Type I DM, 50% blind in 5 years



Maculopathy

Key features
- based on location rather than severity, anything is potentially serious
- hard exudates and other 'background' changes on macula
- check visual acuity
- more common in Type II DM

Management

All patients
- optimise glycaemic control, blood pressure and hyperlipidemia
- regular review by ophthalmology

Maculopathy

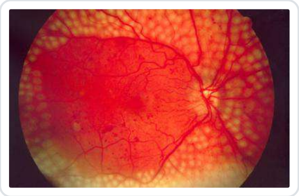
- if there is a change in visual acuity then intravitreal vascular endothelial growth factor (VEGF) inhibitors

Non-proliferative retinopathy

- regular observation
- if severe/very severe consider panretinal laser photocoagulation

Proliferative retinopathy

- panretinal laser photocoagulation
  - following treatment around 50% of patients develop a noticeable reduction in their visual fields due to the scarring of peripheral retinal tissue
  - other complications include a decrease in night vision (rods are predominantly responsible for vision in low light conditions, the majority of rod cells are located in the peripheral retina), a generalised decrease in visual acuity and macular oedema
- intravitreal VEGF inhibitors
  - often now used in combination with panretinal laser photocoagulation
  - examples include ranibizumab
  - strong evidence base suggests they both slow progression of proliferative diabetic retinopathy and improve visual acuity
- if severe or vitreous haemorrhage: vitreoretinal surgery



123

Next question

Textbooks

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Extended textbook

Score: **20%**

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Question 72 of 409

A 40-year-old man presents to the clinic with a three-day history of left-sided headache. He reports tearing in his left eye and pain on eye movement. Examination reveals periorbital swelling and reduced sensation in the distribution of the ophthalmic nerve on the left side. His past medical history is unremarkable, except for a recent visit to an ENT clinic for sinusitis. An MRI of the brain is pending.

What is the most appropriate treatment option for this patient?

- ○ Heparin, Indomethacin, and prednisolone
- ○ Heparin, indomethacin, and metronidazole
- ○ Heparin, prednisolone, and metronidazole
- ○ Heparin, vancomycin, ceftriaxone, and metronidazole
- ○ Prednisolone, ceftriaxone, and vancomycin

Submit answer

Reference ranges

Score: **22%**

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A 40-year-old man presents to the clinic with a three-day history of left-sided headache. He reports tearing in his left eye and pain on eye movement. Examination reveals periorbital swelling and reduced sensation in the distribution of the ophthalmic nerve on the left side. His past medical history is unremarkable, except for a recent visit to an ENT clinic for sinusitis. An MRI of the brain is pending.

What is the most appropriate treatment option for this patient?

Heparin, Indomethacin, and prednisolone	29%
Heparin, indomethacin, and metronidazole	7%
Heparin, prednisolone, and metronidazole	12%
Heparin, vancomycin, ceftriaxone, and metronidazole	32%
Prednisolone, ceftriaxone, and vancomycin	21%

Unilateral headache, periorbital inflammation, ophthalmoplegia → ? cavernous sinus thrombosis

Important for me   Less important

The correct response is **heparin, vancomycin, ceftriaxone, and metronidazole** because the clinical presentation suggests a diagnosis of cavernous sinus thrombosis (CST) in this patient. Features such as unilateral headache, painful ophthalmoplegia, and periorbital swelling are indicative of CST. Additionally, the involvement of the ophthalmic division of the trigeminal nerve corroborates this diagnosis. The patient's history of sinusitis is relevant due to its association with septic CST, which necessitates treatment with intravenous antibiotics and anticoagulation. The appropriate antibiotic regimen includes vancomycin to target methicillin-resistant *Staphylococcus aureus* (MRSA), supplemented by ceftriaxone or cefepime. Metronidazole should be added when a sinus or dental infection is suspected, as indicated in this scenario. Antibiotic therapy should be continued for at least 3 weeks due to limited penetration into the thrombus. Early initiation of anticoagulation with a continuous infusion of heparin has been shown to improve outcomes in patients with septic CST.

Heparin, indometacin, and metronidazole constitutes an incorrect answer because it omits ceftriaxone, which is the first-line antibiotic for managing septic CST. Additionally, vancomycin is essential for MRSA coverage. Indomethacin is not indicated in treating CST; it is utilised for paroxysmal hemicranias characterised by unilateral headache, ptosis, and ophthalmoplegia but lacks efficacy in CST management.

The choice of **heparin, indomethacin, and prednisolone** is also incorrect. Prednisolone does not have a therapeutic role in CST; instead, steroids are primarily used in managing Tolosa-Hunt syndrome—an important differential diagnosis for CST—which involves idiopathic granulomatous inflammation of the cavernous sinus. In contrast to Tolosa-Hunt syndrome which has no preceding infection history typically seen with CST such as sinusitis described here antibiotics combined with heparin remain the cornerstone treatments for septic CST.

Selecting **heparin, prednisolone, and metronidazole** fails to provide an adequate treatment strategy because it lacks an appropriate first-line antibiotic therapy combination necessary for managing CST effectively; moreover vancomycin must be included for MRSA coverage Steroids do not play a part in treating CST

Lastly **prednisolone ceftriaxone and vancomycin** represents an inaccurate response due to the absence of heparin which is integral to the recommended treatment protocol

👍 👎 Discuss Improve

Next question >

Intracranial venous thrombosis ★

Overview
- can cause cerebral infarction, much lesson common than arterial causes
- 50% of patients have isolated sagittal sinus thromboses - the remainder have coexistent lateral sinus thromboses and cavernous sinus thromboses

General features

Common features
- headache (may be sudden onset)
- nausea & vomiting
- reduced consciousness

Investigation
- MRI venography is the gold standard
  - CT venography is an alternative
- non-contrast CT head is normal in around 70% of patients
- D-dimer levels may be elevated

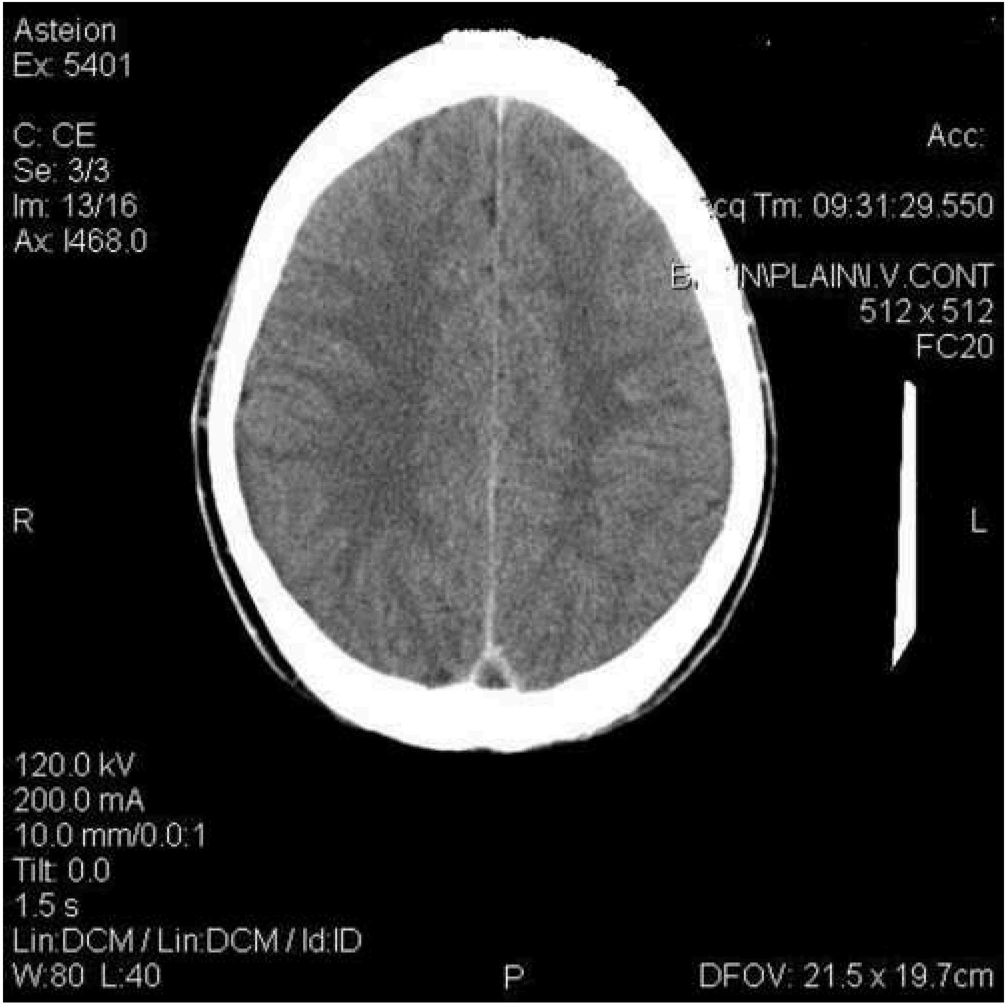
Management
- anticoagulation
  - typically with low molecular weight heparin acutely
  - warfarin is still generally used for longer term anticoagulation

Specific syndromes

Sagittal sinus thrombosis
- may present with seizures and hemiplegia
- parasagittal biparietal or bifrontal haemorrhagic infarctions are sometimes seen
- 'empty delta sign' seen on venography

Cavernous sinus thrombosis
- other causes of cavernous sinus syndrome: local infection (e.g. sinusitis), neoplasia, trauma
- periorbital erythema and oedema
- ophthalmoplegia: 6th nerve damage typically occurs before 3rd & 4th
- trigeminal nerve involvement may lead to hyperaesthesia of upper face and eye pain
- central retinal vein thrombosis

Lateral sinus thrombosis
- 6th and 7th cranial nerve palsies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© Image used on license from Radiopaedia

CT with contrast demonstrating a **superior sagittal sinus thrombosis** showing the typical empty delta sign. Look at the 'bottom' of the scan for the triangular shaped dural sinus. This should normally be white due to it being filled with contrast. The empty delta sign occurs when the thrombus fails to enhance within the dural sinus and is outlined by enhanced collateral channels in the falx. This sign is seen in only about 25%-30% of cases but is highly diagnostic for sagittal sinus thrombosis

＋ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20%**

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Question 73 of 409

A 47-year-old man is brought in by ambulance to the emergency department. He was found agitated and wandering the street by a member of the public. Collateral history from his relative reveals that he has a past medical history of hepatic steatosis, hypertension and asthma. His regular medications include amlodipine and Symbicort. He drinks up to 1 litre of vodka daily and has smoked 20 cigarettes per day for 30 years. There is no history of recreational drug use. He has not been seen by his family for 2-3 days before these events.

His observations are heart rate 111 beats per minute, blood pressure 170/94 mmHg, respiratory rate 23 /min, oxygen saturations 97% on air and temperature 37.2°C. He is tremulous, diaphoretic and agitated. Cardiovascular, respiratory and abdominal examinations are otherwise unremarkable.

Shortly after he is examined, he has a seizure lasting 30 seconds that is self-terminating.

Blood tests:

Hb	164 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	6.0 * 10^9/L	(4.0 - 11.0)
Na$^+$	129 mmol/L	(135 - 145)
K$^+$	4 mmol/L	(3.5 - 5.0)
Urea	8.9 mmol/L	(2.0 - 7.0)
Creatinine	77 µmol/L	(55 - 120)
CRP	3 mg/L	(< 5)
Glucose	4.1 mmol/L	(4 - 11)

A CT head is organized, which is reported as being normal.

What is the most appropriate medication choice to prevent further seizures?

- ○ Oral chlordiazepoxide
- ○ Intravenous glucose
- ○ Intravenous hypertonic saline
- ○ Intravenous levetiracetam

○ Intravenous pabrinex ×

Submit answer

Reference ranges ⌄

Score: **22%**

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A 47-year-old man is brought in by ambulance to the emergency department. He was found agitated and wandering the street by a member of the public. Collateral history from his relative reveals that he has a past medical history of hepatic steatosis, hypertension and asthma. His regular medications include amlodipine and Symbicort. He drinks up to 1 litre of vodka daily and has smoked 20 cigarettes per day for 30 years. There is no history of recreational drug use. He has not been seen by his family for 2-3 days before these events.

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Creatinine	77 µmol/L	(55 - 120)
CRP	3 mg/L	(< 5)
Glucose	4.1 mmol/L	(4 - 11)

A CT head is organized, which is reported as being normal.

What is the most appropriate medication choice to prevent further seizures?

Oral chlordiazepoxide	56%
Intravenous glucose	5%
Intravenous hypertonic saline	10%
Intravenous levetiracetam	6%

Intravenous pabrinex 24%

Chlordiazepoxide, lorazepam or diazepam are used in the treatment of delirium tremens/alcohol withdrawal

Important for me Less important

Chlordiazepoxide is the correct answer. Minor alcohol withdrawal symptoms such as tremor, anxiety and headache start approximately 6-12 hours after alcohol is stopped. This can progress to alcoholic hallucinosis after 12-24 hours and subsequently withdrawal seizures and delirium tremens from 48 hours onwards. The combination of tachypnoea, tachycardia, agitation and seizures suggest delirium tremens and alcohol withdrawal seizures as the diagnosis. The first-line treatment for delirium tremens and to prevent further seizures is chlordiazepoxide. The Clinical Institute Withdrawal Assessment for Alcohol (CIWA) score is used for the management of alcohol withdrawal in hospital. It is a ten item scoring system where higher total scores correspond to a greater severity of alcohol withdrawal. Depending on the score, specific management decisions are suggested, such as the administration of benzodiazepines.

Intravenous glucose is incorrect. The patient's glucose is within the normal range, albeit at the lower end, making this an unlikely cause of seizures.

Intravenous hypertonic saline is incorrect. While his sodium is mildly low at 129, this value is not typically low enough to cause seizures.

Intravenous levetiracetam is incorrect. While this is an effective anti-seizure medication, it is not the most appropriate choice given the underlying diagnosis.

Intravenous pabrinex is incorrect. This will not prevent or terminate seizures but is used to prevent the development of Wernicke's encephalopathy.

👍 👎 Discuss (4) Improve

Next question >

Alcohol withdrawal ★

Mechanism
- chronic alcohol consumption enhances GABA mediated inhibition in the CNS (similar to benzodiazepines) and inhibits NMDA-type glutamate receptors
- alcohol withdrawal is thought to be lead to the opposite (decreased inhibitory GABA and increased NMDA glutamate transmission)

Features

- symptoms start at 6-12 hours: tremor, sweating, tachycardia, anxiety
- peak incidence of seizures at 36 hours
- peak incidence of delirium tremens is at 48-72 hours: coarse tremor, confusion, delusions, auditory and visual hallucinations, fever, tachycardia

Management

- patients with a history of complex withdrawals from alcohol (i.e. delirium tremens, seizures, blackouts) should be admitted to hospital for monitoring until withdrawals stabilised
- first-line: long-acting benzodiazepines e.g. chlordiazepoxide or diazepam. Lorazepam may be preferable in patients with hepatic failure. Typically given as part of a reducing dose protocol
    - lorazepam is often preferred in patients with liver cirrhosis. Lorazepam is metabolised through glucuronidation, which is less affected by liver function. Even in patients with significant liver impairment, glucuronidation tends to remain intact, making lorazepam a safer choice
    - chlordiazepoxide, on the other hand, undergoes hepatic oxidation via the cytochrome P450 system, which is significantly impaired in cirrhosis. This can lead to prolonged drug accumulation and an increased risk of toxicity, including sedation and hepatic encephalopathy.
- carbamazepine also effective in treatment of alcohol withdrawal
- phenytoin is said not to be as effective in the treatment of alcohol withdrawal seizures

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20%**

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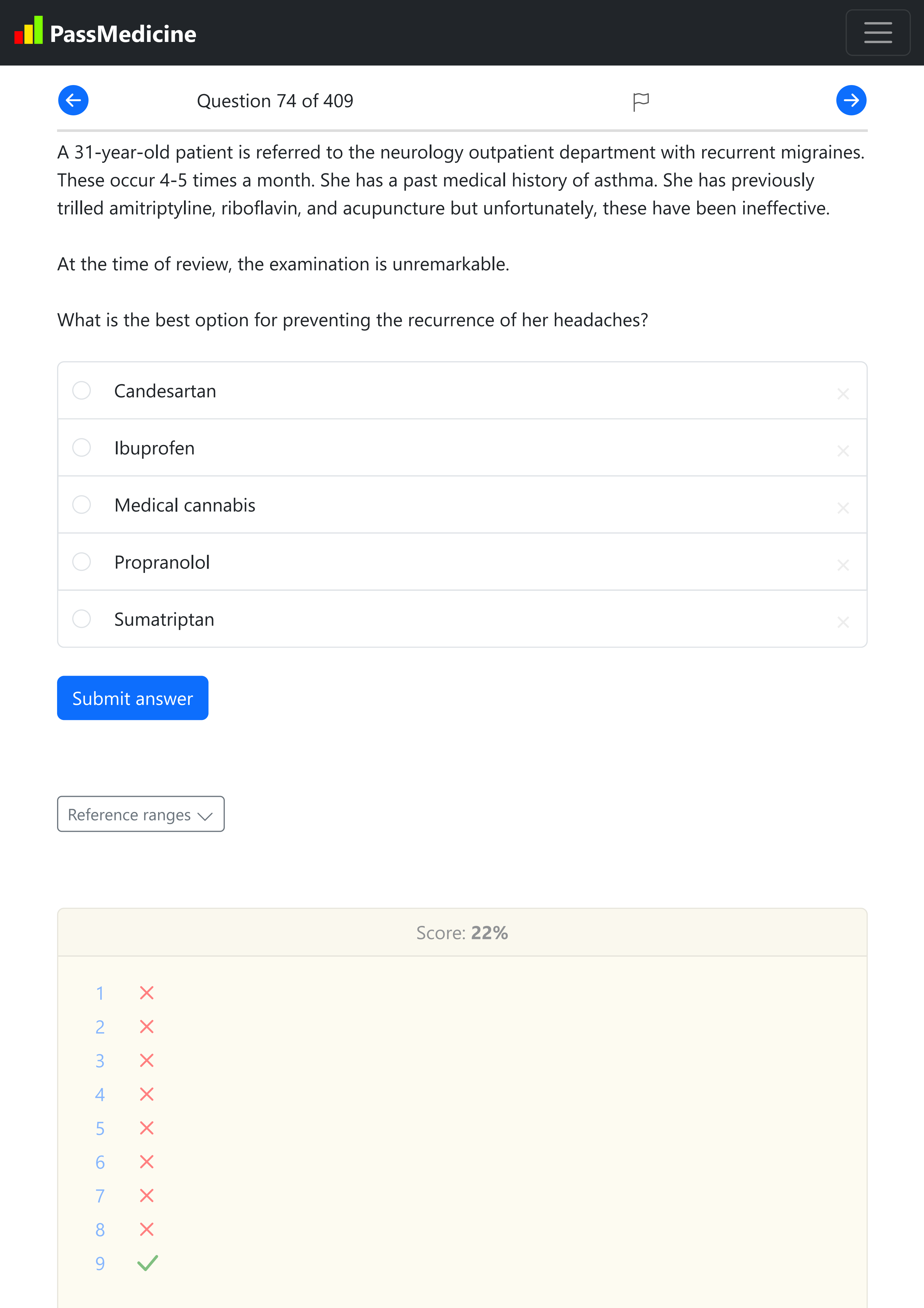

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Question 74 of 409

A 31-year-old patient is referred to the neurology outpatient department with recurrent migraines. These occur 4-5 times a month. She has a past medical history of asthma. She has previously trilled amitriptyline, riboflavin, and acupuncture but unfortunately, these have been ineffective.

At the time of review, the examination is unremarkable.

What is the best option for preventing the recurrence of her headaches?

- ○ Candesartan
- ○ Ibuprofen
- ○ Medical cannabis
- ○ Propranolol
- ○ Sumatriptan

Score: **22%**

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A 31-year-old patient is referred to the neurology outpatient department with recurrent migraines. These occur 4-5 times a month. She has a past medical history of asthma. She has previously trilled amitriptyline, riboflavin, and acupuncture but unfortunately, these have been ineffective.

At the time of review, the examination is unremarkable.

What is the best option for preventing the recurrence of her headaches?

Candesartan	53%
Ibuprofen	1%
Medical cannabis	4%
Propranolol	30%
Sumatriptan	12%

Candesartan may be considered for migraine prophylaxis if other first-line options either don't work or are contradicted

Important for me Less important

Candesartan is correct. This patient presents with recurrent migraines and requires prophylactic treatment in an effort to decrease their frequency. Candesartan is an option for this indication if other options are unsuitable, ineffective, or not tolerated.

Ibuprofen is incorrect. This is a treatment for acute migraine rather than prevention. Furthermore, her history of asthma makes this option unsuitable.

Medical cannabis is incorrect. There is emerging evidence that cannabidiol (CBD) oil may relieve the symptoms of migraine. However, it is not currently approved for migraine prophylaxis in the UK and so this is incorrect.

Propranolol is incorrect. This is a good option for the prevention of migraine but it is contraindicated in patients with asthma.

Sumatriptan is incorrect. This is a treatment for the acute relief of migraine. It is not indicated in the prophylaxis of migraine.

👍 👎 Discuss (3) Improve

Next question >

Migraine: management ★

It should be noted that as a general rule 5-HT receptor agonists are used in the acute treatment of migraine whilst 5-HT receptor antagonists are used in prophylaxis. NICE produced guidelines in 2012 on the management of headache, including migraines.

Acute treatment

- first-line: offer combination therapy with
  - an oral triptan and an NSAID, or
  - an oral triptan and paracetamol
- for young people aged 12-17 years consider a nasal triptan in preference to an oral triptan
- if the above measures are not effective or not tolerated offer a non-oral preparation of metoclopramide or prochlorperazine and consider adding a non-oral NSAID or triptan
  - caution should be exercised when prescribing metoclopramide to young patients as acute dystonic reactions may develop

Prophylaxis

- prophylaxis should generally be given if '*Migraine attacks are having a significant impact on quality of life and daily function, for example they occur frequently (more than once a week on average) or are prolonged and severe despite optimal acute treatment*'
- options:
  - propranolol
  - topiramate: should be avoided in women of childbearing age as it may be teratogenic and it can reduce the effectiveness of hormonal contraceptives
  - amitriptyline
- if these measures fail NICE recommends 'a course of up to 10 sessions of acupuncture over 5-8 weeks'
- NICE recommend: '*Advise people with migraine that riboflavin (400 mg once a day) may be effective in reducing migraine frequency and intensity for some people*'
- for women with predictable menstrual migraine treatment NICE recommend either frovatriptan (2.5 mg twice a day) or zolmitriptan (2.5 mg twice or three times a day) as a type of 'mini-prophylaxis'
- treatment options that may be considered by specialists, but fall outside the NICE guidelines:
  - candesartan: recommended by the British Association for the Study of Headache guidelines

    - monoclonal antibodies directed against the calcitonin gene-related peptide (CGRP) receptor: examples include erenumab
- pizotifen is no longer recommended. Adverse effects such as weight gain & drowsiness are common

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Association for the Study of Headache Headache guidelines	14 21
Clinical Knowledge Summaries Migraine guidelines	14 7
NICE 2012 Headache guidelines	16 4

Suggest link

Report broken link

Media

Management of Migraine
Podmedics

Migraine

Podmedics - YouTube 👍 1 👎 1

Report broken media

Score: **20%**

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Question 75 of 409

A 20-year-old man presents to the neurology clinic following his second seizure. His partner describes episodes during which the patient is unresponsive and appears to be 'staring into space'. The patient has no recollection of these events but does recall that after his reported absences, he often feels nauseous and disorientated for several hours.

Blood tests to identify electrolyte abnormalities are all within normal limits. An MRI head is reportedly within normal limits. An EEG (electroencephalogram) shows interictal epileptiform discharges.

Given the likely diagnosis, which of the following drug therapies would be the most appropriate first-line agent?

- ○ Sodium valproate
- ○ Levetiracetam
- ○ Carbamazepine
- ○ Phenytoin
- ○ Lamotrigine

Submit answer

Reference ranges

Score: **22%**

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A 20-year-old man presents to the neurology clinic following his second seizure. His partner describes episodes during which the patient is unresponsive and appears to be 'staring into space'. The patient has no recollection of these events but does recall that after his reported absences, he often feels nauseous and disorientated for several hours.

Blood tests to identify electrolyte abnormalities are all within normal limits. An MRI head is reportedly within normal limits. An EEG (electroencephalogram) shows interictal epileptiform discharges.

Given the likely diagnosis, which of the following drug therapies would be the most appropriate first-line agent?

Sodium valproate	59%
Levetiracetam	15%
Carbamazepine	11%
Phenytoin	1%
Lamotrigine	14%

Absence seizures: sodium valproate and ethosuximide are first-line

Important for me Less important

This patient has a history and EEG consistent with a diagnosis of absence seizures.

Sodium valproate or ethosuximide are considered first-line in the treatment of absence seizures.

Discuss (6) Improve

Next question

Absence seizures

Absence seizures (petit mal) are a form of generalised epilepsy that is mostly seen in children. The typical age of onset of 3-10 years old and girls are affected twice as commonly as boys

Features

- absences last a few seconds and are associated with a quick recovery
- seizures may be provoked by hyperventilation or stress
- the child is usually unaware of the seizure
- they may occur many times a day
- EEG: bilateral, symmetrical 3Hz spike and wave pattern

Management

- sodium valproate and ethosuximide are first-line treatment
- good prognosis - 90-95% become seizure free in adolescence

＋ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

NICE

2012 Epilepsy guidelines

👍 9 👎 5

Suggest link

Report broken link

Score: **20%**

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A 55-year-old woman attends neurology clinic for follow-up. At her first appointment 6 months previously, a clinical diagnosis of trigeminal neuralgia had been made and treatment with carbamazepine initiated. During clinic review the patient complained of ongoing symptoms with only limited benefit from drug treatment.

On her previous assessment, the patient had reported pain episodes associated with the cheek and jaw of the right side of her face. Subsequently, in addition to similar ongoing episodes, the patient had suffered from two episodes of pain affecting the forehead and peri-orbital region on the left side of her face. On one occasion, she had suffered from both right and left sided pain at the same time. She reported that the carbamazepine treatment had initially reduced the intensity of the pain episodes by perhaps 50 %, but that the severity of recent attacks had again worsened to their original level. When her drug history was checked, it was apparent that the carbamazepine dose had been titrated up appropriately by the patients GP.

The patient also stated that following her previous clinic appointment she had been discussing the situation with her sister, who had reminded the patient that their mother had suffered several episodes of visual loss during her life but had not undergone any medical investigation.

Cranial nerve examination demonstrated normal eye movements and pupillary responses. There was no evidence of facial nerve weakness, however a subjective numbness of sensation was noted in the left cheek region (this had not been noted on previous examination in clinic). There were no lesions around the head and neck or inside the mouth. Peripheral neurological examination was remarkable for borderline dysdiadochokinesia in the right upper limb and a positive Babinski response in the right lower limb.

What is the most appropriate investigation for this patient?

○ Visual evoked potentials

○ Three-plane fine-slice MRI brain with contrast

○ CSF oligoclonal bands

○ CT brain with contrast

○ Standard protocol MRI brain with contrast

Submit answer

Reference ranges

Score: **20%**

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What is the most appropriate investigation for this patient?

Visual evoked potentials	7%
Three-plane fine-slice MRI brain with contrast	37%
CSF oligoclonal bands	9%
CT brain with contrast	3%
[✓ Standard protocol MRI brain with contrast]	43%

The patients previous clinical diagnosis of trigeminal neuralgia clearly needs to be reviewed in light of several red-flag features. In particular, the limited response to carbamazepine, the occurrence of simultaneous bilateral pain and the history of pain associated with the ophthalmic division are inconsistent with a typical presentation of trigeminal neuralgia. Moreover, the patient has developed some soft neurological signs that are suggestive of CNS lesions in various areas of the brain.

Multiple sclerosis is the most likely alternative diagnosis, although other causes of trigeminal pain such as space occupying lesions, malignant invasion of the trigeminal nerve or thalamic infarction are also possibilities. The best investigation to assess for all these differentials is standard protocol MRI brain with contrast. Demyelination or trigeminal nerve infiltration would not be reliably noted on CT brain with contrast making this less likely to progress the diagnosis. Visual evoked potentials and CSF oligoclonal bands are other investigations that may support a diagnosis of multiple sclerosis but would not confirm or refute this diagnosis in isolation.

Typical cases of trigeminal neuralgia are considered to result from contact between blood vessels and the trigeminal nerve. Such vascular contacts can be observed on specialised MRI protocols requiring fine-slicing in three-planes. However, given the additional expense and the fact that such contacts can be a normal variant, this investigation is not normally performed. A standard protocol MRI brain is sufficient to assess for the important differentials of trigeminal neuralgia discussed above.

Zakrzewska J, Linskey M. Trigeminal neuralgia. BMJ 2014;348:g474.

👍 👎 💬 Discuss (4) Improve

Next question >

Trigeminal neuralgia ★

Trigeminal neuralgia is a pain syndrome characterised by severe unilateral pain. The vast majority of cases are idiopathic but compression of the trigeminal roots by tumours or vascular problems may occur.

The International Headache Society defines trigeminal neuralgia as:
- a unilateral disorder characterised by brief electric shock-like pains, abrupt in onset and termination, limited to one or more divisions of the trigeminal nerve
- the pain is commonly evoked by light touch, including washing, shaving, smoking, talking, and brushing the teeth (trigger factors), and frequently occurs spontaneously
- small areas in the nasolabial fold or chin may be particularly susceptible to the precipitation of pain (trigger areas)
- the pains usually remit for variable periods

The following as red flag symptoms and signs suggesting a serious underlying cause:
- Sensory changes
- Deafness or other ear problems

- History of skin or oral lesions that could spread perineurally
- Pain only in the ophthalmic division of the trigeminal nerve (eye socket, forehead, and nose), or bilaterally
- Optic neuritis
- A family history of multiple sclerosis
- Age of onset before 40 years

Management

- carbamazepine is first-line
- failure to respond to treatment or atypical features (e.g. < 50 years old) should prompt referral to neurology

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries Trigeminal Neuralgia guidelines	18 3

Suggest link

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Score: **20%**

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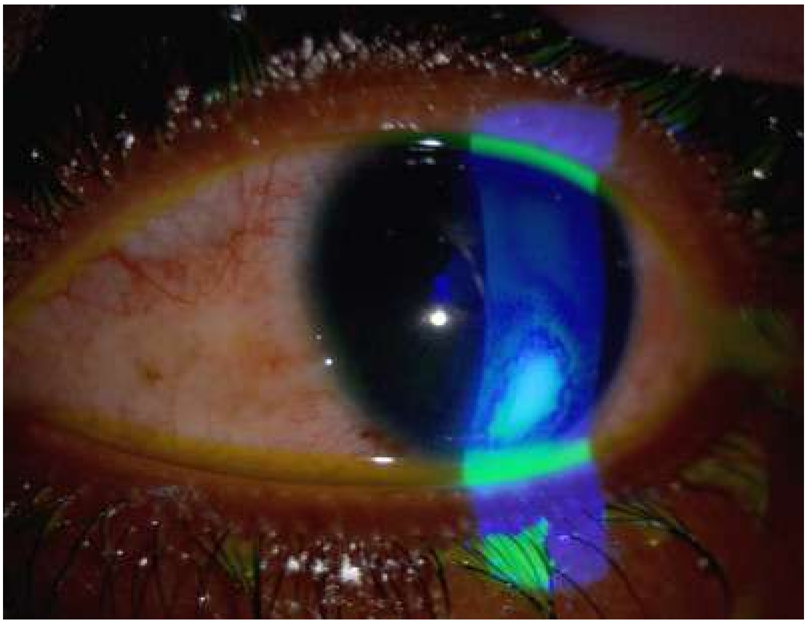
A 23-year-old man presents to the eye casualty department with 3 days of pain in the right eye, accompanied by photophobia and watering. He has also noticed frequent bouts of yellow discharge. The patient has no significant past medical history but is a contact lens wearer. He admits to having fallen asleep and leaving his contact lenses in, several nights ago.

The eye is urgently examined using a slit lamp and appears as shown below:



Given the likely diagnosis, what is the most appropriate management?

- ○ Intramuscular ceftriaxone
- ○ Oral moxifloxacin
- ○ Topical dexamethasone
- ○ Topical ganciclovir
- ○ Topical gentamicin

Submit answer

Reference ranges

A 23-year-old man presents to the eye casualty department with 3 days of pain in the right eye, accompanied by photophobia and watering. He has also noticed frequent bouts of yellow discharge. The patient has no significant past medical history but is a contact lens wearer. He admits to having fallen asleep and leaving his contact lenses in, several nights ago.

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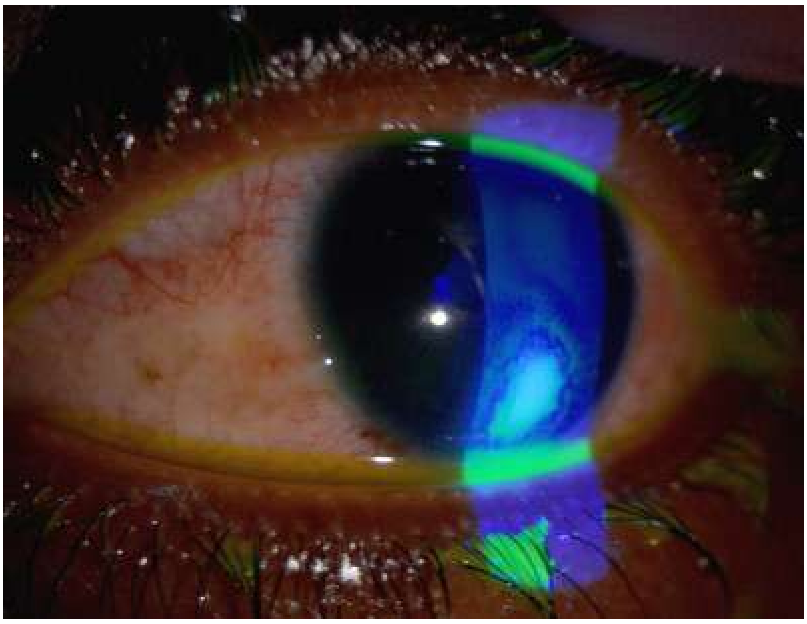


Given the likely diagnosis, what is the most appropriate management?

Intramuscular ceftriaxone	3%
Oral moxifloxacin	8%
Topical dexamethasone	13%
Topical ganciclovir	39%
Topical gentamicin	37%

This patient's presentation, coupled with his history of contact lens overuse, is suggestive of keratitis. This is confirmed by the examination - fluorescein staining has been used and, under slit lamp investigation, a corneal ulcer is clearly demonstrated on the right eye. Treatment depends on the underlying cause. With the history of discharge, a bacterial cause can be presumed and so treatment involves the use of antibiotics - **topical gentamicin** is an appropriate choice and is often combined with another antibiotic such as topical vancomycin.

Intramuscular ceftriaxone is generally not recommended except for suspected gonococcal eye infection. In the absence of any systemic symptoms being mentioned here, this is unlikely.

Oral moxifloxacin is a suitable choice when oral antibiotics are indicated for keratitis. This is not usually necessary except for in cases of corneal perforation, which is not seen here.

Topical dexamethasone is used in a variety of eye diseases to reduce inflammation. However, it is not usually used in bacterial keratitis or corneal ulcers, as the evidence demonstrates no significant benefit.

Topical ganciclovir is used in the management of dendritic or herpetic keratitis, which is viral in nature. This is differentiated upon careful slit lamp examination - fluorescein staining instead would show a distinct branching pattern.

👍 👎 Discuss (2) Improve

Next question >

Corneal ulcer ★

A corneal ulcer describes a defect in the cornea, typically secondary to an infective cause. The term corneal abrasion is typically used for corneal defects secondary to physical trauma.

Risk factors

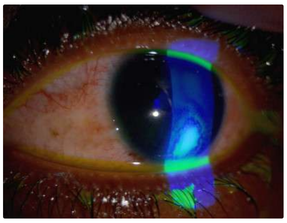
- contact lens use
- vitamin A deficiency: a particular problem in the developing world

Pathophysiology

- bacterial keratitis
- fungal keratitis
- viral keratitis: herpes simplex, herpes zoster - may lead to a dendritic ulcer
- *Acanthamoeba* keratitis: associated with contact lens use

Features

- eye pain
- photophobia
- watering of the eye
- focal fluorescein staining of the cornea



123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Score: **20%**

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A 27 year-old woman attends the neurology clinic complaining of headache and visual disturbance. She has recently immigrated from Ghana. Her symptoms began approximately one month ago, shortly after the birth of her first child. She experiences dull frontal headache which is worst in the mornings and on coughing or straining, as well as transient episodes of 'darkening' of her vision. She saw a doctor in Ghana and was diagnosed with idiopathic intracranial hypertension. She is taking acetazolamide 250mg BD and no other medication.

On examination the visual fields are markedly constricted and the right blind spot is enlarged. Fundoscopy shows bilateral papilloedema worse on the right. The remainder of the neurological examination is unremarkable. BMI is 18 kg/m^2.

Plain computed tomography of the brain is normal.

Incidentally as she is leaving the clinic she mentions that she has also been experiencing pins and needles in the hands and feet.

What is the best course of action?

- ○ Increase dose of acetazolamide ×
- ○ Request nerve conduction studies ×
- ○ Organise for therapeutic lumbar puncture ×
- ○ Refer to neurosurgeons for consideration of ventriculo-peritoneal shunting ×
- ○ Request CT venography ×

Submit answer

Reference ranges

Score: **20%**

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Plain computed tomography of the brain is normal.

Incidentally as she is leaving the clinic she mentions that she has also been experiencing pins and needles in the hands and feet.

What is the best course of action?

Increase dose of acetazolamide	2%
Request nerve conduction studies	6%
Organise for therapeutic lumbar puncture	25%
Refer to neurosurgeons for consideration of ventriculo-peritoneal shunting	23%
Request CT venography	44%

The history of headache suggestive of raised intracranial pressure associated with transient visual obscurations, as well as the examination findings, are all compatible with idiopathic intracranial hypertension (IIH). However, the onset of symptoms in the puerperium, in a slim patient with no other risk factors for IIH, raises the suspicion that this may actually be a cerebral venous sinus thrombosis. All patients with IIH should have imaging of the venous system with CT or MR to exclude a thrombus which can be more appropriately treated with anticoagulation.

Therapeutic lumbar puncture can ease the headache of IIH but is a short-term measure. Ventriculo-peritoneal shunting can be used where medical management has failed.

There is no role for nerve conduction studies in this patient. Paraesthesia are a common side effect of acetazolamide, and this patient is unlikely to tolerate an increase in the dose.

Intracranial venous thrombosis ★

Overview
- can cause cerebral infarction, much lesson common than arterial causes
- 50% of patients have isolated sagittal sinus thromboses - the remainder have coexistent lateral sinus thromboses and cavernous sinus thromboses

General features

Common features
- headache (may be sudden onset)
- nausea & vomiting
- reduced consciousness

Investigation
- MRI venography is the gold standard
  - CT venography is an alternative
- non-contrast CT head is normal in around 70% of patients
- D-dimer levels may be elevated

Management
- anticoagulation
  - typically with low molecular weight heparin acutely
  - warfarin is still generally used for longer term anticoagulation

Specific syndromes

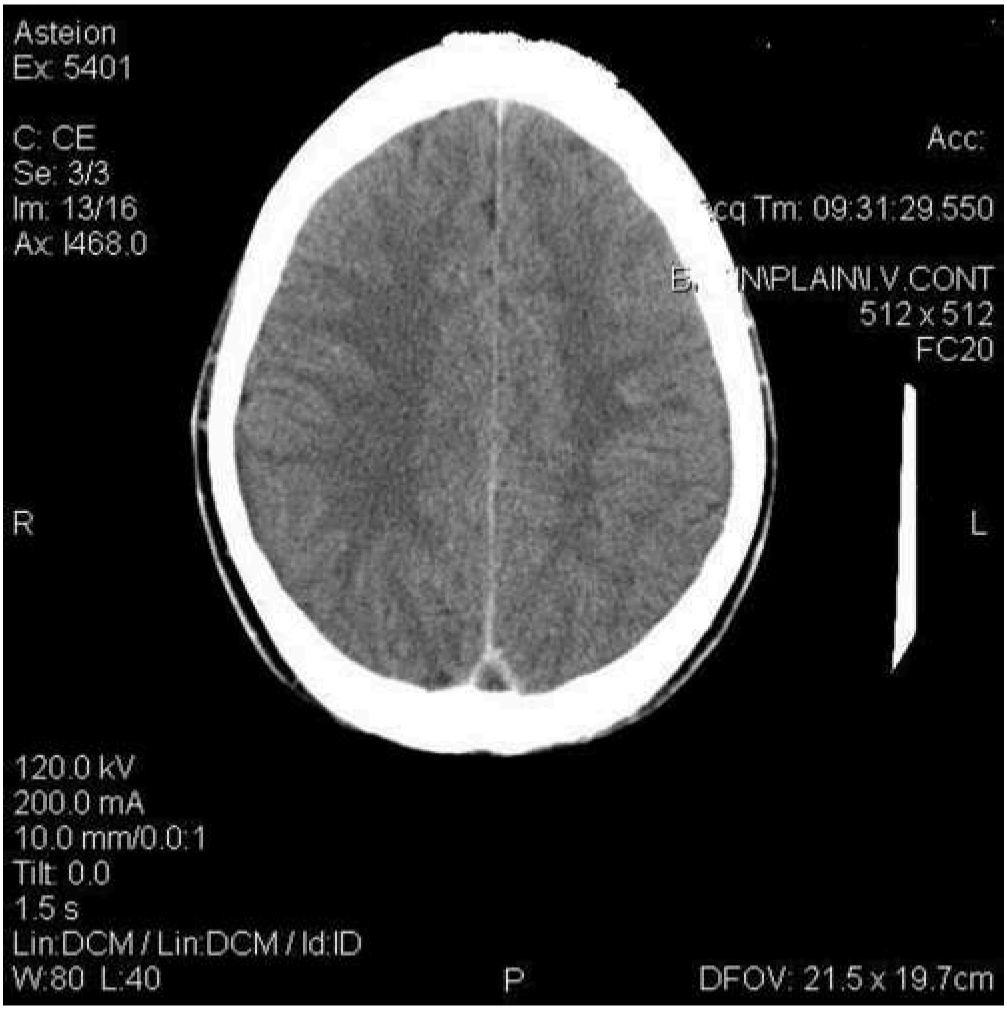
Sagittal sinus thrombosis
- may present with seizures and hemiplegia
- parasagittal biparietal or bifrontal haemorrhagic infarctions are sometimes seen
- 'empty delta sign' seen on venography

Cavernous sinus thrombosis
- other causes of cavernous sinus syndrome: local infection (e.g. sinusitis), neoplasia, trauma
- periorbital erythema and oedema

- ophthalmoplegia: 6th nerve damage typically occurs before 3rd & 4th
- trigeminal nerve involvement may lead to hyperaesthesia of upper face and eye pain
- central retinal vein thrombosis

Lateral sinus thrombosis

- 6th and 7th cranial nerve palsies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CT with contrast demonstrating a **superior sagittal sinus thrombosis** showing the typical empty delta sign. Look at the 'bottom' of the scan for the triangular shaped dural sinus. This should normally be white due to it being filled with contrast. The empty delta sign occurs when the thrombus fails to enhance within the dural sinus and is outlined by enhanced collateral channels in the falx. This sign is seen in only about 25%-30% of cases but is highly diagnostic for sagittal sinus thrombosis

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **20%**

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Question 79 of 409

A 56 year-old consultant surgeon has recently been diagnosed with idiopathic Parkinson's disease, and attends clinic for review.

You read the previous clinic letter and see that he presented with tremor affecting mainly the right hand, which forced him to stop operating. On examination he also had some bradykinesia and rigidity of the right hand. He was started on co-beneldopa 100/25, initially one tablet three times daily, with instructions to up-titrate as tolerated.

When you see him today, he is concerned that there has been no improvement in the tremor. This is causing him considerable distress as he hopes to return to clinical work. His motor control is otherwise good, and throughout the day and night he has no difficulty with 'off' periods or freezing. He currently takes co-beneldopa 100/25, two tablets three times daily, and reports no complications.

On examination there is a resting tremor of the right hand. There is no discernable rigidity or bradykinesia.

What is the best treatment option?

○ Increase dose of co-beneldopa

○ Increase frequency of co-beneldopa

○ Add procyclidine

○ Add entacapone

○ Add selegiline

Submit answer

Reference ranges

Score: **20%**

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On examination there is a resting tremor of the right hand. There is no discernable rigidity or bradykinesia.

What is the best treatment option?

- Increase dose of co-beneldopa 13%
- Increase frequency of co-beneldopa 11%
- Add procyclidine 37%
- Add entacapone 26%
- Add selegiline 13%

Tremor-predominant Parkinson's disease can be highly disabling and tremor may persist despite standard treatment with dopaminergic agents, which primarily improve bradykinesia. First-line treatment is such cases is the addition of an anticholinergic drug such as procyclidine, orphenadrine, or trihexyphenidyl.

The main side-effect which limits the use of anticholinergics in older patients is cognitive dysfunction, and dementia is a relative contra-indication. Other side effects include blurred vision, urinary retention, nausea, constipation, and dry mouth.

Second-line treatments for persistent tremor include amantidine, clozapine, and propranolol. Deep brain stimulation may be used in refractory cases.

Increasing the dose of L-dopa is required when 'off' symptoms (bradykinesia, rigidity, freezing) do not respond to the starting dose, however this patient is taking a reasonable dose of L-dopa (600mg) and is not troubled by 'off' symptoms.

Increasing the frequency of L-dopa, which has a short half-life, is required when patients become 'off' in between doses. As the disease progresses the on period after each dose becomes shorter.

Entacapone and selegiline are used as adjuncts to prolong the 'on' period after a dose of L-dopa.

👍 👎 Discuss (2) Improve

Next question >

Parkinson's disease: management

Parkinsons disease should only be diagnosed, and management initiated, by a specialist with expertise in movement disorders. However, it is important for all doctors to be aware of the medications used in Parkinson's given the prevalence of this condition. NICE published guidelines in 2017 regarding the management of Parkinson's disease.

For first-line treatment:
- if the motor symptoms are affecting the patient's quality of life: levodopa
- if the motor symptoms are not affecting the patient's quality of life: dopamine agonist (non-ergot derived), levodopa or monoamine oxidase B (MAO-B) inhibitor

Whilst all drugs used to treat Parkinson's can cause a wide variety of side-effects NICE produced tables to help with decision making:

	Levodopa	**Dopamine agonists**	**MAO-B inhibitors**
Motor symptoms	More improvement in motor symptoms	Less improvement in motor symptoms	Less improvement in motor symptoms
Activities of daily living	More improvement in activities of daily living	Less improvement in activities of daily living	Less improvement in activities of daily living
Motor complications	More motor complications	Fewer motor complications	Fewer motor complications
Adverse events	Fewer specified adverse events*	More specified adverse events*	Fewer specified adverse events*

* excessive sleepiness, hallucinations and impulse control disorders

If a patient continues to have symptoms despite optimal levodopa treatment or has developed dyskinesia then NICE recommend the addition of a dopamine agonist, MAO-B inhibitor or catechol-O-methyl transferase (COMT) inhibitor as an adjunct. Again, NICE summarise the main points in terms of decision making:

	Dopamine agonists	**MAO-B inhibitors**	**COMT inhibitors**	**Amantadine**
Motor symptoms	Improvement in motor symptoms	Improvement in motor symptoms	Improvement in motor symptoms	No evidence of improvement in motor symptoms
Activities of daily living	Improvement in activities of daily living	Improvement in activities of daily living	Improvement in activities of daily living	No evidence of improvement in activities of daily living
Off time	More off-time reduction	Off-time reduction	Off-time reduction	No studies reporting this outcome
Adverse events	Intermediate risk of adverse events	Fewer adverse events	More adverse events	No studies reporting this outcome
Hallucinations	More risk of hallucinations	Lower risk of hallucinations	Lower risk of hallucinations	No studies reporting this outcome

Specific points regarding Parkinson's medication

NICE reminds us of the risk of acute akinesia or neuroleptic malignant syndrome if medication is not taken/absorbed (for example due to gastroenteritis) and advise against giving patients a 'drug holiday' for the same reason.

Impulse control disorders have become a significant issue in recent years. These can occur with any dopaminergic therapy but are more common with:

- dopamine agonist therapy
- a history of previous impulsive behaviours
- a history of alcohol consumption and/or smoking

If excessive daytime sleepiness develops then patients should not drive. Medication should be adjusted to control symptoms. Modafinil can be considered if alternative strategies fail.

If orthostatic hypotension develops then a medication review looking at potential causes should be done. If symptoms persist then midodrine (acts on peripheral alpha-adrenergic receptors to increase arterial resistance) can be considered.

Consider glycopyrronium bromide to manage drooling of saliva in people with Parkinson's disease.

Further information regarding specific anti-Parkinson's medication

Levodopa

- nearly always combined with a decarboxylase inhibitor (e.g. carbidopa or benserazide)
    - this prevents the peripheral metabolism of levodopa to dopamine outside of the brain and hence can reduce side effects
- common adverse effects:
    - dry mouth
    - anorexia
    - palpitations
    - postural hypotension
    - psychosis
- some adverse effects are due to the difficulty in achieving a steady dose of levodopa
    - end-of-dose wearing off: symptoms often worsen towards the end of dosage interval. This results in a decline of motor activity
    - 'on-off' phenomenon: large variations in motor performance, with normal function during the 'on' period, and weakness and restricted mobility during the 'off' period
    - dyskinesias at peak dose: dystonia, chorea and athetosis (involuntary writhing movements)
    - these effects may worsen over time with - clinicians therefore may limit doses until necessary
- it is important not to acutely stop levodopa, for example, if a patient is admitted to hospital
    - if a patient with Parkinson's disease cannot take levodopa orally, they can be given a dopamine agonist patch as rescue medication to prevent acute dystonia

Dopamine receptor agonists

- e.g. bromocriptine, ropinirole, cabergoline, apomorphine
- ergot-derived dopamine receptor agonists (bromocriptine, cabergoline) have been associated with pulmonary, retroperitoneal and cardiac fibrosis. The Committee on Safety of Medicines advice that an echocardiogram, ESR, creatinine and chest x-ray should be obtained prior to treatment and patients should be closely monitored
- patients should be warned about the potential for dopamine receptor agonists to cause impulse control disorders and excessive daytime somnolence
- more likely than levodopa to cause hallucinations in older patients. Nasal congestion and postural hypotension are also seen in some patients

MAO-B (Monoamine Oxidase-B) inhibitors

- e.g. selegiline
- inhibits the breakdown of dopamine secreted by the dopaminergic neurons

Amantadine

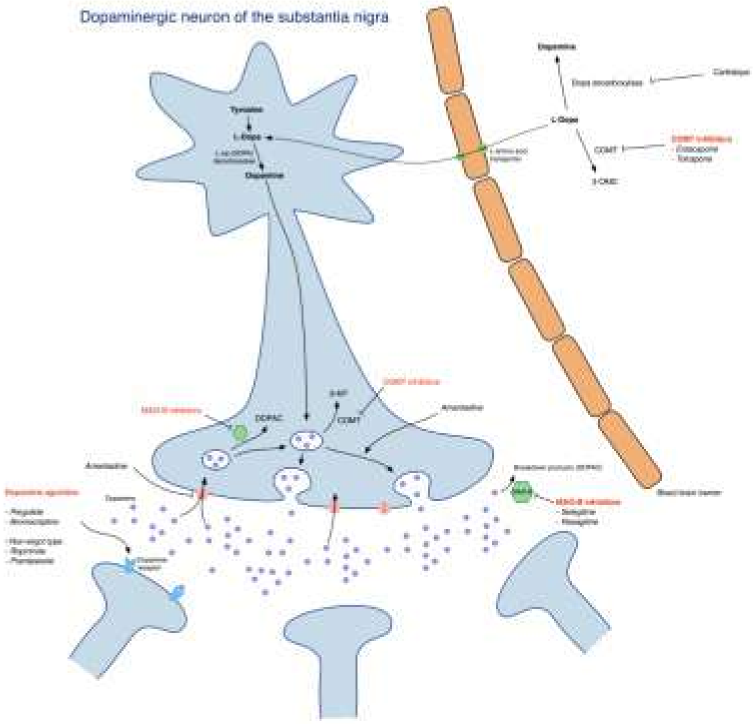
- mechanism is not fully understood, probably increases dopamine release and inhibits its uptake at dopaminergic synapses
- side-effects include ataxia, slurred speech, confusion, dizziness and livedo reticularis

COMT (Catechol-O-Methyl Transferase) inhibitors

- e.g. entacapone, tolcapone
- COMT is an enzyme involved in the breakdown of dopamine, and hence may be used as an adjunct to levodopa therapy
- used in conjunction with levodopa in patients with established PD

Antimuscarinics

- block cholinergic receptors
- now used more to treat drug-induced parkinsonism rather than idiopathic Parkinson's disease
- help tremor and rigidity
- e.g. procyclidine, benzotropine, trihexyphenidyl (benzhexol)


Dopaminergic neuron of the substantia nigra
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Amantadine

Image sourced from Wikipedia

Diagram showing the mechanism of action of Parkinson's drugs

Next question

Textbooks

High-yield textbook

Extended textbook

Links

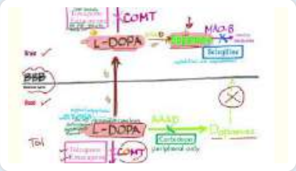
NICE 23 12

2017 Parkinson's disease guidelines

Suggest link

Report broken link

Media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The use of Levodopa/ Carbidopa in the treatment of Parkinson's disease

Medicosis Perfectionalis - YouTube 13 0

PARKINSON'S
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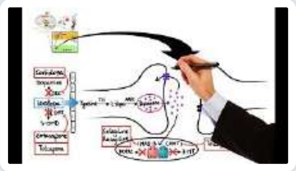
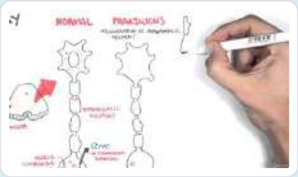
Parkinson's disease pharmacology

Soton Brain Hub - YouTube 4 0



Pharmacology - DRUGS FOR PARKINSON'S DISEASE

Speed Pharmacology - YouTube 👍 8 👎 1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Pharmacology - Parkinson's Disease

Armando Hasudungan - YouTube 👍 16 👎 2

PARKINSON'S
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Parkinson's disease

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PARKINSON'S
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Understanding Parkinson's Disease

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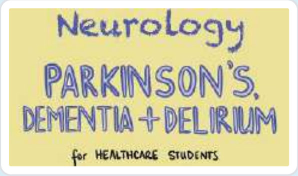
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for HEALTHCARE STUDENTS

Parkinson's disease

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Score: **20%**

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You see a 46 year-old man who has been referred by his GP to the neurology clinic.

He gives a one year history of facial pain. The pain particularly comes on when he is shaving or brushing his teeth, and he describes it as 'stabbing' through the teeth of his upper jaw and over the left side of his face. He has seen a succession of dentists and had several teeth removed, with no relief. The pain has been getting progressively worse, and whereas before it occurred in discrete attacks, it now occurs almost all the time. He has read several online sources and has become convinced that he has a brain tumour, which has led to him becoming depressed and withdrawn.

His past medical history includes essential hypertension, for which he takes perindopril. He also suffers from sinusitis, and has had a sinus washout on more than one occasion. Two years ago whilst on a business trip abroad he had a problem with the vision in his right eye, which spontaneously resolved over a few weeks, and for which he sought no treatment.

General examination is unremarkable. Cranial nerve examination is largely normal but you notice that there is a patch of numbness over the left cheek. Power is 5/5 across all muscle groups in the limbs, reflexes are normal, and plantars are downgoing. Sensation is the limbs is normal.

What is the most appropriate course of action?

○ Reassure him he has trigeminal neuralgia, for which unfortunately there is no treatment

○ Reassure him he has trigeminal neuralgia, and start carbamazepine 300mg daily

○ Refer to the neurosurgeons for microvascular decompression

○ Perform MR head with gadolinium contrast

○ Perform CT head with contrast

Submit answer

Reference ranges

Score: **20%**

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What is the most appropriate course of action?

Reassure him he has trigeminal neuralgia, for which unfortunately there is no treatment	1%
Reassure him he has trigeminal neuralgia, and start carbamazepine 300mg daily	45%
Refer to the neurosurgeons for microvascular decompression	6%
Perform MR head with gadolinium contrast	44%
Perform CT head with contrast	4%

This man needs investigation before any further management is considered. He has pain which is typical for trigeminal neuralgia. However, objective sensory loss does not occur in idiopathic trigeminal neuralgia and is highly suggestive of an underlying cause. This may occur due to compression of the trigeminal nerve by a space-occupying lesion such a tumour, or by a demyelinating plaque of multiple sclerosis affecting the trigeminal root at the pons. The episode of transient unilateral visual disturbance that this man suffered two years ago raises the possibility of optic neuritis, and thus the history is suspicious for MS. Malignancy is also a distinct possibility. Gadolinium-enhanced MR of the head is the best investigation to detect both.

Carbamazepine is an effective treatment for trigeminal neuralgia and produces at least some response in 70% of patients. Neurosurgical options such as microvascular decompression are considered only once medical options have been exhausted.

Discuss (5) Improve

Next question

Trigeminal neuralgia

Trigeminal neuralgia is a pain syndrome characterised by severe unilateral pain. The vast majority of cases are idiopathic but compression of the trigeminal roots by tumours or vascular problems may occur.

The International Headache Society defines trigeminal neuralgia as:

- a unilateral disorder characterised by brief electric shock-like pains, abrupt in onset and termination, limited to one or more divisions of the trigeminal nerve
- the pain is commonly evoked by light touch, including washing, shaving, smoking, talking, and brushing the teeth (trigger factors), and frequently occurs spontaneously
- small areas in the nasolabial fold or chin may be particularly susceptible to the precipitation of pain (trigger areas)
- the pains usually remit for variable periods

The following as red flag symptoms and signs suggesting a serious underlying cause:

- Sensory changes
- Deafness or other ear problems
- History of skin or oral lesions that could spread perineurally
- Pain only in the ophthalmic division of the trigeminal nerve (eye socket, forehead, and nose), or bilaterally
- Optic neuritis
- A family history of multiple sclerosis
- Age of onset before 40 years

Management

- carbamazepine is first-line
- failure to respond to treatment or atypical features (e.g. < 50 years old) should prompt referral to neurology

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 18 👎 3

Trigeminal Neuralgia guidelines

Suggest link Report broken link

Score: **20%**

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A 68-year-old man who has a history of Type 2 diabetes for the past 12 years comes to the clinic for review. His diabetes is currently managed with BD mixed insulin and over the past year he has suffered increasing pins and needles and pain in both feet. Apart from Type 2 diabetes, a previous myocardial infarction and intermittent urinary retention over the past year are noted. His GP has diagnosed him with benign prostatic hypertrophy. Examination reveals a blood pressure of 148/82 mmHg, his pulse is 80 beats per minute and regular. There is peripheral neuropathy affecting both feet, which are numb to just below the ankle. Routine bloods including renal function are unremarkable.

Which of the following is the most appropriate intervention?

○ Amitriptyline

○ Carbamazepine

○ Duloxetine

○ Pregabalin

○ Tramadol

Submit answer

Reference ranges

Score: **20%**

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A 68-year-old man who has a history of Type 2 diabetes for the past 12 years comes to the clinic for review. His diabetes is currently managed with BD mixed insulin and over the past year he has suffered increasing pins and needles and pain in both feet. Apart from Type 2 diabetes, a previous myocardial infarction and intermittent urinary retention over the past year are noted. His GP has diagnosed him with benign prostatic hypertrophy. Examination reveals a blood pressure of 148/82 mmHg, his pulse is 80 beats per minute and regular. There is peripheral neuropathy affecting both feet, which are numb to just below the ankle. Routine bloods including renal function are unremarkable.

Which of the following is the most appropriate intervention?

Amitriptyline	10%
Carbamazepine	3%
Duloxetine	50%
Pregabalin	36%
Tramadol	1%

The correct answer is pregabalin. Pregabalin is a GABA analogue and is recommended as an option by NICE for the treatment of neuropathic pain. it has the advantage versus gabapentin of less frequent daily dosing which drives improved compliance. It is also not associated with urinary retention which drives its selection versus other options listed.

Both amitriptyline and duloxetine are alternative first-line options for the treatment of diabetic neuropathic pain, although they are avoided here because both can be associated with urinary retention. Carbamazepine is not recommended for first-line treatment of diabetic neuropathy, and nor is tramadol because it is only partially effective and has addictive potential.

Discuss (4) Improve

Next question >

Neuropathic pain

Neuropathic pain may be defined as pain which arises following damage or disruption of the nervous system. It is often difficult to treat and responds poorly to standard analgesia.

Examples include:

- diabetic neuropathy
- post-herpetic neuralgia
- trigeminal neuralgia
- prolapsed intervertebral disc

NICE updated their guidance on the management of neuropathic pain in 2013:

- first-line treatment*: amitriptyline, duloxetine, gabapentin or pregabalin
    - if the first-line drug treatment does not work try one of the other 3 drugs
    - in contrast to standard analgesics, drugs for neuropathic pain are typically used as monotherapy, i.e. if not working then drugs should be switched, not added
- tramadol may be used as 'rescue therapy' for exacerbations of neuropathic pain
- topical capsaicin may be used for localised neuropathic pain (e.g. post-herpetic neuralgia)
- pain management clinics may be useful in patients with resistant problems

*please note that for some specific conditions the guidance may vary. For example carbamazepine is used first-line for trigeminal neuralgia

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 13 4

2013 Neuropathic pain guidelines

Suggest link

Report broken link

Media

HEART ATTACK
REFERRED PAIN
ACUTE PAIN
CHRONIC PAIN
PAIN
BRAIN
SPINAL CORD
C FIBRES
A-DELTA FIBRES
RECEPTORS
PERIPHERAL NERVES

Understanding The Causes of Pain and Pain Management

Zero To Finals - YouTube 👍 1 👎 0

Report broken media

Score: **20%**

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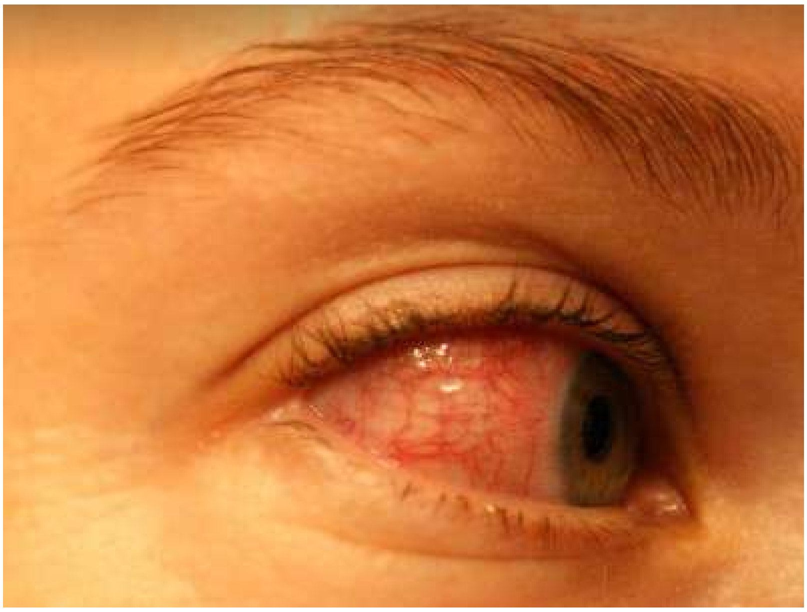
Question 82 of 409

A 42-year-old woman with rheumatoid arthritis presents to the clinic with complaints of pain and redness in the right eye. Her symptoms of rheumatoid arthritis are well-controlled and she is currently taking methotrexate and hydroxychloroquine. On examination, her right eye is watery and red. An image of her right eye is attached.



What is the most appropriate next step?

- ○ Reassurance only
- ○ Same-day assessment by an ophthalmologist
- ○ Stop hydroxychloroquine
- ○ Topical chloramphenicol
- ○ Topical glucocorticoids

Submit answer

Reference ranges

Score: **20%**

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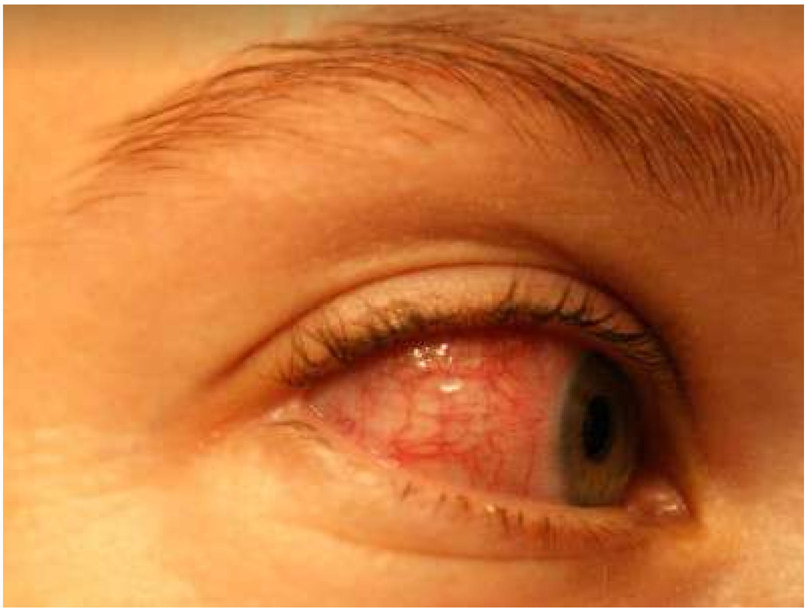
Question 82 of 409

A 42-year-old woman with rheumatoid arthritis presents to the clinic with complaints of pain and redness in the right eye. Her symptoms of rheumatoid arthritis are well-controlled and she is currently taking methotrexate and hydroxychloroquine. On examination, her right eye is watery and red. An image of her right eye is attached.



What is the most appropriate next step?

Reassurance only	16%
Same-day assessment by an ophthalmologist	43%
Stop hydroxychloroquine	9%
Topical chloramphenicol	4%
Topical glucocorticoids	28%

The patient in this scenario has developed scleritis and therefore requires **same-day assessment by an ophthalmologist** . Painful red eye in a patient with rheumatoid arthritis and the image shown in this scenario is typical of scleritis. The image shows a thickening of the sclera (pinkish-coloured) with dilated episcleral vessels. Scleritis is a full-thickening inflammation of the sclera and is characterised by pain and redness of the eye with a gradual decrease in vision. Rheumatoid arthritis is the most common disease associated with scleritis. Patients with scleritis should be offered a same-day assessment by an ophthalmologist because vision-threatening complications can occur if scleritis is left untreated.

Episcleritis is the most important differential here that can be managed by **reassurance only** , in contrast to scleritis. Episcleritis is characterised by a gritty sensation in the eye and redness of the sclera with normal vision. Episcleritis is a painless condition and a history of painful red eye in this scenario rules out episcleritis.

There is no indication to **stop hydroxychloroquine** as this drug has no role in causing scleritis. Hydroxychloroquine is known for its ophthalmic toxicities but it rarely causes scleritis. Ocular side-effects of hydroxychloroquine include retinopathy, colour blindness, lens opacity, and keratopathy.

Topical chloramphenicol has no role in treating scleritis. Chloramphenicol ointment is the first-line agent to treat conjunctivitis which can be considered here given the acute onset of red eye. There is no history of itching or discharge in this scenario which makes conjunctivitis a less-likely diagnosis.

Oral glucocorticoids, and not **topical glucocorticoids** are used in treating scleritis. Oral glucocorticoids are reserved for only severe presentations of scleritis. Other immunosuppressive drugs are used to treat resistant cases of scleritis.

👍 👎 Discuss (3) Improve

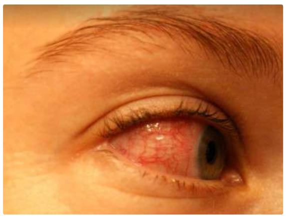
Next question >

Scleritis ★

Scleritis describes full-thickness inflammation of the sclera. It generally has a non-infective cause and typically causes a red, painful eye.

Risk factors
- rheumatoid arthritis: the most commonly associated condition
- systemic lupus erythematosus
- sarcoidosis
- granulomatosis with polyangiitis

Features
- red eye
- classically painful (in comparison to episcleritis), but sometimes only mild pain/discomfort is present
- watering and photophobia are common
- gradual decrease in vision



Management

- same-day assessment by an ophthalmologist
- oral NSAIDs are typically used first-line
- oral glucocorticoids may be used for more severe presentations
- immunosuppressive drugs for resistant cases (and also to treat any underlying associated diseases)

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20%**

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A 52-year-old man is admitted to the stroke unit with a right total anterior circulation syndrome (TACS) infarct. He arrived at hospital 2.5 hours after the onset of his symptoms and was treated with intravenous alteplase at 3 hours post-onset.

He is known have an atrial septal defect which was discovered after a murmur was heard at a routine insurance medical several years ago. He works in the oil business and has recently returned from a business trip to Saudi Arabia.

On examination the following day there subtle signs of improvement with increased movement in his left hand. However, the rest of his arm remains flaccid and he has persisting dense hemiplegia affecting his right leg. He has a notable homonymous hemianopia on examination. A routine CT Brain 24-hours post-thrombolysis revealed established ischaemic changes in the MCA territory with new petechial haemorrhage along the border of the infarct.

Later that evening, his conscious level falls. His Glasgow Coma Scale changes from E4 M6 V2, to E2, M4 V2. His blood pressure is 187/112 mmHg.

Urgent bloods reveal:

Haemoglobin	120 g/l
Prothrombin time	27 seconds
Activated partial thromboplastin time (APTT)	49 seconds

What intervention is likely to have the most benefit?

- ○ Correction of coagulopathy
- ○ Decompressive craniotomy
- ○ Control of blood pressure
- ○ Protamine sulphate
- ○ Tranexamic acid

Submit answer

Reference ranges ⌵

Score: **20%**

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Urgent bloods reveal:

Haemoglobin	120 g/l
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Activated partial thromboplastin time (APTT)	49 seconds

What intervention is likely to have the most benefit?

Correction of coagulopathy	14%
Decompressive craniotomy	40%
Control of blood pressure	38%
Protamine sulphate	4%
Tranexamic acid	4%

This patient is at high risk of malignant middle cerebral artery (MCA) syndrome. Malignant MCA syndrome typically occurs in younger patients who have suffered an extensive ischaemic stroke (usually in the MCA territory). After infarction the brain swells. As younger brains are likely to have less atrophy, there is less room for expansion. Subsequently, intracranial hypertension develops.

This typically presents 48 hours after onset of stroke with reduced conscious level.

Thrombolysis should reduce the risk of infarction and subsequent brain oedema, however, re-canalisation is not achieved in every patient and thus thrombolysed patients can still develop malignant MCA syndrome. Furthermore, this patient was thrombolysed 3 hours after onset and so is likely to have established tissue infarction regardless. Treatment with urgent decompressive craniotomy can be life-saving. The procedure is particularly indicated in situations like this, where the patient has a non-dominant stroke and is, therefore, likely to recover language functions and have a more favourable outcome if they survive the acute event.

Acute haemorrhagic transformation is the main differential in this case. While the change in his conscious level should prompt repeat imaging (which would discriminate bleeding from oedema) the risk of large haemorrhagic transformation >24 hours post-thrombolysis is lower than the risk of malignant MCA syndrome. This is especially true when the previous scan has shown only grade I Haemorrhagic infarction (HI-1) as classified using the ECAS II grading system. Urgent discussion with neurosurgery is recommended.

👍 👎 Discuss (8) Improve

Next question >

Stroke: management of acute ischaemic stroke ★

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
    - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
    - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic

transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:
- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.
- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
  - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
  - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and

- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

+ Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 11 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 16

Stroke and TIA guidelines

Age and Ageing 👍 21 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

2023 National Clinical Guideline for Stroke

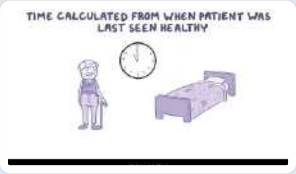
NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link

Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia.org
Acute ischaemic Stroke

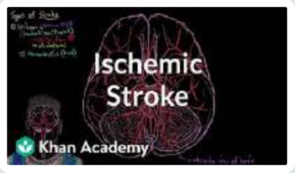
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

Khan Academy - YouTube 👍 1 👎 1

Report broken media

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Question 84 of 409

An 86-year-old man is brought in by ambulance following suspicion of a stroke. His wife noticed sudden right-sided weakness. He describes sudden onset of weakness with no preceding symptoms. He has a past medical history of hypertension, diabetes and glaucoma. On examination, he has right sided pyramidal weakness of the arm and leg with associated sensory loss as well as reduced tone. His left side is normal. Cranial nerve examination shows homonymous hemianopia but is otherwise normal. What is the most likely diagnosis?

- ○ Total anterior circulation stroke
- ○ Partial anterior circulation stroke
- ○ Lacunar stroke
- ○ Posterior circulation stroke
- ○ Brainstem stroke

Submit answer

Reference ranges

Score: **20%**

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Total anterior circulation stroke	38%
Partial anterior circulation stroke	41%
Lacunar stroke	7%
Posterior circulation stroke	11%
Brainstem stroke	4%

The correct answer is partial anterior circulation stroke. He has had sudden onset of unilateral weakness and sensory loss with homonymous hemianopia but no other evidence of higher cognitive dysfunction. The most likely diagnosis, as per the Oxford classification, is partial anterior circulation stroke. If he would have had evidence of higher further higher cognitive dysfunction, such as dysphasia, then a total anterior circulation stroke would be a better description. A lacunar stroke would cause isolated sensory or motor symptoms, not both. A posterior circulation stroke is more likely to affect cerebellar function whilst a brainstem stroke would affect cranial nerves profoundly.

Discuss (4) | Improve

Next question >

Stroke: types ★

The **Oxford Stroke Classification** (also known as the Bamford Classification) classifies strokes based on the initial symptoms. A summary is as follows:

The following criteria should be assessed:

- 1. unilateral hemiparesis and/or hemisensory loss of the face, arm & leg
- 2. homonymous hemianopia

- 3. higher cognitive dysfunction e.g. dysphasia

Total anterior circulation infarcts (TACI, c. 15%)

- involves middle and anterior cerebral arteries
- all 3 of the above criteria are present

Partial anterior circulation infarcts (PACI, c. 25%)

- involves smaller arteries of anterior circulation e.g. upper or lower division of middle cerebral artery
- 2 of the above criteria are present

Lacunar infarcts (LACI, c. 25%)

- involves perforating arteries around the internal capsule, thalamus and basal ganglia
- presents with 1 of the following:
- 1. unilateral weakness (and/or sensory deficit) of face and arm, arm and leg or all three.
- 2. pure sensory stroke.
- 3. ataxic hemiparesis

Posterior circulation infarcts (POCI, c. 25%)

- involves vertebrobasilar arteries
- presents with 1 of the following:
- 1. cerebellar or brainstem syndromes
- 2. loss of consciousness
- 3. isolated homonymous hemianopia

Other recognised patterns of stroke:

Lateral medullary syndrome (posterior inferior cerebellar artery)

- aka Wallenberg's syndrome
- ipsilateral: ataxia, nystagmus, dysphagia, facial numbness, cranial nerve palsy e.g. Horner's
- contralateral: limb sensory loss

Weber's syndrome

- ipsilateral III palsy
- contralateral weakness

+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Media

Strokes & The Rule of 4s

Dirty Medicine - YouTube 👍 19 👎 0

Normal HIT (worrisome)

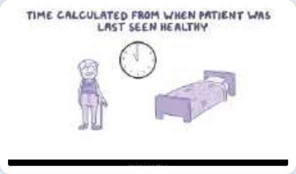
The HINTS exam in vertigo

Individual - YouTube 👍 5 👎 0

Brainstem Stroke

Brainstem stroke syndromes

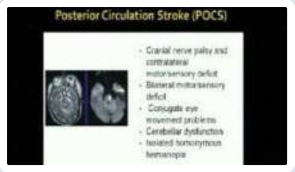
Soton Brain Hub - YouTube 👍 3 👎 0


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube

👍 2 👎 0


Posterior Circulation Stroke (POCS)

- Cranial nerve palsy and contralateral motor/sensory deficit
- Bilateral motor/sensory deficit
- Conjugate eye movement problems
- Cerebellar dysfunction
- Isolated homonymous hemianopia

Bamford classification

Individual - YouTube 👍 1 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 0 👎 0

NEUROLOGY
MIDBRAIN SYNDROMES

Midbrain Syndromes - Weberâ€™s Syndrome, Benediktâ€™s Syndrome and Parinaud Syndrome

Armando Hasudungan - YouTube 👍 0 👎 0

Report broken media

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A 24-year-old female is reporting difficulty in walking in an inpatient rehabilitation unit during recovery from spinal surgery. Six weeks previously, she underwent emergency spinal haematoma evacuation surgery under the neurosurgeons from T2 to T4 after sustaining a fall playing netball. She was previously fit and well, with no past medical history, giving birth to a healthy baby boy 18 months ago.

On examination, the patient has normal tone in her upper and lower limbs. Examination of her power while lying on her bed demonstrates 5/5 on the MRC power scale with normal sensation to cotton wool, pin prick and proprioception. Reflexes were present (2+) in her biceps, triceps, supinator, patella and ankles, both plantars were downgoing. On walking, the patient's gait is markedly abnormal, with both feet sliding along the floor for 80 metres of walking without lifting of her feet between steps. Cognition is intact. What is the optimal management?

- ○ Repeat MRI head
- ○ Repeat MRI whole spine
- ○ Nerve conduction studies and electromyography
- ○ Lumbar puncture
- ○ Education and reassurance

Submit answer

Reference ranges

Score: **20%**

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Repeat MRI head	5%
Repeat MRI whole spine	27%
Nerve conduction studies and electromyography	22%
Lumbar puncture	2%
✓ Education and reassurance	44%

This patient's gait is inconsistent with any pattern of organic pathology: it is inconsistent to be unable to lift your feet but have normal ankle dorsiflexion and plantarflexion. The patient has a combination of recent significant organic pathology and inorganic pathology, which is frequently the case in functional neurological disorders. Repeat MRI imaging, electrophysiology or lumbar puncture are unlikely to help and only medicalise the disorder. It is crucial to reinforce this message by avoiding unnecessary investigations or treatments. Management of functional neurology is based on education and reassurance, telling the patient that it is a 'conceptualisation' problem and not an issue with the brain or nerves. Psychiatric input may also have a role in this situation if the patient continues to be refractory to first line management: cognitive behavioural therapy may be helpful.

Discuss (6) Improve

Next question

Unexplained symptoms

There are a wide variety of psychiatric terms for patients who have symptoms for which no organic cause can be found:

Somatisation disorder
- multiple physical SYMPTOMS present for at least 2 years
- patient refuses to accept reassurance or negative test results

Illness anxiety disorder (hypochondriasis)
- persistent belief in the presence of an underlying serious DISEASE, e.g. cancer
- patient again refuses to accept reassurance or negative test results

Functional neurological disorder (conversion disorder)
- typically involves loss of motor or sensory function
- the patient doesn't consciously feign the symptoms (factitious disorder) or seek material gain (malingering)
- patients may be indifferent to their apparent disorder - la belle indifference - although this has not been backed up by some studies

Dissociative disorder
- dissociation is a process of 'separating off' certain memories from normal consciousness
- in contrast to conversion disorder involves psychiatric symptoms e.g. Amnesia, fugue, stupor
- dissociative identity disorder (DID) is the new term for multiple personality disorder as is the most severe form of dissociative disorder

Factitious disorder
- also known as Munchausen's syndrome
- the intentional production of physical or psychological symptoms

Malingering
- fraudulent simulation or exaggeration of symptoms with the intention of financial or other gain

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 12 👎 16

2013 Functional neurological symptoms

Suggest link

Report broken link

Media

Somatic symptom disorder

Osmosis - YouTube 👍 1 👎 0

Factitious disorder

Osmosis - YouTube 👍 1 👎 0

DISSOCIATIVE DISORDERS

Dissociative disorders

Osmosis - YouTube 👍 1 👎 0

Report broken media

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A 36-year-old male presents to your outpatient clinic with a progressive history over the past 5 years of increasing, progressive 'clumsiness'. His work colleagues had a long running joke with him that he is poorly coordinated for about the past five years but in recent weeks, he has noticed that he is unable to write legibly or even hold a key still using either hand to open a door. He denies any recent weight loss of night sweats, is otherwise healthy with no other past medical history. He is a lifelong non-smoker with a minimal alcohol history and lives with his wife and 2 children.

On examination, his cranial nerves were unremarkable except for mild multidirectional nystagmus at primary gaze. Fundoscopy was normal. Limb examination revealed a significant impairment of finger-nose and heel-shin testing. His gait, tone, power, sensation and reflexes were normal with downgoing plantars. A brief mini-mental state examination scored 30/30. An MRI head is awaited. His blood tests are as below:

Hb	158 g/l
Platelets	323 * 10^9/l
WBC	6.5 * 10^9/l

Na^+	141 mmol/l
K^+	4.9 mmol/l
Urea	6.6 mmol/l
Creatinine	85 µmol/l
CRP	2 mg/l
Creatine kinase	223 IU/l (50-335)
TSH	3.3 mu/l
Free T4	17 nmol/l
HIV	negative
Anti-neuronal antibodies	negative

Which investigation is most likely to yield the diagnosis?

○ Neurogenetics testing

○ CT chest, abdomen, pelvis

○ Vitamin B12 and folate levels

○ Lumbar puncture for cerebrospinal fluid including 14-3-3 and S100

○ Anti-GQ1b antibodies

Submit answer

Reference ranges

Score: **20%**

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Creatine kinase	223 IU/l (50-335)
TSH	3.3 mu/l
Free T4	17 nmol/l
HIV	negative
Anti-neuronal antibodies	negative

Which investigation is most likely to yield the diagnosis?

Neurogenetics testing	44%
CT chest, abdomen, pelvis	5%
Vitamin B12 and folate levels	7%

Lumbar puncture for cerebrospinal fluid including 14-3-3 and S100 24%

Anti-GQ1b antibodies 20%

There appears to be a chronic onset syndrome of pure ataxia, without systemic features or cognitive impairment, nor history of alcohol excess. The likely diagnosis is one of spinocerebellar ataxias (SCA), in particular one of the classified 'type III' syndrome such as SCA6, an adult onset cerebellar ataxia diagnosed by neurogenetic testing.

It is important to rule out key differential diagnoses however: patients with acute onset cerebellar syndrome should be considered for Miller-Fisher syndrome, a post-viral variant of Guillain Barre syndrome resulting in a triad of ophthalmoplegia, ataxia and areflexia, commonly after gastrointestinal infections, diagnosed with positive anti GQ1b antibodies. The time course for this illness over 5 years makes Miller-Fisher unlikely.

A subacute course of weeks and small number of months should alert you to possible prion disease such as Creutzfeldt-Jakob disease (CJD) and a paraneoplastic syndrome. The former typically presents with rapid cognitive decline and myoclonus, classically with MRI features (pulvinar sign, cortical ribboning), CSF features (14-3-3, S100, real-time QUIC) and EEG features (periodic spiking). The latter can present years before a solid tumour is identified on structural imaging with positive anti-neuronal antibodies, resulting in progressive symptoms as the cerebellum degenerate over months. Due to the low sensitivity of anti-neuronal antibodies, a negative result does not necessarily rule out paraneoplastic syndrome but such a long time course of 5 years would be unusual. The likelihood of identifying a significant mass on cross sectional CT imaging is low.

Lastly, the lack of a alcohol or gastrointestinal history makes cerebellar degeneration due to vitamin deficiency very unlikely. As a result, neurogenetics testing for SCA is most likely to yield the underlying diagnosis.

Discuss (5) Improve

Next question

Spinocerebellar ataxia ★

Spinocerebellar ataxias are a group of autosomal dominant disorders which are associated with the progressive development of ataxic features such as gait disturbance, nystagmus and tremor. The majority of affected patients develop symptoms within the 3rd and 4th decade.

Next question ›

Textbooks

High-yield textbook

Extended textbook

Score: **20%**

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A 32-year-old female presents with a 6-month history of progressive lower limb weakness and suprapubic tenderness. On examination, cranial nerves and upper limbs were unremarkable. You note 1/5 power in both lower limbs, hyperreflexic patella and ankle reflexes, bilateral clonus and upgoing plantar reflexes. The abdominal examination also revealed a suprapubic mass. After a urethral catheter was inserted, you note a residual volume of 1.8l. She is known to be HIV positive, diagnosed 8 years ago. Blood tests from an outpatient appointment revealed:

CMV IgG	positive
EBV IgG	positive
HTLV antibody	positive
Hepatitis B	negative
Hepatitis C	negative

An MRI spine is awaited. What is the most likely diagnosis?

○ Tropical spastic paraparesis

○ Spinal toxoplasmosis

○ CMV transverse myelitis

○ EBV transverse myelitis

○ Tabes dorsalis

Submit answer

Reference ranges

Score: **20%**

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A 32-year-old female presents with a 6-month history of progressive lower limb weakness and suprapubic tenderness. On examination, cranial nerves and upper limbs were unremarkable. You note 1/5 power in both lower limbs, hyperreflexic patella and ankle reflexes, bilateral clonus and upgoing plantar reflexes. The abdominal examination also revealed a suprapubic mass. After a urethral catheter was inserted, you note a residual volume of 1.8l. She is known to be HIV positive, diagnosed 8 years ago. Blood tests from an outpatient appointment revealed:

CMV IgG	positive
EBV IgG	positive
HTLV antibody	positive
Hepatitis B	negative
Hepatitis C	negative

An MRI spine is awaited. What is the most likely diagnosis?

Tropical spastic paraparesis	39%
Spinal toxoplasmosis	7%
CMV transverse myelitis	38%
EBV transverse myelitis	6%
Tabes dorsalis	10%

This is a HIV and HTLV-1 positive patient presenting with paraparesis and urinary retention, a classic presentation of tropical spastic paraparesis. Toxoplasmosis in HIV patients typically presents in the brain, lungs or as a chorioretinitis. Transient viral inflammatory transverse myelitis by CMV and EBV typically resolves within 3 months, the presence of IgG in both suggest previous exposure and is of limited significance. Tabes dorsalis is a possibility but peripheral reflexes are typically lost with an extensor plantar reflex. Diagnosis of HTLV-1 tropical spastic paraparesis relies on a combination of clinical features and HTLV-1 load in serum and CSF. Treatments are limited, with steroids, presumably as an anti-inflammatory, producing only mild effects in a small population of patients.

Discuss (4) Improve

Next question >

Spastic paraparesis

Spastic paraparesis describes a upper motor neuron pattern of weakness in the lower limbs

Causes
- demyelination e.g. multiple sclerosis
- cord compression: trauma, tumour
- parasagittal meningioma
- tropical spastic paraparesis
- transverse myelitis e.g. HIV
- syringomyelia
- hereditary spastic paraplegia
- osteoarthritis of the cervical spine

123

Next question >

Textbooks

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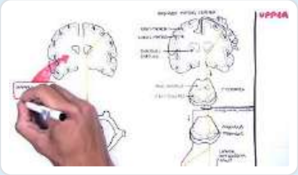
Extended textbook

Media



Introduction to Upper and Lower Motor Neuron Lesions

Armando Hasudungan - YouTube

👍 2 👎 0

Report broken media

Score: **20%**

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Question 88 of 409

A patient with refractory focal epilepsy on two agents is commenced on levetiracetam in addition. Which adverse effect should patients that have been commenced on this drug most importantly be warned about?

- ○ Oral hairy leukoplakia ×
- ○ Weight gain ×
- ○ Irritability and aggression ×
- ○ QT-interval prolongation ×
- ○ Nystagmus ×

Submit answer

Reference ranges

Score: **20%**

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Question 88 of 409 ✗

A patient with refractory focal epilepsy on two agents is commenced on levetiracetam in addition. Which adverse effect should patients that have been commenced on this drug most importantly be warned about?

Oral hairy leukoplakia	9%
Weight gain	25%
🟩 Irritability and aggression	37%
QT-interval prolongation	20%
🟥 Nystagmus	8%

It is important to recognise and know something about levetiracetam as it is increasingly becoming a favourite for neurologists to use these days, not least because of its favourable adverse effects profile (wherein it hardly has any compared to many others), but also its ease of use given that you do not need to monitor levels, and its oral and IV bioavailability are the same (useful in nil by mouth patients). It is also recently more affordable having now lost its patent. The one thing to watch out (and ensure you warn patients about) with levetiracetam is behavioural abnormalities and psychotic symptoms. These range from irritability to frank aggression. If there is a background of psychotic symptoms you may wish to avoid levetiracetam. It is something not to miss in the drug history of an epileptic patient who presents to the acute take with a change in behaviour.

1) Mbizvo GK, Dixon P, Hutton JL, Marson AG. The adverse effects profile of levetiracetam in epilepsy: a more detailed look. The International journal of neuroscience. 2014 Sep;124(9):627-34. PubMed PMID: 24256446.
2) Mbizvo GK, Dixon P, Hutton JL, Marson AG. Levetiracetam add-on for drug-resistant focal epilepsy: an updated Cochrane Review. The Cochrane database of systematic reviews. 2012;9:CD001901. PubMed PMID: 22972056.

Discuss (2) | Improve

Next question >

Epilepsy: treatment ★

Most neurologists now start antiepileptics following a second epileptic seizure. NICE guidelines suggest starting antiepileptics after the first seizure if any of the following are present:

- the patient has a neurological deficit
- brain imaging shows a structural abnormality
- the EEG shows unequivocal epileptic activity
- the patient or their family or carers consider the risk of having a further seizure unacceptable

It should be remembered that the following are only general guidelines. For example, **maternal use of sodium valproate is associated with a significant risk of neurodevelopmental delay in children**. Guidance is now clear that sodium valproate should not be used during pregnancy and in women of childbearing age unless clearly necessary.

Drug treatment according to the type of epilepsy

Generalised tonic-clonic seizures

- males: sodium valproate
- females: lamotrigine or levetiracetam
- girls aged under 10 years and who are unlikely to need treatment when they are old enough to have children or women who are unable to have children may be offered sodium valproate first-line

Focal seizures

- first line: lamotrigine or levetiracetam
- second line: carbamazepine, oxcarbazepine or zonisamide

Absence seizures (Petit mal)

- first line: ethosuximide
- second line:
  - male: sodium valproate
  - female: lamotrigine or levetiracetam
- carbamazepine may exacerbate absence seizures

Myoclonic seizures

- males: sodium valproate
- females: levetiracetam

Tonic or atonic seizures

- males: sodium valproate
- females: lamotrigine

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 28 👎 31

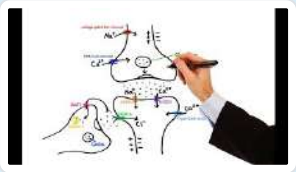
2022 Epilepsy guidelines

Royal College of Physicians 👍 15 👎 16

2013 Modern management of epilepsy

Suggest link Report broken link

Media



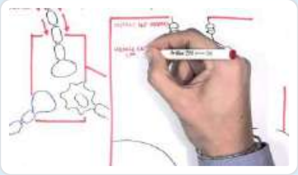
Antiepileptic drugs made easy

Speed Pharmacology - YouTube 👍 3 👎 0

EPILEPSY
Osmosis.org

Epilepsy (generalized, focal) - simple, complex, tonic, clonic, causes, symptoms

Osmosis - YouTube 👍 3 👎 0



Pharmacology - Antiepileptics

Armando Hasudungan - YouTube 👍 3 👎 1

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Question 89 of 409

A 48-year-old lady develops a sudden-onset left-sided headache in the face and neck areas 36 hours ago whilst at rest. It is of 10/10 severity and reached this maximum intensity within seconds. It has not subsided since and is throbbing in nature. She also developed a transient period of loss of vision in the left eye lasting 2 hours before resolving. She also says that 'food tastes funny' since these problems developed. On examination, she has small, sluggishly light-responsive left pupil compared to the right and partial left ptosis. The face is otherwise unremarkable to examine as is the remaining neurological examination. Routine blood investigations are unremarkable. Plain computerised tomography (CT) of the head is unremarkable. Lumbar puncture is negative for xanthochromia. CT angiogram of the head and neck vessels demonstrates a pseudo-lumen of the carotid artery. Which of the following treatments would you initiate?

- ○ High flow 100% oxygen ×
- ○ Prednisolone ×
- ○ Indomethacin ×
- ○ Aspirin ×
- ○ Acetazolamide ×

Submit answer

Reference ranges

Score: **20%**

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High flow 100% oxygen	15%
Prednisolone	14%
Indomethacin	22%
Aspirin	41%
Acetazolamide	8%

She has an internal carotid artery dissection on the left side culminating in a partial left-sided Horner's syndrome. The term 'partial' Horner's is used because anhidrosis is absent. The sympathetic fibres innervating the facial sweat glands are anatomically located on the external rather than the internal carotid artery; thus, anhidrosis is not a finding in the setting of internal carotid dissection.

In terms of management, there is little role for surgery in spontaneous carotid artery dissections. All dissections put people at risk of thromboembolic complications because of turbulent flow at the sight, therefore we normally initiate antiplatelet or anticoagulation therapy. There is little evidence for which is better over the other in this area although pragmatically speaking antiplatelets are safer. Candidates for angioplasty and stent placement include patients with persistent ischaemic symptoms despite adequate anticoagulation, patients with a contraindication to anticoagulant/antiplatelet therapy, and patients with significantly compromised cerebral blood flow.

In this particular case, the loss of vision points towards possible amaurosis fugax, backing up the need for anti-thromboembolic agents.

Discuss (6) Improve

Next question

Horner's syndrome

Features

- miosis (small pupil)
- ptosis
- enophthalmos* (sunken eye)
- anhidrosis (loss of sweating one side)

Distinguishing between causes

- heterochromia (difference in iris colour) is seen in congenital Horner's
- anhidrosis: see below
- pharmacologic tests
  - can be useful to confirm the diagnosis of Horner syndrome and localise the lesion
  - apraclonidine drops (an alpha-adrenergic agonist) can be used: causes pupillary dilation in Horner's syndrome due to denervation supersensitivity but produces mild pupillary constriction in the normal pupil by down-regulating the norepinephrine release at the synaptic cleft

Central lesions	**Pre-ganglionic lesions**	**Post-ganglionic lesions**
Anhidrosis of the face, arm and trunk	Anhidrosis of the face	No anhidrosis
Stroke **S**yringomyelia Multiple **s**clerosis Tumour Encephalitis	Pancoast's **t**umour **T**hyroidectomy **T**rauma Cervical rib	**C**arotid artery dissection **C**arotid aneurysm **C**avernous sinus thrombosis **C**luster headache

*in reality the appearance is due to a narrow palpebral aperture rather than true enophthalmos

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

HORNER'S SYNDROME

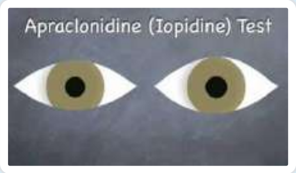
Horner's syndrome

Osmosis - YouTube 👍 12 👎 0

NEUROLOGY
HORNER SYNDROME

Horner's syndrome

Armando Hasudungan - YouTube 👍 5 👎 0


Apraclonidine (Iopidine) Test

How to Examine Horner's Syndrome

Individual - YouTube 👍 3 👎 0

Pancoast tumors

Osmosis - YouTube 👍 1 👎 0

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Score: **20%**

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Question 90 of 409

A 28-year-old gentleman student from Germany presents to you with right foot drop ongoing for two weeks with some numbness and tingling of the foot. These symptoms developed after he knelt down to pick something up from the floor. Three years ago he woke up from sleep with clawing of his fourth and fifth digit after having been asleep in a prone position and this lasted a week. Eight years ago he also had a left wrist and finger drop lasting three weeks after he sat on the couch with his left arm draped over the back of the couch for ten minutes. He denies falling asleep or remaining on the couch for a prolonged period. He has no other past medical history of note and has never sought medical advice for his problems. On examination, there is right foot drop (2/5 power) and similar weakness of dorsiflexion and eversion of the right foot. There is also sensory loss over the lower lateral part of the right leg and dorsum of the right foot in all modalities. Reflexes are intact. Neurological examination and general examination are otherwise unremarkable. Which of the following tests would confirm the suspected diagnosis?

- ○ Nerve conduction studies ×
- ○ Electromyography ×
- ○ Nerve biopsy ×
- ○ PMP22 gene testing ×
- ○ HBA1C ×

Submit answer

Reference ranges

Score: **20%**

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Nerve conduction studies	35%
Electromyography	8%
Nerve biopsy	10%
PMP22 gene testing	46%
HBA1C	1%

The diagnosis is Hereditary Neuropathy with Liability to Pressure Palsy (HNPP). This is a neurological syndrome in which trivial trauma to a peripheral nerve e.g. sleeping on a limb, results in a mononeuropathy which may take weeks to resolve. It usually presents in second or third decade of life. The condition is most common in families with Dutch or German ancestry. It is caused by a deletion in the peripheral myelin protein 22 gene on chromosome 17. It is an autosomal dominant condition.

The patient's presenting mononeuropathy is a common peroneal nerve palsy. Three years ago he had an ulnar nerve palsy and eight years ago he had a radial nerve palsy (also called Saturday night palsy).

Nerve conduction studies in HNPP are characteristic of a demyelinating neuropathy: conduction is slow and action potentials are small. Nerve biopsy may show a predominance of smaller fibres and localised thickening of the myelin sheath. These investigations are helpful in supporting the diagnosis although gene testing, if positive, is confirmatory.

Management is conservative with e.g. wrist splints, ankle-foot orthoses, and protective padding.

Discuss (8) Improve

Next question

Peripheral neuropathy: demyelinating vs. axonal

Demyelinating pathology

- Guillain-Barre syndrome
- chronic inflammatory demyelinating polyneuropathy (CIDP)
- amiodarone
- hereditary sensorimotor neuropathies (HSMN) type I
- paraprotein neuropathy

Axonal pathology

- alcohol
- diabetes mellitus*
- vasculitis
- vitamin B12 deficiency*
- hereditary sensorimotor neuropathies (HSMN) type II

* may also cause a demyelinating picture

AI + Q 123

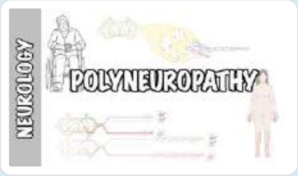
Next question

Textbooks

High-yield textbook

Extended textbook

Media


NEUROLOGY
POLYNEUROPATHY

Approach to Polyneuropathy - causes, pathophysiology, investigations

Armando Hasudungan - YouTube 👍 4 👎 0

Report broken media

Score: **20%**

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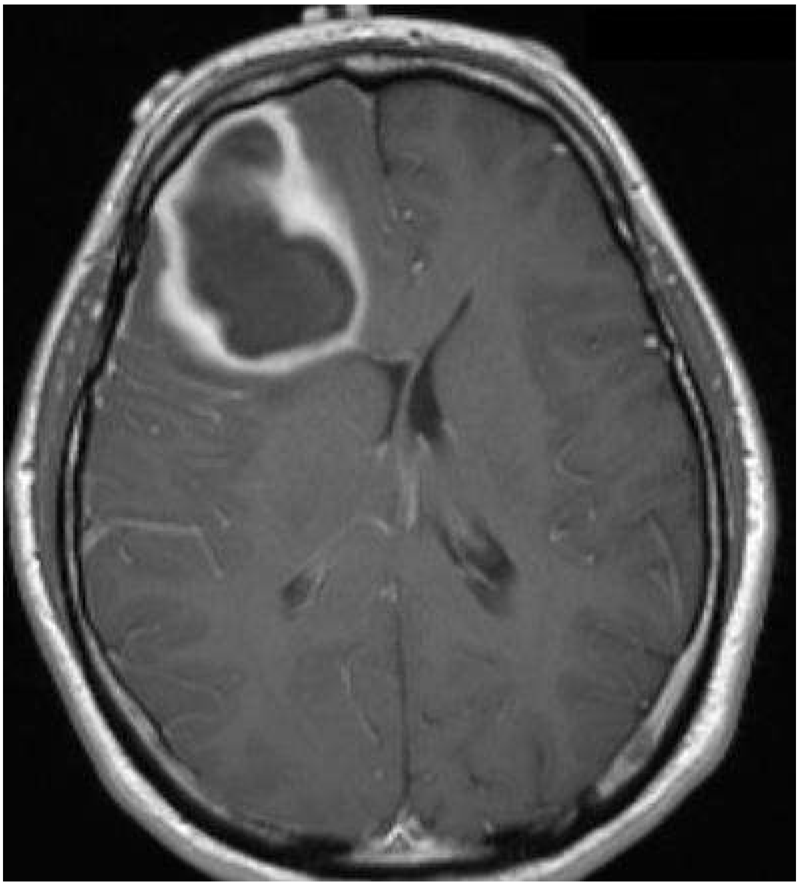
A 51-year-old man presents to the emergency department following a witnessed tonic-clonic seizure of 2 minutes duration. There is no history of epilepsy and he has never had a seizure before. Over the last week, he has been complaining of intermittent headaches and fevers. His past medical history includes hypertension for which he takes amlodipine, ramipril, and indapamide once daily.

On examination, he is drowsy with a GCS of 13 (E3V4M6). His chest sounds are clear and his heart sounds are normal. He has a soft and non-tender abdomen. Temperature is recorded as 38.1ºC.

A contrast-enhanced MRI scan is performed, as shown below:



© Image used on license from Radiopaedia

Given the likely diagnosis, which of the following is the most probable causative pathogen?

- ○ Cytomegalovirus
- ○ *Cryptococcus neoformans*
- ○ *Mycobacterium tuberculosis*
- ○ *Staphylococcus aureus*

○ *Toxoplasma gondii* ×

Submit answer

Reference ranges

Score: **20%**

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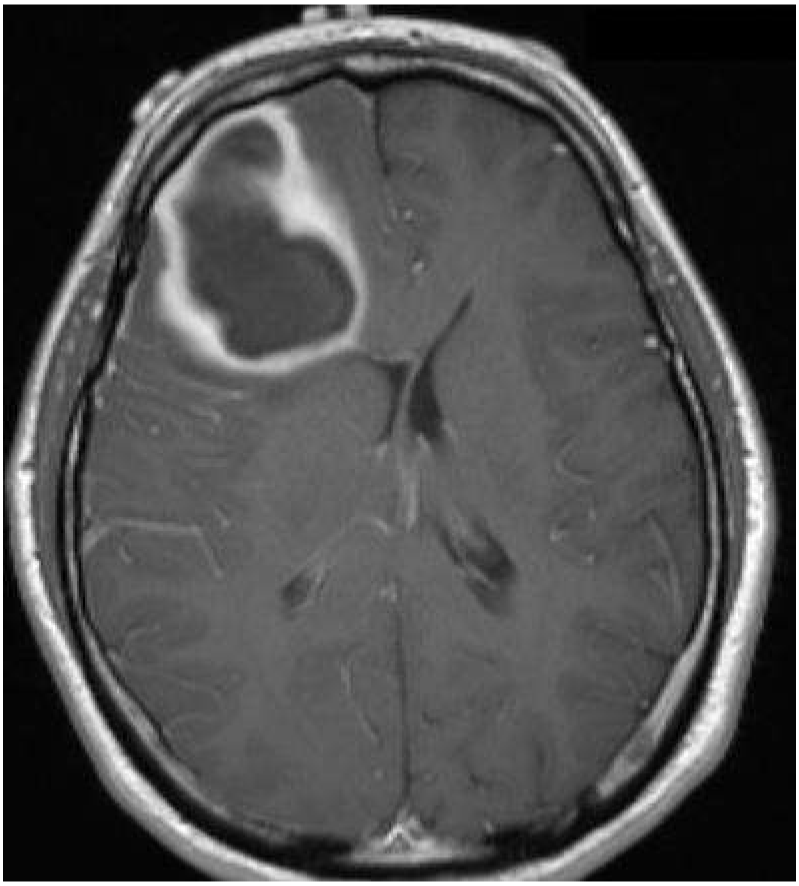

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Given the likely diagnosis, which of the following is the most probable causative pathogen?

Cytomegalovirus	5%
Cryptococcus neoformans	14%
Mycobacterium tuberculosis	8%
Staphylococcus aureus	44%

Toxoplasma gondii 27%

This patient's MRI brain shows a radiolucent space-occupying lesion with outermost ring enhancement in the frontal lobe. Causes of intracerebral lesions with ring enhancement include toxoplasmosis and intracerebral abscesses. Bacterial intracranial abscesses commonly arise from infections in adjacent structures including otitis media, sinusitis or mastoiditis. Symptoms of intracerebral abscesses depend on the structures involved but commonly include headache, fever, signs of increased intracranial pressure (e.g. papilloedema and seizures) and focal neurological signs. The clinical history of fever, headache and seizures combined with the imaging point toward the diagnosis of an intracerebral brain abscess. The most common pathogens responsible for intracerebral abscesses include *Staphylococcus aureus* and *Streptococcus pneumoniae*.

Cytomegalovirus (CMV) is a viral infection that commonly causes illness in patients with HIV, particularly in those with a CD4 count < 50 cells/mL. The most common form of CMV is CMV retinitis. However, CMV encephalitis is also common. On imaging, cerebral oedema may be seen. Meningoencephalitis and ventriculitis are also features of infection with CMV. These are not seen here, making CMV encephalitis unlikely.

Cryptococcus neoformans causes an opportunistic fungal infection (most frequently seen in patients with a history of human immunodeficiency virus) that causes headache, fevers, confusion and signs of raised intracranial pressure. Imaging studies show leptomeningeal enhancement, cryptococcomas, hydrocephalus or dilated perivascular spaces. These features are not seen in the imaging above, and the absence of a history of immunosuppression makes cryptococcal meningitis unlikely.

Mycobacterium tuberculosis can cause tuberculous meningitis with similar symptoms to those of bacterial meningitis (e.g. fever, headache, meningism). Tuberculous meningitis is more common in patients with HIV and can be seen on imaging as leptomeningitis from the presence of thick exudative material in the subarachnoid space.

Toxoplasmosis is an opportunistic infection caused by the parasite *Toxoplasma gondii*. It is the most common cause of neurological brain abscesses in patients with HIV with symptoms including headache, fever and neurological changes including seizures, hemiplegia, cerebellar signs, or an acute confusional state. Toxoplasmosis also presents with ring enhancement on contrast imaging. However, toxoplasmosis often presents with multiple ring-enhancing lesions. Furthermore, the absence of a history of immunosuppression makes an intracerebral abscess more likely.

👍 👎 Discuss (2) Improve

Next question >

Brain abscess ★

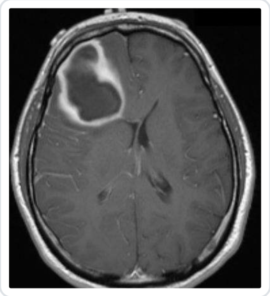
Brain abscesses may result from a number of causes including, extension of sepsis from middle ear or sinuses, trauma or surgery to the scalp, penetrating head injuries and embolic events from endocarditis

The presenting symptoms will depend upon the site of the abscess (those in critical areas e.g. motor cortex) will present earlier. Abscesses have a considerable mass effect in the brain and raised intracranial pressure is common.

- headache
    - often dull, persistent
- fever
    - may be absent and usually not the swinging pyrexia seen with abscesses at other sites
- focal neurology
    - e.g. oculomotor nerve palsy or abducens nerve palsy secondary to raised intracranial pressure
- other features consistent with raised intracranial pressure
    - nausea
    - papilloedema
    - seizures

Investigations

- Assessment of the patient includes imaging with CT scanning



Management

- surgery
    - a craniotomy is performed and the abscess cavity debrided
    - the abscess may reform because the head is closed following abscess drainage.
- IV antibiotics: IV 3rd-generation cephalosporin + metronidazole
- intracranial pressure management: e.g. dexamethasone

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **20%**

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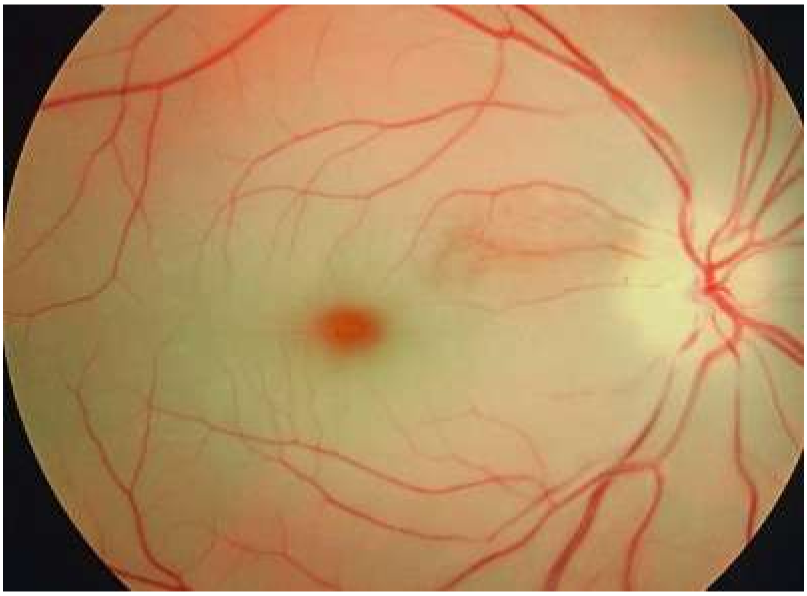
A 65-year-old man presents to the emergency department with sudden onset blindness in his left eye. He reports only being able to see out of his right eye since waking this morning. There is no history of eye pain or photophobia. His past medical history includes hypertension and hypothyroidism. He takes amlodipine, levothyroxine and regular co-codamol for recurrent headaches over the past 4-6 weeks.

On examination, he is alert and oriented with a GCS of 15/15. There is normal power and sensation in his upper and lower limbs bilaterally with normal tone and coordination. The visual acuity in his right eye is 6/9 and his left eye is only able to make out gross hand movements. The remainder of his cranial nerves are intact.

Fundoscopy of his eye is completed, as shown below:



Laboratory investigations are requested:

ESR	55 mm/hr	< (age / 2)

What is the recommended management for this patient?

○ Hyperbaric oxygen therapy

○ Intravenous methylprednisolone

○ Intravitreal anti-vascular endothelial growth factor (anti-VEGF)

○ Oral aspirin and referral for intra-arterial thrombolytic therapy

○ Topical corticosteroids

Submit answer

Reference ranges

Score: **20%**

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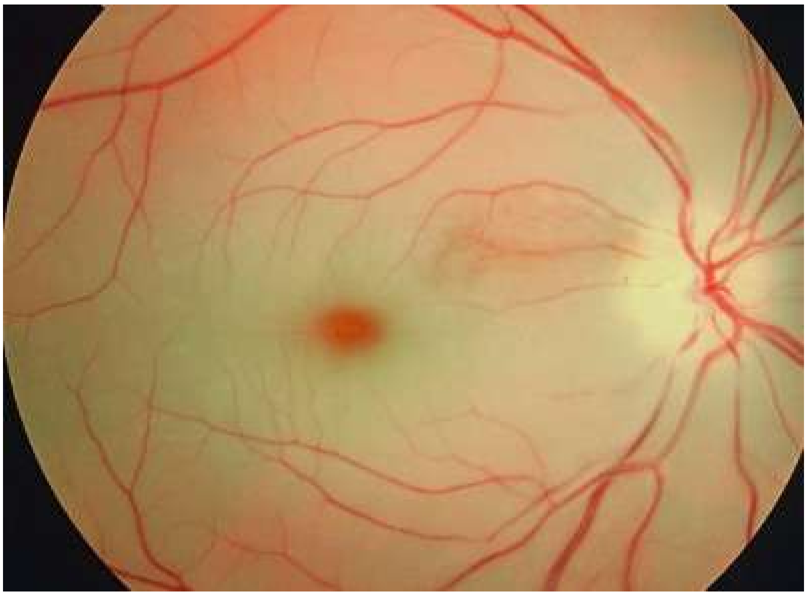


Laboratory investigations are requested:

ESR	55 mm/hr	< (age / 2)

What is the recommended management for this patient?

Hyperbaric oxygen therapy	2%
Intravenous methylprednisolone	34%
Intravitreal anti-vascular endothelial growth factor (anti-VEGF)	10%

Oral aspirin and referral for intra-arterial thrombolytic therapy 53%

Topical corticosteroids 1%

This patient has a fundoscopic image representing central retinal artery occlusion with the classic appearance of a 'cherry red spot' on a pale retina. Central retinal artery occlusion presents as an acute onset painless loss of vision that can either be secondary to thromboembolic or arteriopathy (e.g. temporal arteritis). The history of recurrent headaches and raised ESR makes temporal arteritis the more likely underlying diagnosis.

It is important to note that temporal arteritis can cause visual loss via two mechanisms. Most commonly, patients lose their vision secondary to arteritic anterior ischaemic optic neuropathy, which causes occlusion of blood supply to the optic nerve. However, in 10% of patients with visual loss secondary to temporal arteritis, the visual disturbance is caused by central retinal artery occlusion, as in this case.

Intravenous methylprednisolone is correct. Visual loss secondary to temporal arteritis is an ophthalmological and rheumatological emergency and treatment should be initiated promptly including intravenous methylprednisolone to avoid permanent vision loss. This can later be de-escalated to oral prednisolone.

Hyperbaric oxygen therapy is incorrect. This has been used as a treatment method to maintain adequate oxygenation of the retina whilst waiting for reperfusion. However, this treatment is not routinely used and would not reverse the underlying inflammatory process in temporal arteritis.

Intravitreal anti-vascular endothelial growth factor (anti-VEGF) is incorrect. This is used in the management of proliferative retinopathy (e.g. proliferative diabetic retinopathy). Proliferative retinopathy presents with multiple tortuous vessels and pre-retinal haemorrhages which are not seen here.

Oral aspirin and referral for intra-arterial thrombolytic therapy is incorrect. This would be an appropriate treatment for a thromboembolic cause of central retinal artery occlusion. However, this patient has a history of chronic headaches in association with a raised ESR which is suggestive of temporal arteritis. The pathology of artery occlusion in temporal arteritis is arteriopathy and inflammatory change rather than emboli and, therefore, methylprednisolone would be more appropriate.

Topical corticosteroids is incorrect. This is used to treat inflammatory ocular diseases such as anterior uveitis. This usually presents with eye pain and reduced visual acuity. Topical corticosteroids are not potent enough to treat arteriopathy. Rather, intravenous methylprednisolone is needed in the initial stages.

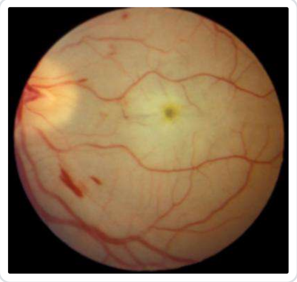
Discuss (3) Improve

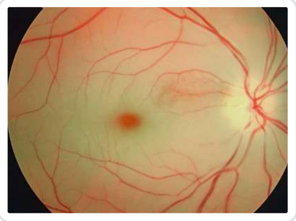
Next question

Central retinal artery occlusion

Central retinal artery occlusion is a relatively rare cause of sudden unilateral visual loss. It is due to thromboembolism (from atherosclerosis) or arteritis (e.g. temporal arteritis)

Features
- sudden, painless unilateral visual loss
- relative afferent pupillary defect
- 'cherry red' spot on a pale retina





Management is difficult and the prognosis is poor
- any underlying conditions should be identified and treated (e.g. intravenous steroids for temporal arteritis)
- if a patient presents acutely then Intraarterial thrombolysis may be attempted but currently, trials show mixed results.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

American Academy of Ophthalmology 👍 9 👎 4

Central Retinal Artery Occlusion

Suggest link Report broken link

Score: **20%**

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A 34 year old man attended the Emergency Department after experiencing a severe sudden onset headache at home. The pain was felt across the whole of the head and reached a 10/10 severity within a few seconds of onset. Since the headache began the patient had experienced a loud pulsatile pounding noise in his right ear. There was no reported loss of consciousness or other neurological symptoms. In the preceding days the patient had been fit and well. On close questioning he recalled that that morning he had been struck hard in the face by a soccer ball while watching his son's team.

The patient had no significant past medical history and took no medications. There was no family history of neurological disease.

On examination, the patient's right pupil was constricted compared to his left, with both pupils reactive to light. There was a partial ptosis of the right eye. Cranial nerve examination was otherwise unremarkable except for possible right hypoglossal palsy. Examination of the remainder of the peripheral nervous system was remarkable.

Details of initial investigations are given below.

CT brain (non-contrast): no acute intracranial pathology identified; normal ventricular system; no boney injury.

Lumbar puncture:

CSF red cells	2 / mm³
CSF white cells	4 / mm³
CSF gram stain	unremarkable
CSF glucose	60 % serum level
CSF protein	0.65 g / L
CSF	negative for haemoglobin break down products

What is the correct diagnosis?

○ Vertebral artery dissection ×

○ Internal carotid artery dissection ×

○ Posterior reversible encephalopathy syndrome ×

○ Reversible cerebrovascular vasoconstriction syndrome

○ Posterior communicating artery aneurysm

Submit answer

Reference ranges

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A 34 year old man attended the Emergency Department after experiencing a severe sudden onset headache at home. The pain was felt across the whole of the head and reached a 10/10 severity within a few seconds of onset. Since the headache began the patient had experienced a loud pulsatile pounding noise in his right ear. There was no reported loss of consciousness or other neurological symptoms. In the preceding days the patient had been fit and well. On close questioning he recalled that that morning he had been struck hard in the face by a soccer ball while watching his son's team.

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Details of initial investigations are given below.

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Lumbar puncture:

CSF red cells	2 / mm^3
CSF white cells	4 / mm^3
CSF gram stain	unremarkable
CSF glucose	60 % serum level
CSF protein	0.65 g / L
CSF	negative for haemoglobin break down products

What is the correct diagnosis?

Vertebral artery dissection	23%
Internal carotid artery dissection	36%
Posterior reversible encephalopathy syndrome	6%

Reversible cerebrovascular vasoconstriction syndrome 16%

Posterior communicating artery aneurysm 19%

Internal carotid artery dissection can present with thunderclap headache, commonly following minor trauma, as in this case. Possible associated symptoms and signs include homolateral Horner's syndrome, tinnitus or tongue palsy. CT brain and lumbar puncture are usually normal (or near normal in the case of LP) with diagnosis confirmed by angiography.

The remaining options are all possible causes of thunderclap headache. Vertebral artery dissection would typically cause a posterior circulation stroke pattern of signs and symptoms. Reversible cerebrovascular vasoconstriction syndrome is associated with hypertension and seizures and diagnosed by arterial beeding on angiography. Posterior reversible encephalopathy syndrome is also associated with hypertension and specific changes on MRI brain secondary to vasogenic brain oedema. Posterior communicating artery aneurysm causes compression of the third nerve with unilateral mydriasis without other signs of third nerve palsy.

Ducros A, Bousser M. Thunderclap headache. BMJ 2012;345:e8557

Discuss (9) Improve

Next question >

Thunderclap headache ★

Thunderclap headache describes a sudden (reaches maximum severity within seconds to minutes of onset) and severe headache.

Causes
- subarachnoid hemorrhage
- cerebral venous sinus thrombosis
- internal carotid artery dissection
- pituitary apoplexy
- reversible cerebral vasoconstriction syndrome
- primary sexual headache
- posterior reversible leucoencephalopathy syndrome
- acute hypertensive crisis

+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

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Question 94 of 409

An 18-year-old female attends the clinic complaining of speech difficulties and slowing of movements. On examination you note bilateral rigidity, akinesia with loss of arm swing, and bradykinesia.

What drug may have caused this disorder?

- ○ Sodium valproate
- ○ Lithium
- ○ Metoclopramide
- ○ Fluoxetine
- ○ Domperidone

Submit answer

Reference ranges

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An 18-year-old female attends the clinic complaining of speech difficulties and slowing of movements. On examination you note bilateral rigidity, akinesia with loss of arm swing, and bradykinesia.

What drug may have caused this disorder?

Sodium valproate	3%
Lithium	5%
Metoclopramide	79%
Fluoxetine	4%
Domperidone	9%

Metoclopramide can cause extrapyramidal effects, especially in children and young adults (15-19 years old)

Important for me   Less important

The clinical features are suggestive of Parkinsonism. Drug induced Parkinsonism often presents with bilateral feature (in contrast to idiopathic Parkinson's disease which often presents asymmetrically). It can be caused by several drugs including anti-psychotics, anti depressants, anti-emetics and calcium channel blockers.

Metoclopramide has a high risk of extrapyramidal effects, especially in children and young adults (15-19 years old), making this the best answer.

Several weak studies have shown an association of sodium valproate, lithium, domperidone and SSRIs to drug induced Parkinsonism, however the evidence is much weaker and they are less common causes compared to metoclopramide.

In many cases, the first approach to treatment will be to try stopping the offending drug for a sufficient length of time, reducing it, or changing it to another drug that may be less likely to cause drug-induced parkinsonism. If it is not possible to stop taking the offending drug, then anticholinergic drugs may be used, for example procyclidine.

Discuss (1) | Improve

Next question

Parkinsonism

Causes of Parkinsonism

- Parkinson's disease
- drug-induced e.g. antipsychotics, metoclopramide*
- progressive supranuclear palsy
- multiple system atrophy
- Wilson's disease
- post-encephalitis
- dementia pugilistica (secondary to chronic head trauma e.g. boxing)
- toxins: carbon monoxide, MPTP

*Domperidone does not cross the blood-brain barrier and therefore does not cause extra-pyramidal side-effects.

AI + Q 123

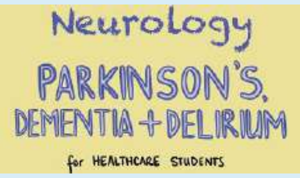
Next question

Textbooks

High-yield textbook

Extended textbook

Media


Neurology
PARKINSON'S
DEMENTIA + DELIRIUM
for HEALTHCARE STUDENTS

Parkinson's disease

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Question 95 of 409

A 74-year-old man is admitted to the acute medical unit with loss of consciousness. His wife states that she found him slumped in the arm chair in an unresponsive state approximately one hour ago. He had been feeling well before hand.

On examination you note bilateral 1mm pupils. His Glasgow coma score (GCS) is 4 (M2, E1, V1).

His past medical history includes hypertension, hypercholesterolaemia and osteoarthritis. His drug history includes codeine, paracetamol, ramipril and atorvastatin.

You administer 400 micrograms of naloxone. There is no change in GCS or pupil size.

What is the most likely diagnosis?

○ Pontine haemorrhage
○ Medulla haemorrhage
○ Midbrain haemorrhage
○ Subarachnoid haemorrhage
○ Opioid overdose

Submit answer

Reference ranges

Score: **20%**

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His past medical history includes hypertension, hypercholesterolaemia and osteoarthritis. His drug history includes codeine, paracetamol, ramipril and atorvastatin.

You administer 400 micrograms of naloxone. There is no change in GCS or pupil size.

What is the most likely diagnosis?

Pontine haemorrhage	73%
Medulla haemorrhage	5%
Midbrain haemorrhage	14%
Subarachnoid haemorrhage	4%
Opioid overdose	5%

Pontine haemorrhage commonly presents with reduced GCS, paralysis and bilateral pin point pupils

Important for me Less important

The patient has severely reduced GCS and is in a coma. The differential of coma is wide and includes vascular (e.g. intracerebral haemorrhage), inflammatory (e.g. cerebral vasculitis), neoplastic (e.g. paraneoplastic encephalitis, or cerebral space occupying lesion), drugs (e.g. sedatives), infective (e.g. meningitis, encephalitis), and metabolic (e.g. hyponatraemia, hypoglycaemia, hypercapnia) causes.

Pontine haemorrhage commonly presents with reduced GCS, paralysis and bilateral pin point pupils. Lesions of the pons can compress the first order sympathetic tracts resulting in bilateral Horner's syndrome.

The differential clearly includes opioid overdose, however there is nothing to suggest that an overdose has occurred, and the fact that his GCS and pupil size do not respond to naloxone further suggests that this is not the correct diagnosis.

Midbrain lesions typically cause fixed, midpoint pupils.

Lesions of the lateral medulla can result in an ipsilateral Horner's syndrome and a pin point pupil. A bilateral medulla lesion is a possible differential, however the sudden onset of coma and pin point pupils is more characteristic of pontine haemorrhage.

👍 👎 Discuss (4) Improve

Next question >

Stroke by anatomy ★

Site of the lesion	Associated effects
Anterior cerebral artery	Contralateral hemiparesis and sensory loss, lower extremity > upper
Middle cerebral artery	Contralateral hemiparesis and sensory loss, upper extremity > lower Contralateral homonymous hemianopia Aphasia
Posterior cerebral artery	Contralateral homonymous hemianopia with macular sparing Visual agnosia
Weber's syndrome (branches of the posterior cerebral artery that supply the midbrain)	Ipsilateral CN III palsy Contralateral weakness of upper and lower extremity
Posterior inferior cerebellar artery (lateral medullary syndrome, Wallenberg syndrome)	Ipsilateral: facial pain and temperature loss Contralateral: limb/torso pain and temperature loss Ataxia, nystagmus
Anterior inferior cerebellar artery (lateral pontine syndrome)	Symptoms are similar to Wallenberg's (see above), but: Ipsilateral: facial paralysis and deafness
Retinal/ophthalmic artery	Amaurosis fugax

Site of the lesion	Associated effects
Basilar artery	'Locked-in' syndrome

Lacunar strokes

- present with either isolated hemiparesis, hemisensory loss or hemiparesis with limb ataxia
- strong association with hypertension
- common sites include the basal ganglia, thalamus and internal capsule

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 75 10

Brainstem Rules of 4

Suggest link Report broken link

Media

Stroke

Podmedics - YouTube

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Brainstem Stroke

Brainstem stroke syndromes

Soton Brain Hub - YouTube 👍 13 👎 1

NEUROLOGY
MIDBRAIN SYNDROMES

Midbrain Syndromes - Weberâ€™s Syndrome, Benediktâ€™s Syndrome and Parinaud Syndrome

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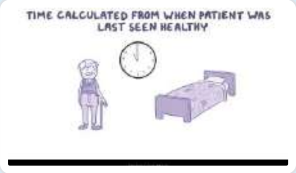
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Stroke

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A 28-year-old female is admitted after a paracetamol overdose. She took twenty 500mg tablets. She states that she wants to die and that life is not worth living any more. She had left a note for her parents. On mental state examination she has poverty of thought, a low affect and evidence of delusions of nihilism. She has a past medical history of severe depression. She is deemed to be medically fit for discharge however due to ongoing suicidal ideation she is transferred to a psychiatric unit for further assessment. During the admission she develops catatonia and stops eating and drinking.

Your team decides that electroconvulsive therapy is indicated. Which one of the following options is an absolute contraindication for electroconvulsive therapy?

- ○ Retinal detachment ×
- ○ Cardiac conduction disease ×
- ○ Malignant hypertension ×
- ○ Raised intracranial pressure ×
- ○ Myocardial infarction within the last 3 months ×

Submit answer

Reference ranges

Score: **20%**

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Your team decides that electroconvulsive therapy is indicated. Which one of the following options is an absolute contraindication for electroconvulsive therapy?

Retinal detachment	4%
Cardiac conduction disease	23%
Malignant hypertension	10%
Raised intracranial pressure	56%
Myocardial infarction within the last 3 months	7%

The only absolute contraindication for electroconvulsive therapy is raised intracranial pressure

Important for me Less important

With the exception of raised intracranial pressure, there are no absolute contraindications to ECT, although there are a number of clinical situations in which extra caution is required.

All the other options listed are relative contraindications.

ECT leads to increases in cerebral blood flow and increased intracranial pressure (ICP). This is the reason that raised ICP is an absolute contraindication.

Initially, the seizure activity causes a large parasympathetic discharge with bradycardia, atrial or ventricular premature beats, and occasional asystole. Therefore, cardiac conductive disease is a relative contraindication.

The initial parasympathetic stimulus is followed by sympathetic discharge causing tachycardia, hypertension, ST-segment depression and T-wave inversion not associated with myocardial enzyme changes. It can rarely cause ventricular tachycardia. Therefore, uncontrolled hypertension, and recent MI are relative contraindications.

Discuss (2) Improve

Next question

Electroconvulsive therapy

Electroconvulsive therapy is a useful treatment option for patients with severe depression refractory to medication (e.g. catatonia) those with psychotic symptoms. The only absolute contraindications is raised intracranial pressure.

Short-term side-effects

- headache
- nausea
- short term memory impairment
- memory loss of events prior to ECT
- cardiac arrhythmia

Long-term side-effects

- some patients report impaired memory

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Video patient uploaded of his ECT session

Individual - YouTube 👍 1 👎 1

Report broken media

Score: **20%**

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A 28-year-old female presents with a frontal headache with associated fever and photophobia. She states that she has been suffering from an ear infection over the past week.

Lumbar puncture results are as follows:

Cell count	228 (95% neutrophils)
Protein	1.2 g/l (normal 0.2-0.4)
Glucose	2.2 mmol/l

A plasma glucose is as follows:

Plasma glucose	6.8 mmol/l

What is the most likely organism?

- ○ ECHO virus
- ○ *Streptococcus pneumoniae*
- ○ *Neisseria meningitidis*
- ○ Listeria monocytogenes
- ○ Mycobacterium tuberculosis

Submit answer

Reference ranges

Score: **20%**

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Protein	1.2 g/l (normal 0.2-0.4)
Glucose	2.2 mmol/l

A plasma glucose is as follows:

Plasma glucose	6.8 mmol/l

What is the most likely organism?

ECHO virus	3%
Streptococcus pneumoniae	75%
Neisseria meningitidis	16%
Listeria monocytogenes	5%
Mycobacterium tuberculosis	2%

Meningitis which occurs as a complication of an ear infection is nearly always caused by *Streptococcus pneumoniae*

Important for me Less important

The patient has clinical features of meningitis. The CSF results confirm a neutrophilic pleocytosis with a high protein and low glucose. This is indicative of bacterial meningitis. *Streptococcus pneumoniae* and *Neisseria meningitidis* are high up the differential. Meningitis occurring as a complication of an ear infection is almost always caused by *Streptococcus pneumoniae*, making this the best answer.

ECHO virus is a cause of viral meningitis which presents with a lymphocytic pleocytosis and normal glucose, and normal / mildly raised protein.

Listeria monocytogenes meningitis tends to only affect elderly and immunosuppressed individuals.

Mycobacterium tuberculosis meningitis causes a lymphocytic pleocytosis and tends to present subacutely with a several week prodrome phase.

Discuss Improve

Next question ›

Meningitis in adults: aetiology

Meningitis may be caused by a variety of infective agents, including bacteria, viruses and fungi. This will focus on bacterial meningitis.

Common bacteria in adults
- *Streptococcus pneumoniae*
  - most common cause
  - may follow on from an episode of otitis media
- *Neisseria meningitidis*
- *Listeria monocytogenes*
  - more common in immunocompromised patients and the elderly
- *Haemophilus influenzae type b*

+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Score: **20%**

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Question 98 of 409

A 24-year-old woman presents with right eye vision loss and painful eye movements. She had a previous episode affecting her left eye last year. Following an MRI head and spine with contrast and a lumbar puncture examination, she is diagnosed with multiple sclerosis. She is started on high-dose steroids which induces remission.

You start the patient on maintenance ocrelizumab.

What is the mechanism of action of this drug?

- ○ Anti-TNF ×
- ○ Dihydrofolate reductase inhibitor ×
- ○ Humanized anti-CD20 monoclonal antibody ×
- ○ Recombinant monoclonal antibody that antagonises alpha-4 beta-1-integrin ×
- ○ Sphingosine 1-phosphate (S1P) receptor modulator ×

Submit answer

Reference ranges

Score: **20%**

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You start the patient on maintenance ocrelizumab.

What is the mechanism of action of this drug?

Anti-TNF	7%
Dihydrofolate reductase inhibitor	1%
Humanized anti-CD20 monoclonal antibody	51%
Recombinant monoclonal antibody that antagonises alpha-4 beta-1-integrin	33%
Sphingosine 1-phosphate (S1P) receptor modulator	7%

Ocrelizumab is considered a high-efficacy drug that is often used first-line to reduce the risk of relapse in MS

Important for me Less important

Humanized anti-CD20 monoclonal antibody is correct. Ocrelizumab selectively targets the CD20-positive B-cells by binding to the surface proteins of the cells and protecting the nerve cells from mediated damage caused by CD20-positive B lymphoma cell lines.

Anti-TNF is incorrect. Drugs in this class include adalimumab, infliximab, and etanercept.

Dihydrofolate reductase inhibitor is incorrect. This is the mechanism of action of methotrexate.

Recombinant monoclonal antibody that antagonises alpha-4 beta-1-integrin is incorrect. This is the mechanism of action of natalizumab.

Sphingosine 1-phosphate (S1P) receptor modulator is incorrect. This is the mechanism of action of fingolimod.

Discuss (3) Improve

Next question

Multiple sclerosis: management

Treatment in multiple sclerosis is focused on reducing the frequency and duration of relapses. There is no cure.

Acute relapse

High-dose steroids (e.g. oral or IV methylprednisolone) may be given for 5 days to shorten the length of an acute relapse. It should be noted that steroids shorten the duration of a relapse and do not alter the degree of recovery (i.e. whether a patient returns to baseline function)

Disease modifying drugs

A number of drugs have been shown to reduce the risk of relapse in patients with MS. Typical indications for disease-modifying drugs include:
- relapsing-remitting disease + 2 relapses in past 2 years + able to walk 100m unaided
- secondary progressive disease + 2 relapses in past 2 years + able to walk 10m (aided or unaided)

Drug options for reducing the risk of relapse in MS
- natalizumab
  - a recombinant monoclonal antibody that antagonises alpha-4 beta-1-integrin found on the surface of leucocytes
  - inhibit migration of leucocytes across the endothelium across the blood-brain barrier
  - generally considered to have the strongest evidence base for preventing relapse of the disease-modifying and hence is often used first-line
  - given intravenously
- ocrelizumab
  - humanized anti-CD20 monoclonal antibody
  - like natalizumab, it is considered a high-efficacy drug that is often used first-line
  - given intravenously
- fingolimod
  - sphingosine 1-phosphate (S1P) receptor modulator
  - prevents lymphocytes from leaving lymph nodes
  - oral formulations are available
- beta-interferon
  - not considered to be as effective as alternative disease-modifying drugs
  - given subcutaneously/intramuscularly

- glatiramer acetate
    - immunomodulating drug - acts as an 'immune decoy'
    - given subcutaneously
    - along with beta-interferon considered an 'older drug' with less effectiveness compared to monoclonal antibodies and S1P) receptor modulators

Some specific problems

Fatigue
- once other problems (e.g. anaemia, thyroid or depression) have been excluded NICE recommend a trial of amantadine
- other options include mindfulness training and CBT

Spasticity
- baclofen and gabapentin are first-line. Other options include diazepam, dantrolene and tizanidine
- physiotherapy is important
- cannabis and botox are undergoing evaluation

Bladder dysfunction
- may take the form of urgency, incontinence, overflow etc
- guidelines stress the importance of getting an ultrasound first to assess bladder emptying - anticholinergics may worsen symptoms in some patients
- if significant residual volume → intermittent self-catheterisation
- if no significant residual volume → anticholinergics may improve urinary frequency

Oscillopsia (visual fields appear to oscillate)
- gabapentin is first-line

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Multiple Sclerosis Society 👍 6 👎 14

Primary care guidelines

NICE 👍 15 👎 7

2014 Multiple Sclerosis guidelines

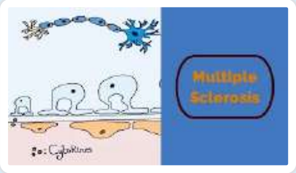
Suggest link

Report broken link

Media

Multiple sclerosis

Podmedics - YouTube 👍 4 👎 0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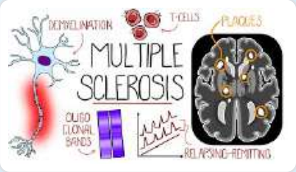
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RELAPSING-REMITTING

Multiple sclerosis

Rhesus Medicine - YouTube 👍 1 👎 0

MULTIPLE
SCLEROSIS

Multiple sclerosis - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 7 👎 1

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Question 99 of 409

A 56-year-old man comes to the office due to blurred vision and drooping of his left eyelid for over two weeks. He was diagnosed with hypertension and hyperlipidaemia 2 years ago, for which he takes lisinopril and atorvastatin. The patient has smoked a pack of cigarettes daily for the past 30 years.

Vital signs are within normal limits. Physical examination shows normal pupils and eye movements. After closing the eyes for 5 minutes, the drooping resolves. However, after a few minutes, the drooping recurs, worsening progressively. His cardiovascular, respiratory and neurological examinations are normal.

Which of the following is the best next step in the management of this patient?

○ Botulinum antitoxin ×

○ Intravenous immunoglobulin ×

○ Prednisone ×

○ Pyridostigmine ×

○ Smoking cessation ×

Submit answer

Reference ranges

Score: **20%**

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A 56-year-old man comes to the office due to blurred vision and drooping of his left eyelid for over two weeks. He was diagnosed with hypertension and hyperlipidaemia 2 years ago, for which he takes lisinopril and atorvastatin. The patient has smoked a pack of cigarettes daily for the past 30 years.

Vital signs are within normal limits. Physical examination shows normal pupils and eye movements. After closing the eyes for 5 minutes, the drooping resolves. However, after a few minutes, the drooping recurs, worsening progressively. His cardiovascular, respiratory and neurological examinations are normal.

Which of the following is the best next step in the management of this patient?

Botulinum antitoxin	4%
Intravenous immunoglobulin	6%
Prednisone	5%
Pyridostigmine	77%
Smoking cessation	8%

Pyridostigmine is the 1st line drug in the management of ocular myasthenia gravis

Important for me Less important

This patient with ptosis and eyelid fatigue has ocular myasthenia gravis. Ocular myasthenia gravis presents with ptosis and oculomotor paresis. The ptosis is often unilateral and may alternate from one side to another. Eyelid fatigability is another classic sign of ocular myasthenia gravis. In myasthenia gravis, autoantibodies against acetylcholine receptors prevent acetylcholine from properly binding to these receptors. Furthermore, acetylcholine is quickly broken down into choline and acetic acid by the cholinesterase enzyme at the neuromuscular junction. Pyridostigmine is a cholinesterase inhibitor. It slows down the breakdown of acetylcholine and is the first-line drug in the management of symptomatic myasthenia, like ocular myasthenia gravis.

Patients with botulism present with acute symmetric descending flaccid paralysis which progresses over time. This patient's presentation is not progressing, and is therefore, unlikely to be botulism.

IV immunoglobulins and plasmapheresis are used in the management of Guillain-Barre syndrome. They don't have a role in treating ocular myasthenia gravis.

The use of prednisone and other corticosteroids is controversial in ocular myasthenia gravis. Corticosteroids are associated with severe adverse effects and difficulty weaning off. Moreover, several weeks to months of corticosteroid therapy are required to maintain beneficial effects in patients with ocular myasthenia gravis. Anticholinesterase agents, like pyridostigmine, should be used before resorting to corticosteroids.

While this patient should consider smoking cessation, quitting smoking is unlikely to improve symptoms of ocular myasthenia gravis.

👍 👎 Discuss (3) Improve

Next question >

Myasthenia gravis

Myasthenia gravis is an autoimmune disorder resulting in insufficient functioning acetylcholine receptors. Antibodies to acetylcholine receptors are seen in 85-90% of cases*. Myasthenia is more common in women (2:1)

The key feature is muscle fatigability - muscles become progressively weaker during periods of activity and slowly improve after periods of rest:
- extraocular muscle weakness: diplopia
- proximal muscle weakness: face, neck, limb girdle
- ptosis
- dysphagia

Associations
- thymomas in 15%
- autoimmune disorders: pernicious anaemia, autoimmune thyroid disorders, rheumatoid, SLE
- thymic hyperplasia in 50-70%

Investigations
- single fibre electromyography: high sensitivity (92-100%)
- CT thorax to exclude thymoma
- CK normal
- antibodies to acetylcholine receptors
    - positive in around 85-90% of patients
    - n the remaining patients, about about 40% are positive for anti-muscle-specific tyrosine kinase antibodies

- Tensilon test: IV edrophonium reduces muscle weakness temporarily - not commonly used any more due to the risk of cardiac arrhythmia

Management

- long-acting acetylcholinesterase inhibitors
    - pyridostigmine is first-line
- immunosuppression is usually not started at diagnosis, but the majority of patients eventually require it in addition to long-acting acetylcholinesterase inhibitors:
    - prednisolone initially
    - azathioprine, cyclosporine, mycophenolate mofetil may also be used
- thymectomy

Management of myasthenic crisis

- plasmapheresis
- intravenous immunoglobulins

*antibodies are less commonly seen in disease limited to the ocular muscles

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

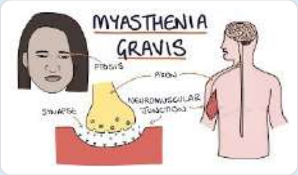
Postgraduate Medical Journal 👍 13 👎 3

Review of myasthenia gravis

Suggest link

Report broken link

Media


MYASTHENIA GRAVIS
PTOSIS
AXON
NEUROMUSCULAR JUNCTION
SYNAPSE

Myasthenia gravis

Zero to Finals - YouTube 👍 12 👎 0

NEUROLOGY
MYASTHENIA GRAVIS

Myasthenia gravis

Armando Hasudungan - YouTube 👍 1 👎 1

MYASTHENIA GRAVIS

Myasthenia gravis - causes, symptoms, treatment, pathology

Osmosis - YouTube 👍 2 👎 2

Report broken media

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Question 100 of 409

A 23-year-old woman presents to the emergency department having been found by her father in a reduced state of consciousness one hour ago. She has a past medical history of epilepsy for which she is taking levetiracetam and anxiety and depression for which she has been prescribed short courses of diazepam in the past. Several empty packets of diazepam were found by her bedside.

On initial assessment, her oxygen saturations are 91% on 15L via a non-rebreather. Her heart rate is 61/min and her blood pressure is 91/64mmHg. She is responsive to pain only.

What is the most appropriate next step in management?

○ Activated charcoal

○ Anaesthetic support

○ Haemofiltration

○ Intravenous flumazenil

○ Naloxone

Submit answer

Reference ranges

Score: **20%**

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A 23-year-old woman presents to the emergency department having been found by her father in a reduced state of consciousness one hour ago. She has a past medical history of epilepsy for which she is taking levetiracetam and anxiety and depression for which she has been prescribed short courses of diazepam in the past. Several empty packets of diazepam were found by her bedside.

On initial assessment, her oxygen saturations are 91% on 15L via a non-rebreather. Her heart rate is 61/min and her blood pressure is 91/64mmHg. She is responsive to pain only.

What is the most appropriate next step in management?

Activated charcoal	8%
Anaesthetic support	50%
Haemofiltration	2%
Intravenous flumazenil	37%
Naloxone	3%

Benzodiazepine overdose is best managed supportively and with airway protection and ventilation if needed. Flumazenil should be avoided unless for reversal of anaesthesia

Important for me Less important

The primary concern here is related to this woman's airway. Despite 15L of oxygen via a non-rebreather, her oxygen saturations are only holding at 91%. Airway obstruction is likely a result of her reduced conscious level. As per the ALS algorithm, airway manoeuvres and **anaesthetic support** for potential intubation and ventilation should be sought.

Activated charcoal may be helpful in acute ingested poisonings to limit subsequent serum levels however this does not address the most urgent concern here which is airway obstruction and is therefore not the most appropriate first step.

Haemofiltration can be used to clear various drugs however again this will take some time to have an effect and does not address the most pressing concern, airway obstruction. Therefore this is not the correct answer.

Intravenous flumazenil is the primary reversal agent for benzodiazepine overdose however has to be used with extreme caution as its use can cause intractable seizures and arrhythmias. As this woman is young and particularly as she has a history of epilepsy, airway support and ventilation if required would be a more favoured approach in this case.

Naloxone is an agent used to reverse opioid overdose however as there is no indication of opioid use here, this is unlikely to reverse this woman's condition.

Discuss (2) | Improve

Next question >

Status epilepticus

Status epilepticus is defined as:
- a single seizure lasting >5 minutes, or
- >= 2 seizures within a 5-minute period without the person returning to normal between them

This is a medical emergency. The priority is the termination of seizure activity, which if prolonged will lead to irreversible brain damage.

Management
- ABC
  - airway adjunct
  - oxygen
  - check blood glucose
- First-line drugs are benzodiazepines
  - in the prehospital setting PR diazepam or buccal midazolam may be given
  - in hospital IV lorazepam is generally used. This may be repeated once after 5-10minutes
- If ongoing (or 'established') status it is appropriate to start a second-line agent such as levetiracetam, phenytoin or sodium valproate
  - NICE state 'Take into account that levetiracetam may be quicker to administer and have fewer adverse effects than the alternative options.'
- If no response ('refractory status') within 45 minutes from onset, then the best way to achieve rapid control of seizure activity is induction of general anaesthesia or phenobarbital

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE

2022 Epilepsy guidelines

👍 14 👎 9

Suggest link

Report broken link

Score: **20%**

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Question 101 of 409

A 27-year-old woman comes for review. She is having problems with increasingly frequent migraine attacks. These occur throughout her menstrual cycle and have no relation to her periods. She has tried a combination of paracetamol and ibuprofen to try and control the attacks but this seems to have had a limited effect. Her current medication includes paracetamol and ibuprofen as required and Cerazette (a progestogen-only pill).

What is the most appropriate medication to try and reduce the frequency of her migraine attacks?

- ○ Propranolol
- ○ Zolmitriptan
- ○ Topiramate
- ○ Amitriptyline
- ○ Switch Cerazette to a combined oral contraceptive pill

Submit answer

Reference ranges

Score: **20%**

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A 27-year-old woman comes for review. She is having problems with increasingly frequent migraine attacks. These occur throughout her menstrual cycle and have no relation to her periods. She has tried a combination of paracetamol and ibuprofen to try and control the attacks but this seems to have had a limited effect. Her current medication includes paracetamol and ibuprofen as required and Cerazette (a progestogen-only pill).

What is the most appropriate medication to try and reduce the frequency of her migraine attacks?

Propranolol	70%
Zolmitriptan	15%
Topiramate	7%
Amitriptyline	2%
Switch Cerazette to a combined oral contraceptive pill	6%

Propranolol is preferable to topiramate in women of childbearing age (i.e. the majority of women with migraine)

Important for me Less important

Zolmitriptan is useful to abort attacks but is not generally used for prophylaxis, except in the case of menstrual-related migraine (see below for more details). From the history it is clear that the headaches are not confined to the days around menstruation.

NICE recommend either propranolol or topiramate for migraine prophylaxis. Propranolol should be used in preference to topiramate in women of child bearing age as it may be teratogenic and it can reduce the effectiveness of hormonal contraceptives

The combined oral contraceptive pill is contraindicated given her history of migraine.

Discuss (6) Improve

Next question

Migraine: management

It should be noted that as a general rule 5-HT receptor agonists are used in the acute treatment of migraine whilst 5-HT receptor antagonists are used in prophylaxis. NICE produced guidelines in 2012 on the management of headache, including migraines.

Acute treatment

- first-line: offer combination therapy with
  - an oral triptan and an NSAID, or
  - an oral triptan and paracetamol
- for young people aged 12-17 years consider a nasal triptan in preference to an oral triptan
- if the above measures are not effective or not tolerated offer a non-oral preparation of metoclopramide or prochlorperazine and consider adding a non-oral NSAID or triptan
  - caution should be exercised when prescribing metoclopramide to young patients as acute dystonic reactions may develop

Prophylaxis

- prophylaxis should generally be given if '*Migraine attacks are having a significant impact on quality of life and daily function, for example they occur frequently (more than once a week on average) or are prolonged and severe despite optimal acute treatment*'
- options:
  - propranolol
  - topiramate: should be avoided in women of childbearing age as it may be teratogenic and it can reduce the effectiveness of hormonal contraceptives
  - amitriptyline
- if these measures fail NICE recommends 'a course of up to 10 sessions of acupuncture over 5-8 weeks'
- NICE recommend: '*Advise people with migraine that riboflavin (400 mg once a day) may be effective in reducing migraine frequency and intensity for some people*'
- for women with predictable menstrual migraine treatment NICE recommend either frovatriptan (2.5 mg twice a day) or zolmitriptan (2.5 mg twice or three times a day) as a type of 'mini-prophylaxis'
- treatment options that may be considered by specialists, but fall outside the NICE guidelines:
  - candesartan: recommended by the British Association for the Study of Headache guidelines
  - monoclonal antibodies directed against the calcitonin gene-related peptide (CGRP) receptor: examples include erenumab
- pizotifen is no longer recommended. Adverse effects such as weight gain & drowsiness are common

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Association for the Study of Headache 👍 14 👎 21

Headache guidelines

Clinical Knowledge Summaries 👍 14 👎 7

Migraine guidelines

NICE 👍 16 👎 4

2012 Headache guidelines

Suggest link

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Media

Management of Migraine
Podmedics

Migraine

Podmedics - YouTube 👍 1 👎 1

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A 34-year-old woman attends with weakness in her left leg. The symptoms started over a few hours and have persisted for 2 days.

On examination of the left leg, she has a brisk ankle jerk and an upgoing plantar, dorsiflexion is reduced in power, and tone is normal. Examination of the right leg is unremarkable.

Further questioning reveals an episode of numbness and tingling in her right hand 3 months ago.

Given the likely diagnosis, what treatment is most likely to reduce relapses for this patient?

- ○ Dexamethasone ×
- ○ Fingolimod ×
- ○ Glatiramer ×
- ○ Interferon beta ×
- ○ Natalizumab ×

Submit answer

Reference ranges

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A 34-year-old woman attends with weakness in her left leg. The symptoms started over a few hours and have persisted for 2 days.

On examination of the left leg, she has a brisk ankle jerk and an upgoing plantar, dorsiflexion is reduced in power, and tone is normal. Examination of the right leg is unremarkable.

Further questioning reveals an episode of numbness and tingling in her right hand 3 months ago.

Given the likely diagnosis, what treatment is most likely to reduce relapses for this patient?

Dexamethasone	4%
Fingolimod	8%
Glatiramer	7%
Interferon beta	19%
Natalizumab ✓	62%

Monoclonal antibodies such as natalizumab have the strongest evidence base for reducing relapse in multiple sclerosis

Important for me   Less important

The most likely diagnosis here is multiple sclerosis given the upper motor neuron signs separated by time. MR imaging of the brain and spinal cord would be appropriate to confirm the diagnosis. Be aware that, during the acute phase, tone may be normal.

Natalizumab is the correct answer. Natalizumab is a high-efficacy disease-modifying therapy and has the strongest evidence base for reduced relapse rates.

Dexamethasone can be used in acute relapsing disease, but it does not confer any long-term benefit in reducing further relapses.

Fingolimod is a SIP1 modulator. It can be used as a disease-modifying treatment in multiple sclerosis but has less supporting evidence for reducing relapses compared to natalizumab.

Interferon beta is another disease-modifying drug used in the treatment of multiple sclerosis. It is what is termed a 'low-risk' therapy as it has been around for quite a long while and has a good safety profile. Some clinicians favour starting with a 'low-risk' therapy and escalating to 'high efficacy' treatments if relapses aren't controlled. The evidence, however, does state that natalizumab has better evidence for controlling relapse rates overall.

Glatiramer is also termed a 'low-risk' therapy due to its good safety profile. Again, the evidence for a reduction in relapse rate is not as strong as that for natalizumab.

👍 👎 Discuss (2) Improve

Next question >

Multiple sclerosis: management ★

Treatment in multiple sclerosis is focused on reducing the frequency and duration of relapses. There is no cure.

Acute relapse

High-dose steroids (e.g. oral or IV methylprednisolone) may be given for 5 days to shorten the length of an acute relapse. It should be noted that steroids shorten the duration of a relapse and do not alter the degree of recovery (i.e. whether a patient returns to baseline function)

Disease modifying drugs

A number of drugs have been shown to reduce the risk of relapse in patients with MS. Typical indications for disease-modifying drugs include:
- relapsing-remitting disease + 2 relapses in past 2 years + able to walk 100m unaided
- secondary progressive disease + 2 relapses in past 2 years + able to walk 10m (aided or unaided)

Drug options for reducing the risk of relapse in MS
- natalizumab
  - a recombinant monoclonal antibody that antagonises alpha-4 beta-1-integrin found on the surface of leucocytes
  - inhibit migration of leucocytes across the endothelium across the blood-brain barrier
  - generally considered to have the strongest evidence base for preventing relapse of the disease-modifying and hence is often used first-line
  - given intravenously
- ocrelizumab
  - humanized anti-CD20 monoclonal antibody
  - like natalizumab, it is considered a high-efficacy drug that is often used first-line
  - given intravenously

- fingolimod
    - sphingosine 1-phosphate (S1P) receptor modulator
    - prevents lymphocytes from leaving lymph nodes
    - oral formulations are available
- beta-interferon
    - not considered to be as effective as alternative disease-modifying drugs
    - given subcutaneously/intramuscularly
- glatiramer acetate
    - immunomodulating drug - acts as an 'immune decoy'
    - given subcutaneously
    - along with beta-interferon considered an 'older drug' with less effectiveness compared to monoclonal antibodies and S1P) receptor modulators

Some specific problems

Fatigue

- once other problems (e.g. anaemia, thyroid or depression) have been excluded NICE recommend a trial of amantadine
- other options include mindfulness training and CBT

Spasticity

- baclofen and gabapentin are first-line. Other options include diazepam, dantrolene and tizanidine
- physiotherapy is important
- cannabis and botox are undergoing evaluation

Bladder dysfunction

- may take the form of urgency, incontinence, overflow etc
- guidelines stress the importance of getting an ultrasound first to assess bladder emptying - anticholinergics may worsen symptoms in some patients
- if significant residual volume → intermittent self-catheterisation
- if no significant residual volume → anticholinergics may improve urinary frequency

Oscillopsia (visual fields appear to oscillate)

- gabapentin is first-line

Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Multiple Sclerosis Society 👍 6 👎 14

Primary care guidelines

NICE 👍 15 👎 7

2014 Multiple Sclerosis guidelines

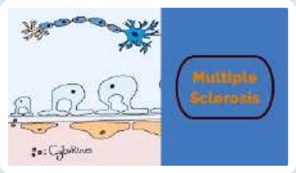
Suggest link

Report broken link

Media

Multiple sclerosis

Podmedics - YouTube 👍 4 👎 0


Multiple Sclerosis

Multiple sclerosis

Soton Brain Hub - YouTube 👍 3 👎 0

DEMYELINATION
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PLAQUES
MULTIPLE SCLEROSIS
OLIGO CLONAL BANDS
RELAPSING-REMITTING

Multiple sclerosis

Rhesus Medicine - YouTube 👍 1 👎 0

MULTIPLE
SCLEROSIS

Multiple sclerosis - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 7 👎 1

Report broken media

Score: **20%**

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Question 103 of 409

A 28-year-old man presents to his general practitioner with peripheral vision loss. He explains that his vision has gradually 'narrowed' over the last 2 months. He also describes difficulty seeing in the dark, which has preceded the current symptoms by several years, although he has not sought medical attention for this.

On examination, fundoscopy reveals black bone spicule-shaped pigmentation in the peripheral retina. Blood tests are also performed:

Na^+	138 mmol/L	(135 - 145)
K^+	3.3 mmol/L	(3.5 - 5.0)
Bicarbonate	29 mmol/L	(22 - 29)
Urea	9.1 mmol/L	(2.0 - 7.0)
Creatinine	320 µmol/L	(55 - 120)

What is the most likely cause of this patient's abnormal blood tests?

- ○ Alport syndrome
- ○ Autosomal dominant polycystic kidney disease (ADPKD)
- ○ Autosomal recessive polycystic kidney disease (ARPKD)
- ○ Bartter syndrome
- ○ Renal artery stenosis

Submit answer

Reference ranges

Score: **20%**

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Urea	9.1 mmol/L	(2.0 - 7.0)
Creatinine	320 µmol/L	(55 - 120)

What is the most likely cause of this patient's abnormal blood tests?

Alport syndrome	62%
Autosomal dominant polycystic kidney disease (ADPKD)	15%
Autosomal recessive polycystic kidney disease (ARPKD)	7%
Bartter syndrome	10%
Renal artery stenosis	6%

Retinitis pigmentosa classically presents with black bone-spicule pigmentation on fundoscopy

Important for me Less important

Tunnel vision, night blindness, and fundoscopy findings in this patient are characteristic of retinitis pigmentosa. The presence of renal disease with retinitis pigmentosa hints at **Alport syndrome** , which is due to an abnormality in the gene that codes for type 4 collagen. Sensorineural hearing loss, although not featured in this scenario, is another prominent feature of Alport syndrome.

The common disease associations of **autosomal dominant polycystic kidney disease (ADPKD)** are cerebral aneurysms, hepatic cysts, colonic diverticula, mitral regurgitation and mitral prolapse.

Retinitis pigmentosa is not associated with ADPKD.

Patients with **autosomal recessive polycystic kidney disease (ARPKD)** rarely survive beyond 5 years of age. ARPKD is not associated with retinitis pigmentosa.

Although mild hypokalemia and alkalosis are present in this scenario, this is less likely to be due to **Bartter syndrome** , which is extremely rare. Furthermore, there is no association between Bartter syndrome and retinitis pigmentosa.

Hypokalemic metabolic alkalosis may be seen with **renal artery stenosis** (RAS) due to the stimulation of the renin-angiotensin-aldosterone system. The normal blood pressure and presence of retinitis pigmentosa make Alport syndrome more likely.

👍 👎 Discuss (2) Improve

Next question >

Retinitis pigmentosa ★

Retinitis pigmentosa primarily affects the peripheral retina resulting in tunnel vision

Features
- night blindness is often the initial sign
- tunnel vision due to loss of the peripheral retina (occasionally referred to as funnel vision)
- fundoscopy: black bone spicule-shaped pigmentation in the peripheral retina, mottling of the retinal pigment epithelium

Associated diseases
- Refsum disease: cerebellar ataxia, peripheral neuropathy, deafness, ichthyosis
- Usher syndrome
- abetalipoproteinemia
- Lawrence-Moon-Biedl syndrome
- Kearns-Sayre syndrome
- Alport's syndrome

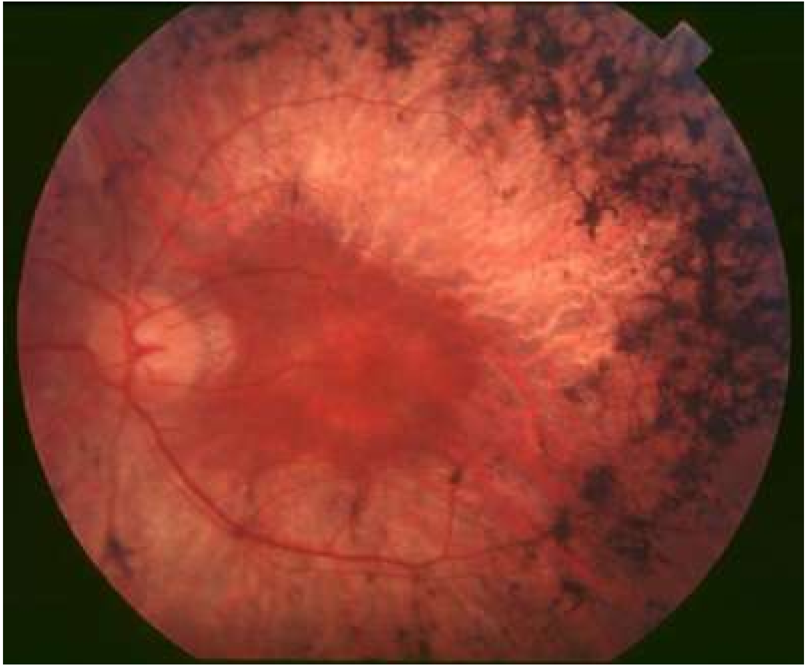

Image sourced from Wikipedia

Fundus showing changes secondary to retinitis pigmentosa

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20%**

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Question 104 of 409

A 42-year-old woman is referred into the TIA clinic due to recurrent left arm weakness. She has been having recurrent episodes of left sided weakness with associated unilateral throbbing headache, nausea and photophobia. During each episode she find that her left arm becomes weak. She has a past medical history of headaches, DVT during pregnancy and hypothyroidism. Currently there are no neurological features. What is the most likely diagnosis?

- ○ TIA
- ○ Subdural haematoma
- ○ Sinus venous thrombosis
- ○ Brain tumour
- ○ Migraines

Submit answer

Reference ranges

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A 42-year-old woman is referred into the TIA clinic due to recurrent left arm weakness. She has been having recurrent episodes of left sided weakness with associated unilateral throbbing headache, nausea and photophobia. During each episode she find that her left arm becomes weak. She has a past medical history of headaches, DVT during pregnancy and hypothyroidism. Currently there are no neurological features. What is the most likely diagnosis?

TIA	3%
Subdural haematoma	0%
Sinus venous thrombosis	20%
Brain tumour	1%
Migraines	76%

Recurrence of the same neurological signs or symptoms is unlikely to be due to TIA

Important for me Less important

This patient has recurrent weakness as a feature of neurology with headache and photophobia. The most likely diagnosis is migraines which can cause weakness. The recurrence of symptoms makes TIA less likely. This is because it is unlikely for clots to occur in the same vessel and cause the same symptoms. Subdural haematoma is unlikely to cause photophobia, and also unlikely to cause intermittent weakness. Brain tumours are associated with progressive and unrelenting headaches as well as mass effect symptoms.

Discuss (4) Improve

Next question

Transient ischaemic attack

A transient ischaemic attack (TIA), as the name suggests, is a brief period of neurological deficit due to a vascular cause, typically lasting less than an hour. The National Clinical Guideline for Stroke was published in 2023 and made a number of updated recommendations, including the use of dual antiplatelet therapy (DAPT). It should be noted that DAPT is not currently recommended for 'major' ischaemic stroke as the risk of haemorrhagic transformation is too high.

The original definition of a TIA was time-based: a sudden onset of a focal neurologic symptom and/or sign lasting less than 24 hours, brought on by a transient decrease in blood flow. However, this has now changed as it is recognised that even short periods of ischaemia can result in pathological changes to the brain. Therefore, a new **'tissue-based'** definition is now used: a transient episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischaemia, without acute infarction. REF

Patients often use the term 'mini-stroke' for TIAs.

Clinical features

The clinical features are similar to those of a stroke, i.e. sudden onset, focal neurological deficit but, rather than persisting, the features resolve, typically within 1 hour.

Possible features include
- unilateral weakness or sensory loss.
- aphasia or dysarthria
- ataxia, vertigo, or loss of balance
- visual problems
    - sudden transient loss of vision in one eye (amaurosis fugax)
    - diplopia
    - homonymous hemianopia

Assessment and referral

Patients with acute focal neurological symptoms that resolve completely within 24 hours of onset (i.e. suspected TIA) should:
- be given aspirin 300 mg immediately unless contraindicated
- assessed urgently within 24 hours by a stroke specialist clinician

Examples of TIA mimics that require exclusion
- hypoglycaemia
- intracranial haemorrhage
    - all patients on anticoagulants or with similar risk factors should be admitted for urgent imaging to exclude haemorrhage

If a patient presents more than 7 days ago they should be seen by a stroke specialist clinician as soon as possible within 7 days.

The ABCD2 prognostic score has previously been used to risk stratify patients who present with a suspected TIA. However, data from studies have suggested it performs poorly and it is therefore no longer recommended.

Patients with suspected TIA should be assessed by a stroke specialist clinician before a decision on brain imaging NICE

- NICE recommend that CT brains should not be done '*unless there is clinical suspicion of an alternative diagnosis that CT could detect*'
    - an example exception would be when there is a concern about haemorrhage as the patient is taking anticoagulants
- MRI (including diffusion-weighted and blood-sensitive sequences) is preferred to determine the territory of ischaemia, or to detect haemorrhage or alternative pathologies
    - it should be done on the same day as the specialist assessment if possible

Management of TIA

It should be remembered that patients who've had a TIA are at high risk of further vascular events, particularly in the first few days. This section covers management following a diagnosis, i.e. after being seen by a stroke specialist clinician.

Medication

Immediate antithrombotic therapy: REF

- patients with TIA or minor ischaemic stroke should be given antiplatelet therapy provided there is neither a contraindication nor a high risk of bleeding
- for patients within 24 hours of onset of TIA or minor ischaemic stroke and with a low risk of bleeding, the following DAPT regimes should be considered:
    - clopidogrel (initial dose 300 mg followed by 75 mg od) + aspirin (initial dose 300 mg followed by 75 mg od for 21 days) followed by monotherapy with clopidogrel 75 mg od
    - ticagrelor + clopidogrel is an alternative
- if not appropriate for DAPT:
    - clopidogrel 300 mg loading dose followed by 75 mg od should be given
- proton pump inhibitor therapy should be considered for DAPT

The different antithrombotic recommendations may seem confusing - it is helpful to think of 3 different phases for a typical patient:

Resolved TIA symptoms, awaiting specialist review within 24 hours	**Reviewed by specialist, initial 21 days when at high risk of further events**	**Long-term secondary prevention after 21 days**
Aspirin	Aspirin + Clopidogrel	Clopidogrel

If a patient has atrial fibrillation they should be anticoagulated as soon as intracranial haemorrhage has been excluded.

Lipid modification

- high-intensity statin (such as atorvastatin 20-80 mg daily) - the aim of statin therapy is to reduce non-HDL cholesterol by more than 40%

Further investigation

Carotid imaging

- atherosclerosis in the carotid artery may be a source of emboli in some patients
- patients who are considered candidates for carotid intervention should have carotid imaging performed within 24 hours of assessment
    - carotid duplex ultrasound or either CT angiography or MR angiography
- carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled NICE
    - should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

+ it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

RCP 13 13

2016 Stroke guidelines

National Clinical Guideline for Stroke 👍 7 👎 3

2023 National Clinical Guideline for Stroke

NICE 👍 17 👎 6

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Clinical Knowledge Summaries 👍 8 👎 3

Transient ischaemic attack

Radiopaedia 👍 7 👎 1

Transient ischaemic attack

Suggest link

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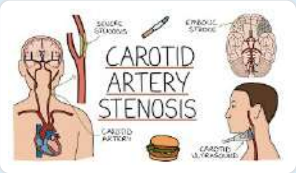
Media



Understanding Carotid Artery Stenosis

Zero To Finals - YouTube 👍 2 👎 1

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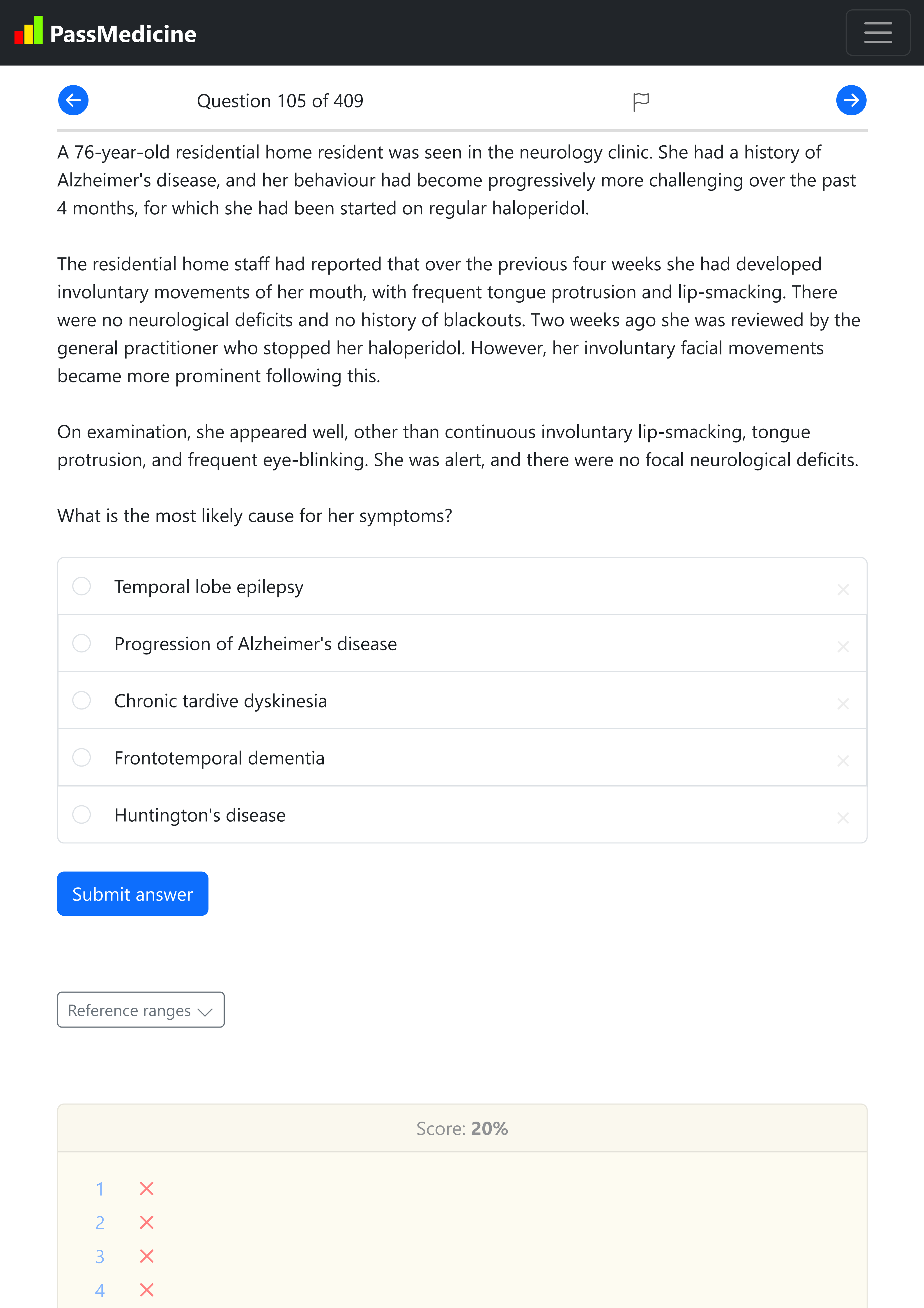

Question 105 of 409

A 76-year-old residential home resident was seen in the neurology clinic. She had a history of Alzheimer's disease, and her behaviour had become progressively more challenging over the past 4 months, for which she had been started on regular haloperidol.

The residential home staff had reported that over the previous four weeks she had developed involuntary movements of her mouth, with frequent tongue protrusion and lip-smacking. There were no neurological deficits and no history of blackouts. Two weeks ago she was reviewed by the general practitioner who stopped her haloperidol. However, her involuntary facial movements became more prominent following this.

On examination, she appeared well, other than continuous involuntary lip-smacking, tongue protrusion, and frequent eye-blinking. She was alert, and there were no focal neurological deficits.

What is the most likely cause for her symptoms?

- ○ Temporal lobe epilepsy
- ○ Progression of Alzheimer's disease
- ○ Chronic tardive dyskinesia
- ○ Frontotemporal dementia
- ○ Huntington's disease

Score: **20%**

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Question 105 of 409

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The residential home staff had reported that over the previous four weeks she had developed involuntary movements of her mouth, with frequent tongue protrusion and lip-smacking. There were no neurological deficits and no history of blackouts. Two weeks ago she was reviewed by the general practitioner who stopped her haloperidol. However, her involuntary facial movements became more prominent following this.

On examination, she appeared well, other than continuous involuntary lip-smacking, tongue protrusion, and frequent eye-blinking. She was alert, and there were no focal neurological deficits.

What is the most likely cause for her symptoms?

Temporal lobe epilepsy	20%
Progression of Alzheimer's disease	2%
Chronic tardive dyskinesia	72%
Frontotemporal dementia	4%
Huntington's disease	2%

Antipsychotics may cause tardive dyskinesia

Important for me   Less important

First-generation antipsychotic agents (e.g. haloperidol, chlorpromazine) may result in extrapyramidal side effects due to relatively non-selective dopamine blockade. This may manifest as chronic tardive dyskinesia, as in this case. Clinical features of chronic tardive dyskinesia include involuntary repetitive movements, such as lip-smacking, tongue protrusion, and distal chorea. It is treated by withdrawal of the offending agent, though it may paradoxically worsen in the first few weeks of stopping the drug. The typical features and temporal relationship make chronic tardive dyskinesia secondary to haloperidol the most likely diagnosis in this case. Other extrapyramidal side effects include acute dystonic reactions, akathisia, and Parkinsonism.

Lip-smacking may occur in temporal lobe epilepsy, but this would be associated with impaired consciousness and would only last a few minutes. Advanced Alzheimer's disease may be associated with involuntary movements (e.g. myoclonus), but is not typically associated with dyskinesias. Parkinsonism may occur in frontotemporal dementia, but dyskinesia is not associated. A new diagnosis of Huntington's disease would be very unlikely in this age group.

👍 👎 Discuss (4) Improve

Next question >

Antipsychotics ★

Antipsychotics are a group of drugs used in the management of schizophrenia and other forms of psychosis, mania and agitation. They are usually divided into typical and atypical antipsychotics. The atypical antipsychotics were developed due to the problematic extrapyramidal side-effects which are associated with the first generation of typical antipsychotics.

	Typical antipsychotics	Atypical antipsychotics
Mechanism of action	Dopamine D2 receptor antagonists, blocking dopaminergic transmission in the mesolimbic pathways	Act on a variety of receptors (D2, D3, D4, 5-HT)
Adverse effects	Extrapyramidal side-effects and hyperprolactinaemia common	Extrapyramidal side-effects and hyperprolactinaemia less common Metabolic effects
Examples	Haloperidol Chlorpromazine	Clozapine Risperidone Olanzapine

The rest of this section will focus on typical antipsychotics, with atypical antipsychotics covered elsewhere.

Extrapyramidal side-effects (EPSEs)

- Parkinsonism
- acute dystonia
  - sustained muscle contraction (e.g. torticollis, oculogyric crisis)
  - may be managed with procyclidine
- akathisia (severe restlessness)
- tardive dyskinesia (late onset of choreoathetoid movements, abnormal, involuntary, may occur in 40% of patients, may be irreversible, most common is chewing and pouting of jaw)

The Medicines and Healthcare products Regulatory Agency has issued specific warnings when antipsychotics are used in elderly patients:

- increased risk of stroke
- increased risk of venous thromboembolism

Other side-effects

- antimuscarinic: dry mouth, blurred vision, urinary retention, constipation
- sedation, weight gain
- raised prolactin
  - may result in galactorrhoea
  - due to inhibition of the dopaminergic tuberoinfundibular pathway
- impaired glucose tolerance
- neuroleptic malignant syndrome: pyrexia, muscle stiffness
- reduced seizure threshold (greater with atypicals)
- prolonged QT interval (particularly haloperidol)

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 1 👎 0

Schizophrenia > Prescribing Information > Adverse effects

BNF 👍 1 👎 2

Psychoses and related disorders

Suggest link

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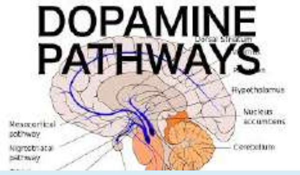
Media

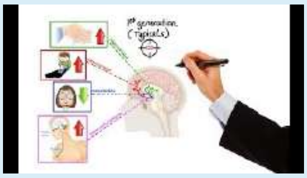

DOPAMINE
PATHWAYS
Hypothalamus
Nucleus accumbens
Mesocortical pathway
Nigrostriatal pathway
Cerebellum

Antipsychotics, Dopamine, and EPS

Dirty USMLE - YouTube 👍 2 👎 0

Video of patients with tardive dyskinesia

Abnormal Involuntary Movement Scale training video - YouTube 👍 0 👎 0



Antipsychotics

Speed Pharmacology - YouTube 👍 1 👎 1

Report broken media

Score: **20%**

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Question 106 of 409

A 42 year old computer programmer is brought to the Emergency Department by his wife with chest pains. He is haemodynamically stable with a blood pressure of 105/69mmHg and a heart rate of 92bpm but he is peripherally shut down with cool, dusky extremities and weak peripheral pulses. He complains of crushing central and left sided chest pain with some evidence of sweating and pallor. He also complains of severe global headache. His chest is clear and his heart sounds are normal. Chest x-ray is unremarkable.

An ECG shows pronounced inferior and lateral ST segment depression. A baseline troponin at 4 hours since onset of chest pain is raised. His wife tells you he has been taking clarithromycin from the GP for the past 6 days for a chest infection and she has been concerned he has been acting strangely since starting this with confusion, excitability, delusions and visual hallucinations. He has also had stomach cramps and diarrhoea for the past 2 days. His medical history is significant for migraines for which he takes PRN zolmitriptan and prophylactic ergotamine.

Which one of the following explains this patients symptoms most completely?

○ Acute non-ST segment myocardial infarction ×

○ Acute meningoencephalitis ×

○ Clarithromycin induced haemolytic uraemic syndrome (HUS) ×

○ Ergotism ×

○ Serotonin syndrome ×

Submit answer

Reference ranges

Score: **20%**

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Which one of the following explains this patients symptoms most completely?

Acute non-ST segment myocardial infarction	4%
Acute meningoencephalitis	4%
Clarithromycin induced haemolytic uraemic syndrome (HUS)	12%
Ergotism	62%
Serotonin syndrome	18%

Concomitant use of ergotamine and macrolides may cause ergot poisoning (ergotism) with confusion, headache, seizures, psychosis and global vasoconstriction leading to critical limb ischaemia, cardiac ischaemia and bowel hypoperfusion

Important for me Less important

In this case, this patient is quite clearly having an acute coronary event but non ST elevation myocardial infarction does not explain all his symptoms. Meningoencephalitis is a possibility but without further evidence of infection is difficult to prove and does not explain the cardiac ischaemia or bowel and limb symptoms. Serotonin syndrome may well be seen with drugs which modulate serotonergic neurotransmission such as zolmitriptan and cause confusion and agitation but the hallmark of this syndrome is hyperpyrexia which is absent here; cool peripheries would not be expected. Clarithromycin induced HUS could explain the majority of symptoms described here, however it is exceedingly rare and given that this patient has taken both clarithromycin and ergotamine concomitantly, ergotism is a far more likely diagnosis.

Management of ergotism is largely supportive and discontinuation of offending drugs is important. Seizures and psychosis may be controlled with benzodiazepines. Hypotension and haemodynamic instability should be managed with filling; avoid vasopressors such as adrenaline, noradrenaline or vasopressin; dobutamine or milrinone may be used cautiously if needed. Vasodilators such as GTN, iloprost or nitroprusside may be needed for critical ischaemia. Coronary ischaemia should be assessed as in any patient. Rebound migraine may be expected on cessation of ergot derivatives.

👍 👎 Discuss (2) Improve

Next question >

Ergotamine ★

Ergotamine is an old drug and a member of the family of ergot alkaloids. It is licensed as a treatment and prophylaxis for migraines but has been largely superseded by newer agents despite its efficacy, cost and relatively benign side effect profile. A derivative of the drug, ergometrine, is used in obstetrics to reduce the incidence of post partum haemorrhage. Ergotamine, like all ergot alkaloids, is a potent vasoconstrictor which is partly how it exerts its clinical effects, however in overdosage it can cause significant peripheral vasoconstriction causing critical ischaemia and gangrene. Coronary vasoconstriction may occur, with or without flow limiting lesions causing cardiac ischaemia which may be manifest as chest pain, arrhythmia or even sudden death. Contraindications to the use of ergotamine are flow limiting coronary lesions or peripheral vascular disease. Additionally, ergotamine has a complex series of effects on central nervous neurotransmitter systems including serotonergic, dopaminergic and noradrenergic systems which can cause excitement, confusion, paranoia, visual and auditory hallucinations and delusions in overdose. It is also a metabolic precursor to the highly hallucinogenic chemical lysergic acid diethylamide (LSD) which inactivates 5-HT2A receptors in the brain.

At normal doses, side effects of ergotamine are relatively minor and unlikely to cause significant clinical signs in the absence of underlying pathology. However, metabolism of ergot alkaloids is predominantly by the hepatic enzyme CYP3A4 which is almost totally inhibited by macrolide antibiotics. Co-administration of ergotamine and clarithromycin may be expected to produce a rapid picture of ergotism with confusion, psychosis, muscle cramps, seizures, peripheral and coronary vasospasm, severe headache and gastrointestinal symptoms of bowel ischaemia, cramps, diarrhoea and GI haemorrhage. Myocardial infarction, renal infarction, stroke and critical limb ischaemia may occur if not treated. Interestingly, ergot alkaloid derivatives are naturally produced by the fungus Claviceps purpurea which may infect crops. Historically, significant outbreaks of ergotism have been seen due to ingestion of crops contaminated with ergot and there is some historical evidence that claims of witchcraft are ascribable to the psychosis of ergot poisoning.

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

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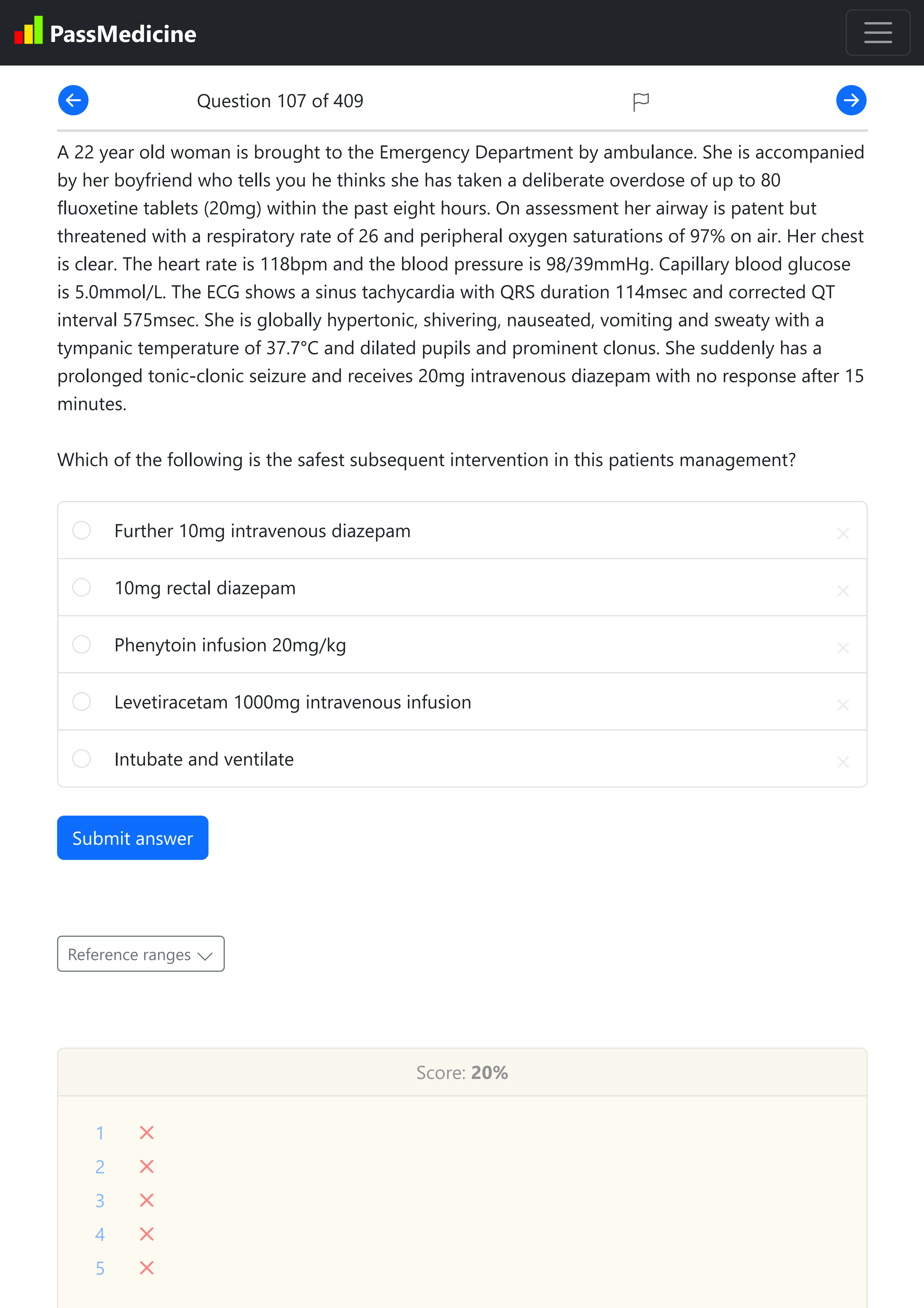

Question 107 of 409

A 22 year old woman is brought to the Emergency Department by ambulance. She is accompanied by her boyfriend who tells you he thinks she has taken a deliberate overdose of up to 80 fluoxetine tablets (20mg) within the past eight hours. On assessment her airway is patent but threatened with a respiratory rate of 26 and peripheral oxygen saturations of 97% on air. Her chest is clear. The heart rate is 118bpm and the blood pressure is 98/39mmHg. Capillary blood glucose is 5.0mmol/L. The ECG shows a sinus tachycardia with QRS duration 114msec and corrected QT interval 575msec. She is globally hypertonic, shivering, nauseated, vomiting and sweaty with a tympanic temperature of 37.7°C and dilated pupils and prominent clonus. She suddenly has a prolonged tonic-clonic seizure and receives 20mg intravenous diazepam with no response after 15 minutes.

Which of the following is the safest subsequent intervention in this patients management?

- ○ Further 10mg intravenous diazepam
- ○ 10mg rectal diazepam
- ○ Phenytoin infusion 20mg/kg
- ○ Levetiracetam 1000mg intravenous infusion
- ○ Intubate and ventilate

Score: **20%**

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Question 107 of 409 ✓

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Which of the following is the safest subsequent intervention in this patients management?

Further 10mg intravenous diazepam	10%
10mg rectal diazepam	1%
Phenytoin infusion 20mg/kg	23%
Levetiracetam 1000mg intravenous infusion	14%
Intubate and ventilate	52%

Care should be taken in status epilepticus due to drug toxicities refractory to benzodiazepines; phenytoin is cardiotoxic and phenobarbital is a potent respiratory depressant. Intubation is often safest

Important for me Less important

Seizures in drug overdoses are relatively common occurrences and may be related to the drug itself, its metabolites or as a consequence of electrolyte derangements. Generally, brief and isolated convulsions do not require specific treatment and may be managed conservatively. Recurrent or prolonged seizures predispose to injury, hypoxia and renal failure; first line treatment is with benzodiazepines such as diazepam, lorazepam or midazolam. In pre-hospital environments, or in those patients in whom intravascular access is yet to be established, rectal diazepam is a reasonable route of administration since it is rapidly absorbed across the bowel wall and enters the pudendal vasculature and is rapidly distributed to the central nervous system without passing through the liver first. If intravenous access is present however, as in the above patient, this route should be used. If a choice of intravenous benzodiazepines is available, lorazepam should be used preferentially since diazepam emulsion has a high risk of thrombophlebitis. In the above vignette, 20mg intravenous diazepam has been administered with no clinical effect; this is the maximal dose permitted in 4 hours and further doses are unlikely to be helpful. Second line agents which are recommended in persistent seizure activity are phenytoin or phenobarbital. Care must be taken when using phenytoin as an anti-seizure drug in overdoses since it exerts its anti-epileptic effects by inhibiting sodium channels; this is an effect shared by many cardiotoxic drugs taken in overdose. Co-administration of phenytoin and many drugs may lead to significant widening of the QRS duration, prolongation of the QT interval and ultimately to torsades de pointes, ventricular tachycardias and cardiac arrest. Phenytoin should not be used when there is any ECG evidence of prolongation of QRS or QT phases.

Levetiracetam is a relatively new antiepileptic drug which works by modulating NMDA glutamate neurotransmission. It can be given intravenously when oral routes are unavailable but it is licensed only for use in established epilepsy and not as a treatment for status epilepticus or drug induced seizures and should not be used here.

Phenobarbital is a drug which is rarely used in status epilepticus due to the risk of significant respiratory depression. It is however considered a second line agent to treat seizures as an alternative to phenytoin and could potentially be used here, although given the rest of the clinical information and probable evolving serotonin syndrome, intubation, ventilation and paralysis is probably safest.

In poisonings with cardiotoxic drugs, use of antiarrhythmic drugs to control ECG anomalies is not recommended; intravenous sodium bicarbonate may be given in patients with prolongation of QRS duration and magnesium sulphate can be given in QT prolongation or torsades de pointes, although the QT duration may not reduce but the incidence of escape rhythms is lessened.

👍 👎 Discuss (11) Improve

Next question >

Status epilepticus ★

Status epilepticus is defined as:

- a single seizure lasting >5 minutes, or
- >= 2 seizures within a 5-minute period without the person returning to normal between them

This is a medical emergency. The priority is the termination of seizure activity, which if prolonged will lead to irreversible brain damage.

Management

- ABC

    - airway adjunct
    - oxygen
    - check blood glucose
- First-line drugs are benzodiazepines
    - in the prehospital setting PR diazepam or buccal midazolam may be given
    - in hospital IV lorazepam is generally used. This may be repeated once after 5-10minutes
- If ongoing (or 'established') status it is appropriate to start a second-line agent such as levetiracetam, phenytoin or sodium valproate
    - NICE state 'Take into account that levetiracetam may be quicker to administer and have fewer adverse effects than the alternative options.'
- If no response ('refractory status') within 45 minutes from onset, then the best way to achieve rapid control of seizure activity is induction of general anaesthesia or phenobarbital

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 14 9

2022 Epilepsy guidelines

Suggest link Report broken link

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A 58-year-old man presents to the emergency department with eight hours of a painful red eye. He has a past medical history of inflammatory bowel disease, type 2 diabetes and hypertension. His regular medications include metformin, amlodipine and mesalazine. He does not smoke or drink alcohol. He wears contact lenses.

On examination, his right eye is red. Visual acuity is decreased in the right eye (6/18) in comparison to the left eye (6/6). The pupil in the right eye is fixed and mid-dilated.

Fluorescein staining of the right eye is normal.

Which of the following options is used in the management of the likely diagnosis?

- ○ IV acetazolamide
- ○ Oral steroids
- ○ Saline irrigation
- ○ Topical antibiotics
- ○ Topical steroids

Submit answer

Reference ranges

Score: **20%**

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A 58-year-old man presents to the emergency department with eight hours of a painful red eye. He has a past medical history of inflammatory bowel disease, type 2 diabetes and hypertension. His regular medications include metformin, amlodipine and mesalazine. He does not smoke or drink alcohol. He wears contact lenses.

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Fluorescein staining of the right eye is normal.

Which of the following options is used in the management of the likely diagnosis?

IV acetazolamide	59%
Oral steroids	9%
Saline irrigation	1%
Topical antibiotics	4%
Topical steroids	27%

Alongside eye drops, IV acetazolamide is used in the initial emergency medical management of acute angle-closure glaucoma

Important for me Less important

IV acetazolamide is the correct answer. The patient presents with a painful red eye with reduced visual acuity. The characteristic finding of acute glaucoma is a fixed mid-dilated pupil. Diabetes and hypertension are risk factors for the development of this condition. Alongside eye drops, IV acetazolamide is used in the emergency management of this condition.

Saline irrigation is incorrect. This has a place in the management of a chemical injury to the eyes but there is no history of such an event.

Topical and oral steroids are both incorrect. Both these options have a place in the management of anterior uveitis. However, the pupil is typically miotic in this condition. Additionally, the onset is more insidious and frequently bilateral when affecting those patients with inflammatory bowel disease.

Topical antibiotics is not correct. This may be indicated in the management of bacterial conjunctivitis or a corneal abrasion. The lack of discharge and the existence of visual obscuration make conjunctivitis an unlikely cause. Normal fluorescein staining makes a corneal abrasion unlikely despite the history of contact lens use.

Discuss (2) | Improve

Next question ›

Acute angle-closure glaucoma

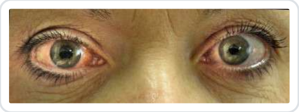
Glaucoma is a group of disorders characterised by optic neuropathy due, in the majority of patients, to raised intraocular pressure (IOP). It is now recognised that a minority of patients with raised IOP do not have glaucoma and vice versa.

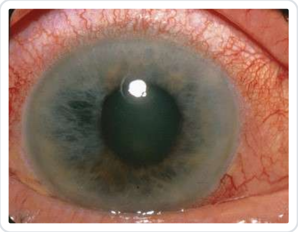
In acute angle-closure glaucoma (AACG) there is a rise in IOP secondary to an impairment of aqueous outflow. Factors predisposing to AACG include:

- hypermetropia (long-sightedness)
- pupillary dilatation
- lens growth associated with age

Features

- severe pain: may be ocular or headache
- decreased visual acuity
- symptoms worse with mydriasis (e.g. watching TV in a dark room)
- hard, red-eye
- haloes around lights
- semi-dilated non-reacting pupil
- corneal oedema results in dull or hazy cornea
- systemic upset may be seen, such as nausea and vomiting and even abdominal pain





Investigations

- tonometry to assess for elevated IOP
- gonioscopy (literally looking, *oscopy*, at the angle, *gonio*): a special lens for the slit lamp that allows visualisation of the angle

Management

The management of AACG is an emergency and should prompt urgent referral to an ophthalmologist. Emergency medical treatment is required to lower the IOP with more definitive surgical treatment given once the acute attack has settled.

There are no guidelines for the initial medical treatment emergency treatment. An example regime would be:

- combination of eye drops, for example:
    - a direct parasympathomimetic (e.g. pilocarpine, causes contraction of the ciliary muscle → opening the trabecular meshwork → increased outflow of the aqueous humour)
    - a beta-blocker (e.g. timolol, decreases aqueous humour production)
    - an alpha-2 agonist (e.g. apraclonidine, dual mechanism, decreasing aqueous humour production and increasing uveoscleral outflow)
- intravenous acetazolamide
    - reduces aqueous secretions
- some guidelines also recommend the use of topical steroids to reduce inflammation

Definitive management

- laser peripheral iridotomy
    - creates a tiny hole in the peripheral iris → aqueous humour flowing to the angle

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

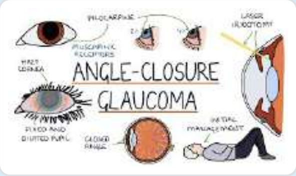
Patient.info 👍 26 👎 9

Acute angle closure glaucoma review

Suggest link

Report broken link

Media


PILOCARPINE
LASER IRIDOTOMY
MUSCARINIC RECEPTORS
HAZY CORNEA
ANGLE-CLOSURE GLAUCOMA
FIXED AND DILATED PUPIL
CLOSED ANGLE
INITIAL MANAGEMENT

Acute angle closure glaucoma

Zero To Finals - YouTube 👍 4 👎 0

GLAUCOMA
Osmosis.org

Glaucoma (open-angle, closed-angle, and normal-tension)

Osmosis - YouTube 👍 6 👎 3

Report broken media

Score: **20%**

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A 72-year-old man is brought to the emergency department by ambulance after complaining of sudden onset weakness. He has a past medical history of hypertension and atrial fibrillation. His medications include amlodipine, bisoprolol and apixaban.

On examination, power is 4/5 in the right upper limb and right lower limb. Cranial nerve examination reveals his left eye to have deviated inferiorly and laterally. There is left-sided ptosis. The pupillary responses are normal.

A CT head is unremarkable.

Based on the likely diagnosis, which cerebral artery is most likely affected?

- ○ Left anterior cerebral artery ×
- ○ Basilar artery ×
- ○ Left middle cerebral artery ×
- ○ Left posterior cerebral artery ×
- ○ Left retinal artery ×

Submit answer

Reference ranges

Score: **20%**

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Question 109 of 409

A 72-year-old man is brought to the emergency department by ambulance after complaining of sudden onset weakness. He has a past medical history of hypertension and atrial fibrillation. His medications include amlodipine, bisoprolol and apixaban.

On examination, power is 4/5 in the right upper limb and right lower limb. Cranial nerve examination reveals his left eye to have deviated inferiorly and laterally. There is left-sided ptosis. The pupillary responses are normal.

A CT head is unremarkable.

Based on the likely diagnosis, which cerebral artery is most likely affected?

Left anterior cerebral artery	11%
Basilar artery	15%
Left middle cerebral artery	26%
Left posterior cerebral artery	47%
Left retinal artery	1%

Weber's syndrome is a form of midbrain stroke characterised by the an ipsilateral CN III palsy and contralateral hemiparesis

Important for me Less important

Posterior cerebral artery is correct. The patient presents with an ipsilateral third nerve palsy and contralateral hemiplegia. The sudden onset and history of hypertension and AF suggest a vascular event. This is suggestive of Weber's syndrome, which is caused by infarction of a branch of the posterior cerebral artery in the midbrain.

Basilar artery is incorrect. Infarction as a consequence of basilar artery thrombosis would cause 'locked-in syndrome. This is when the body and most of the facial muscles are paralyzed but consciousness and eye movements are preserved.

Left middle cerebral artery is incorrect. A stroke affecting this region may result in contralateral (i.e. right-sided) weakness, sensory disturbance and speech disturbance.

Left anterior cerebral artery is incorrect. This is characterized by weakness and sensory loss affecting the lower limbs more than the upper limbs.

Left retinal artery is incorrect. This would cause monocular visual loss.

👍 👎 Discuss (5) Improve

Next question >

Stroke by anatomy ★

Site of the lesion	Associated effects
Anterior cerebral artery	Contralateral hemiparesis and sensory loss, lower extremity > upper
Middle cerebral artery	Contralateral hemiparesis and sensory loss, upper extremity > lower Contralateral homonymous hemianopia Aphasia
Posterior cerebral artery	Contralateral homonymous hemianopia with macular sparing Visual agnosia
Weber's syndrome (branches of the posterior cerebral artery that supply the midbrain)	Ipsilateral CN III palsy Contralateral weakness of upper and lower extremity
Posterior inferior cerebellar artery (lateral medullary syndrome, Wallenberg syndrome)	Ipsilateral: facial pain and temperature loss Contralateral: limb/torso pain and temperature loss Ataxia, nystagmus
Anterior inferior cerebellar artery (lateral pontine syndrome)	Symptoms are similar to Wallenberg's (see above), but: Ipsilateral: facial paralysis and deafness
Retinal/ophthalmic artery	Amaurosis fugax
Basilar artery	'Locked-in' syndrome

Lacunar strokes

- present with either isolated hemiparesis, hemisensory loss or hemiparesis with limb ataxia

- strong association with hypertension
- common sites include the basal ganglia, thalamus and internal capsule

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 75 10

Brainstem Rules of 4

Suggest link

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Media

Management of ischaemic stroke

Stroke

Podmedics - YouTube 0 0

Brainstem Stroke

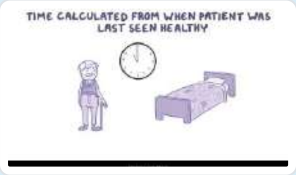
Brainstem stroke syndromes

Soton Brain Hub - YouTube 13 1

NEUROLOGY
MIDBRAIN SYNDROMES

Midbrain Syndromes - Weberâ€™s Syndrome, Benediktâ€™s Syndrome and Parinaud Syndrome

Armando Hasudungan - YouTube 👍 2 👎 1


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

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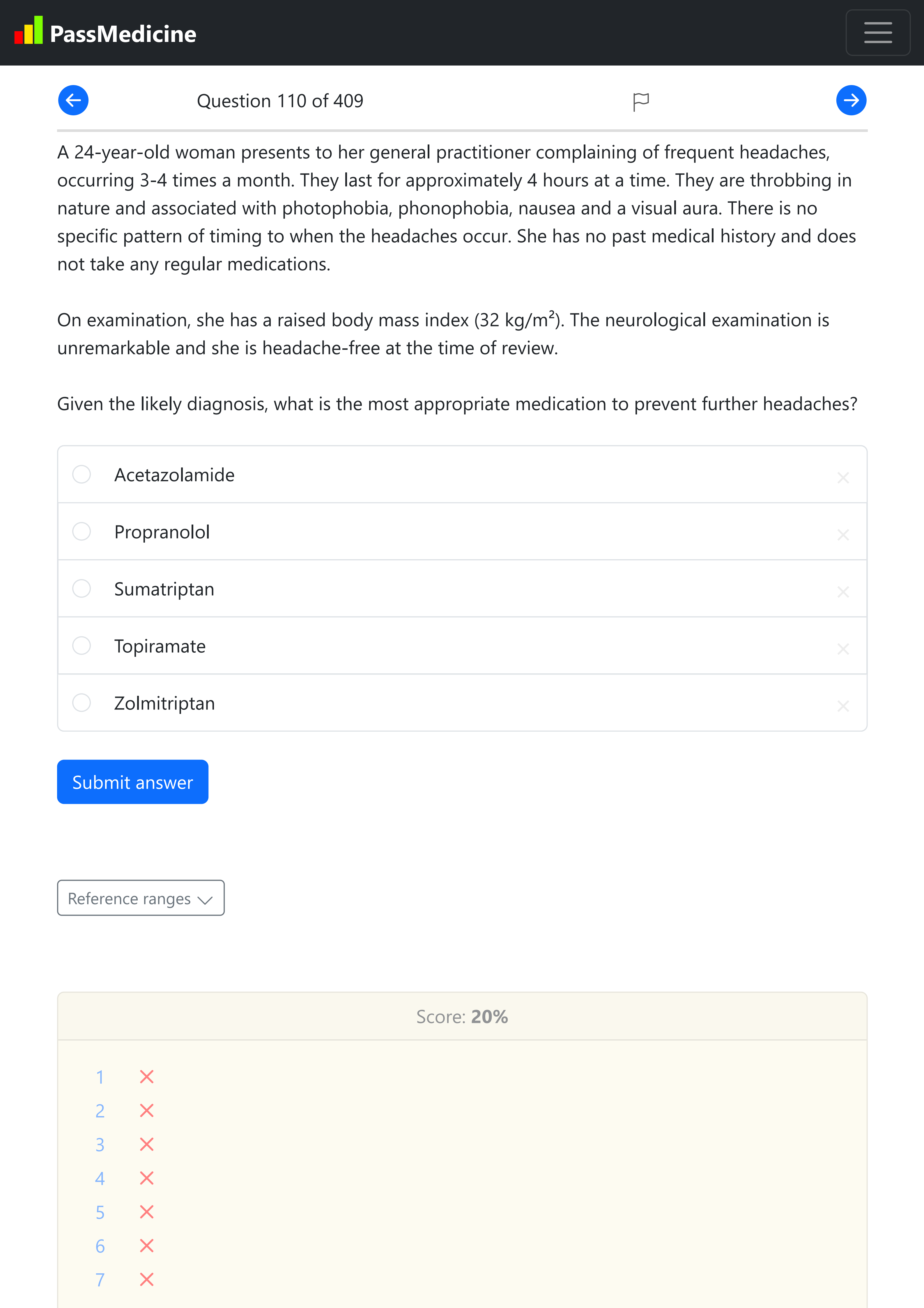

Question 110 of 409

A 24-year-old woman presents to her general practitioner complaining of frequent headaches, occurring 3-4 times a month. They last for approximately 4 hours at a time. They are throbbing in nature and associated with photophobia, phonophobia, nausea and a visual aura. There is no specific pattern of timing to when the headaches occur. She has no past medical history and does not take any regular medications.

On examination, she has a raised body mass index (32 kg/m^2). The neurological examination is unremarkable and she is headache-free at the time of review.

Given the likely diagnosis, what is the most appropriate medication to prevent further headaches?

- ○ Acetazolamide
- ○ Propranolol
- ○ Sumatriptan
- ○ Topiramate
- ○ Zolmitriptan

Submit answer

Reference ranges

Score: **20%**

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A 24-year-old woman presents to her general practitioner complaining of frequent headaches, occurring 3-4 times a month. They last for approximately 4 hours at a time. They are throbbing in nature and associated with photophobia, phonophobia, nausea and a visual aura. There is no specific pattern of timing to when the headaches occur. She has no past medical history and does not take any regular medications.

On examination, she has a raised body mass index (32 kg/m²). The neurological examination is unremarkable and she is headache-free at the time of review.

Given the likely diagnosis, what is the most appropriate medication to prevent further headaches?

Acetazolamide	9%
Propranolol	77%
Sumatriptan	6%
Topiramate	8%
Zolmitriptan	0%

Propranolol is preferable to topiramate in women of childbearing age (i.e. the majority of women with migraine)

Important for me Less important

Propranolol is the correct answer. This woman presents with typical features of migraine (episodic throbbing headache associated with aura, photophobia, phonophobia and nausea). The first choice of prophylaxis of migraine in a woman of childbearing age is propranolol.

Acetazolamide is incorrect. While she does have a raised BMI, which is a risk factor for idiopathic intracranial hypertension, the headache characteristics are far more suggestive of migraines. Additionally, we are told the neurological examination is normal suggesting an absence of papilloedema.

Sumatriptan is incorrect. This is used to treat acute migraine rather than prevent migraine.

Topiramate is incorrect. This can be used for prophylaxis of migraine but is potentially teratogenic and therefore less preferable in women of child-bearing age.

Zolmitriptan is incorrect. This can be used as prophylaxis for menstrual migraines. However, we are told her headaches have no predictable pattern and therefore this is not indicated.

👍 👎 Discuss (2) Improve

Next question ›

Migraine: management ★

It should be noted that as a general rule 5-HT receptor agonists are used in the acute treatment of migraine whilst 5-HT receptor antagonists are used in prophylaxis. NICE produced guidelines in 2012 on the management of headache, including migraines.

Acute treatment

- first-line: offer combination therapy with
  - an oral triptan and an NSAID, or
  - an oral triptan and paracetamol
- for young people aged 12-17 years consider a nasal triptan in preference to an oral triptan
- if the above measures are not effective or not tolerated offer a non-oral preparation of metoclopramide or prochlorperazine and consider adding a non-oral NSAID or triptan
  - caution should be exercised when prescribing metoclopramide to young patients as acute dystonic reactions may develop

Prophylaxis

- prophylaxis should generally be given if '*Migraine attacks are having a significant impact on quality of life and daily function, for example they occur frequently (more than once a week on average) or are prolonged and severe despite optimal acute treatment*'
- options:
  - propranolol
  - topiramate: should be avoided in women of childbearing age as it may be teratogenic and it can reduce the effectiveness of hormonal contraceptives
  - amitriptyline
- if these measures fail NICE recommends 'a course of up to 10 sessions of acupuncture over 5-8 weeks'
- NICE recommend: '*Advise people with migraine that riboflavin (400 mg once a day) may be effective in reducing migraine frequency and intensity for some people*'
- for women with predictable menstrual migraine treatment NICE recommend either frovatriptan (2.5 mg twice a day) or zolmitriptan (2.5 mg twice or three times a day) as a type of 'mini-prophylaxis'
- treatment options that may be considered by specialists, but fall outside the NICE guidelines:

    - candesartan: recommended by the British Association for the Study of Headache guidelines
    - monoclonal antibodies directed against the calcitonin gene-related peptide (CGRP) receptor: examples include erenumab
- pizotifen is no longer recommended. Adverse effects such as weight gain & drowsiness are common

+ Q 123

Next question >

B *I* A T↕

Textbooks

High-yield textbook

Extended textbook

Links

British Association for the Study of Headache Headache guidelines	👍 14	👎 21
Clinical Knowledge Summaries Migraine guidelines	👍 14	👎 7
NICE 2012 Headache guidelines	👍 16	👎 4

Suggest link

Report broken link

Media

Management of Migraine
Podmedics

Migraine

Podmedics - YouTube 👍 1 👎 1

Report broken media

Score: **20%**

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A 64-year-old lady presents with worsening anxiety to the emergency department. She has no history of mental health issues but her husband is worried that over the past month she has become very nervous and developed panic attacks. In addition, she is not able to carry out her daily tasks. She is retired and usually keeps active but over the past few weeks has become more disengaged and no longer is arranging to see friends or leave the house. She has terrors in the night where she screams and takes several hours of calming from her husband.

She is normally well and takes ramipril and a simvastatin for high blood pressure.

On examination, she is withdrawn but alert. She is haemodynamically stable and afebrile. Her abbreviated mental test score is 3/10 and she does not know where she is or the time of day. She has mildly increased tone in both arms with occasional myoclonic jerks and the power is 4/5 in all muscle groups. There is intact coordination and sensation.

Hb	140 g/l
Platelets	$398 * 10^9/l$
WBC	$7.6 * 10^9/l$

CRP	5mg/l
Na^+	134 mmol/l
K^+	4.2 mmol/l
Urea	4.3 mmol/l
Creatinine	67 µmol/l

CT head	brain atrophy, no intracranial haemorrhage or mass effect
MRI head	Hyperintense signals in the left thalamus with some similar changes in both putamen and right thalamus

What treatment should be initiated?

○ Supportive

○ Cyclophosphamide

- ○ Methylprednisolone ×
- ○ Intravenous antibiotics ×
- ○ Venlafaxine ×

Submit answer

Reference ranges

Score: **0%**

1 -

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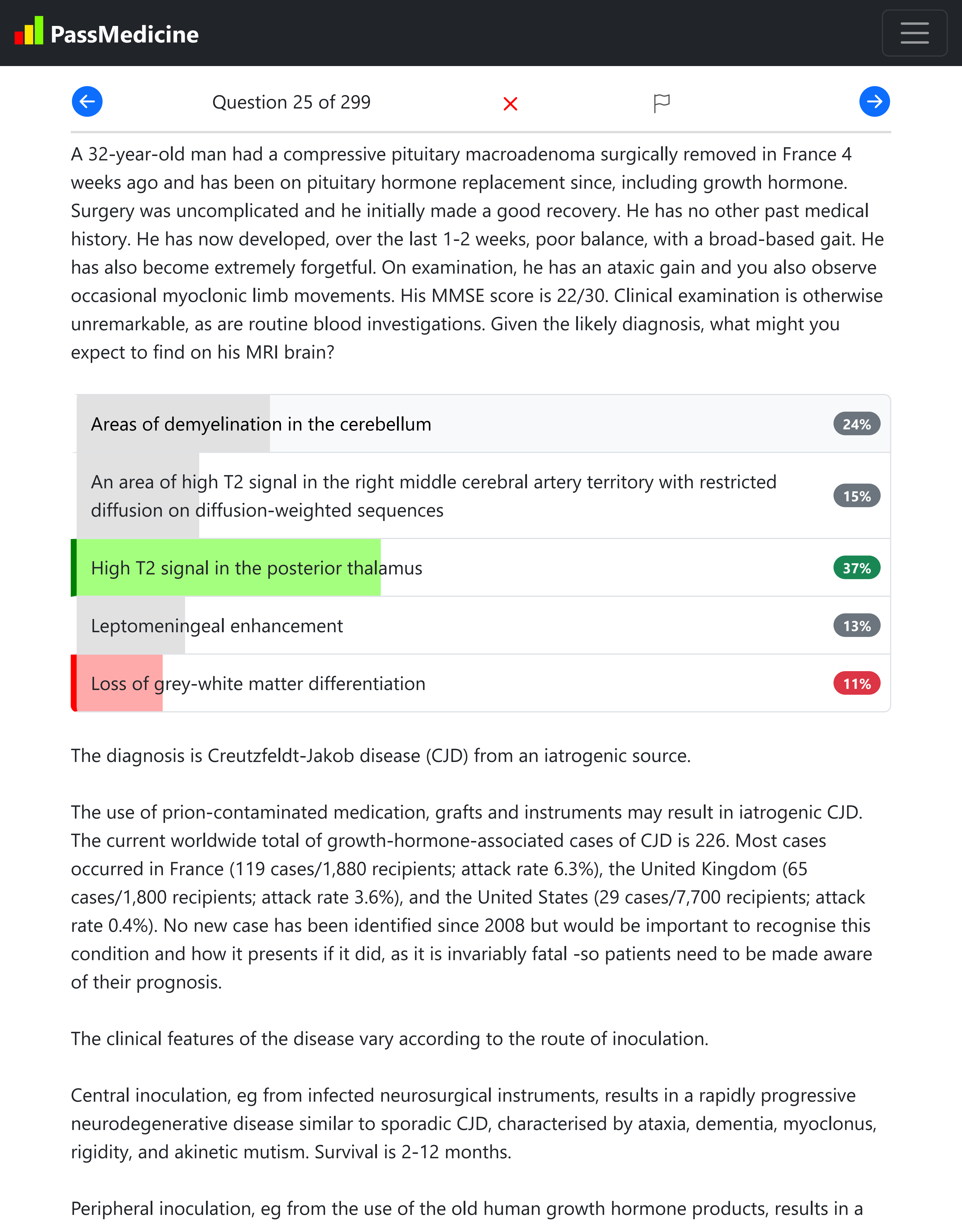

A 32-year-old man had a compressive pituitary macroadenoma surgically removed in France 4 weeks ago and has been on pituitary hormone replacement since, including growth hormone. Surgery was uncomplicated and he initially made a good recovery. He has no other past medical history. He has now developed, over the last 1-2 weeks, poor balance, with a broad-based gait. He has also become extremely forgetful. On examination, he has an ataxic gain and you also observe occasional myoclonic limb movements. His MMSE score is 22/30. Clinical examination is otherwise unremarkable, as are routine blood investigations. Given the likely diagnosis, what might you expect to find on his MRI brain?

Areas of demyelination in the cerebellum	24%
An area of high T2 signal in the right middle cerebral artery territory with restricted diffusion on diffusion-weighted sequences	15%
High T2 signal in the posterior thalamus	37%
Leptomeningeal enhancement	13%
Loss of grey-white matter differentiation	11%

The diagnosis is Creutzfeldt-Jakob disease (CJD) from an iatrogenic source.

The use of prion-contaminated medication, grafts and instruments may result in iatrogenic CJD. The current worldwide total of growth-hormone-associated cases of CJD is 226. Most cases occurred in France (119 cases/1,880 recipients; attack rate 6.3%), the United Kingdom (65 cases/1,800 recipients; attack rate 3.6%), and the United States (29 cases/7,700 recipients; attack rate 0.4%). No new case has been identified since 2008 but would be important to recognise this condition and how it presents if it did, as it is invariably fatal -so patients need to be made aware of their prognosis.

The clinical features of the disease vary according to the route of inoculation.

Central inoculation, eg from infected neurosurgical instruments, results in a rapidly progressive neurodegenerative disease similar to sporadic CJD, characterised by ataxia, dementia, myoclonus, rigidity, and akinetic mutism. Survival is 2-12 months.

Peripheral inoculation, eg from the use of the old human growth hormone products, results in a slightly less acute disease -characterised by progressive cerebellar ataxia and dementia. Survival is 8-18 months.

👍 👎 Discuss (10) Improve

Next question >

Creutzfeldt-Jakob disease ★

Creutzfeldt-Jakob disease (CJD) is rapidly progressive neurological condition caused by prion proteins. These proteins induce the formation of amyloid folds resulting in tightly packed beta-pleated sheets resistant to proteases.

Features
- dementia (rapid onset)
- myoclonus

Investigation
- CSF is usually normal
- EEG: biphasic, high amplitude sharp waves (only in sporadic CJD)
- MRI: hyperintense signals in the basal ganglia and thalamus

Sporadic CJD
- accounts for 85% of cases
- 10-15% of cases are familial
- mean age of onset is 65 years

New variant CJD
- younger patients (average age of onset = 25 years)
- psychological symptoms such as anxiety, withdrawal and dysphonia are the most common presenting features
- the 'prion protein' is encoded on chromosome 20 - it's role is not yet understood
- methionine homozygosity at codon 129 of the prion protein is a risk factor for developing CJD - all patients who have so far died have had this
- median survival = 13 months

Other prion diseases
- kuru

- fatal familial insomnia
- Gerstmann Straussler-Scheinker disease

123

Next question >

Textbooks

High-yield textbook

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Media

PRIONS
(SPONGIFORM
ENCEPHALOPATHY)

Prions (Spongiform encephalopathy)

Osmosis - YouTube 7 0

Report broken media

Score: **16%**

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A 42-year-old woman presents with a headache over the past week. She describes it as left sided, excruciating and it makes it difficult for her to sleep at night. The pain is worse when lying down and she notices that her nose is blocked on that side. She has vomited but has been feeling achy all over for the last few days. She has tried paracetamol and ibuprofen without benefit. She is otherwise well and only takes the combined oral contraceptive pill.

On examination, she appears lethargic. She has normal eye movements and fundoscopy is normal. She is able to move all her limbs with a power of 5/5 and has normal reflexes. She walks with a slow but steady gait. She is coryzal and she will not let you examine her face when you press over the frontal area.

She was seen in the emergency department a few days prior with this headache and the report is below.

CT head	There is no evidence of bleeding, mass effect or hydrocephalus. It is noted that there is a homogeneous collection of moderately enhancing material in the frontal sinus.

What is the most likely diagnosis?

- ○ Idiopathic intracranial hypertension
- ○ Sinusitis
- ○ Subarachnoid haemorrhage
- ○ Hemicrania continua
- ○ Meningioma

Submit answer

Reference ranges

Score: **0%**

1 -

2 -

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Question 2 of 299

A 42-year-old woman presents with a headache over the past week. She describes it as left sided, excruciating and it makes it difficult for her to sleep at night. The pain is worse when lying down and she notices that her nose is blocked on that side. She has vomited but has been feeling achy all over for the last few days. She has tried paracetamol and ibuprofen without benefit. She is otherwise well and only takes the combined oral contraceptive pill.

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She was seen in the emergency department a few days prior with this headache and the report is below.

CT head	There is no evidence of bleeding, mass effect or hydrocephalus. It is noted that there is a homogeneous collection of moderately enhancing material in the frontal sinus.

What is the most likely diagnosis?

Idiopathic intracranial hypertension	6%
Sinusitis	76%
Subarachnoid haemorrhage	1%
Hemicrania continua	12%
Meningioma	5%

Severe constant headache on a background of rhinitis is likely sinusitis

Important for me Less important

The history is an acute headache. Although there are features of raised intracranial pressure, a CT head excludes any meningioma. A subarachnoid haemorrhage typically is the most severe headache that reaches maximal intensity over a few seconds to minutes. Hemicrania continua is a constant headache that often has autonomic features associated with it that are not seen here. Idiopathic intracranial hypertension is possible given her combined OCP use but there are no other features and fundoscopy shows no papilloedema. Examination is normal but the patient is anxious to be examined due to the exquisite tenderness of the facial sinuses in sinusitis. Furthermore, the CT head reports a collection of mucus in the sinuses seen incidentally.

👍 | 👎 | Discuss (1) | Improve

Next question >

Acute sinusitis ★

Sinusitis describes an inflammation of the mucous membranes of the paranasal sinuses. The sinuses are usually sterile - the most common infectious agents seen in acute sinusitis are *Streptococcus pneumoniae*, *Haemophilus influenzae* and rhinoviruses.

Predisposing factors include:

- nasal obstruction e.g. septal deviation or nasal polyps
- recent local infection e.g. rhinitis or dental extraction
- swimming/diving
- smoking

Features

- facial pain
  - typically frontal pressure pain which is worse on bending forward
- nasal discharge: usually thick and purulent
- nasal obstruction

Management of acute sinusitis

- analgesia
- intranasal decongestants or nasal saline may be considered but the evidence supporting these is limited
- intranasal corticosteroids may be considered if the symptoms have been present for more than 10 days
- oral antibiotics are not normally required but may be given for severe presentations.
  - The BNF recommends phenoxymethylpenicillin first-line, co-amoxiclav if 'systemically very unwell, signs and symptoms of a more serious illness, or at high-risk of complications'

    - 'double-sickening' may sometimes be seen, where an initial viral sinusitis worsens due to secondary bacterial infection

123

Next question

Textbooks

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Links

Clinical Knowledge Summaries 12 8

Sinusitis

Suggest link

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Media

Sinusitis

Osmosis - YouTube 0 0

ANTIBIOTICS
INFECTION
NASAL SPRAY TECHNIQUE
SINUSITIS
POLYPS
HAYFEVER
SINUSES

Understanding Sinusitis

Zero To Finals - YouTube 👍 0 👎 0

Report broken media

Score: **16%**

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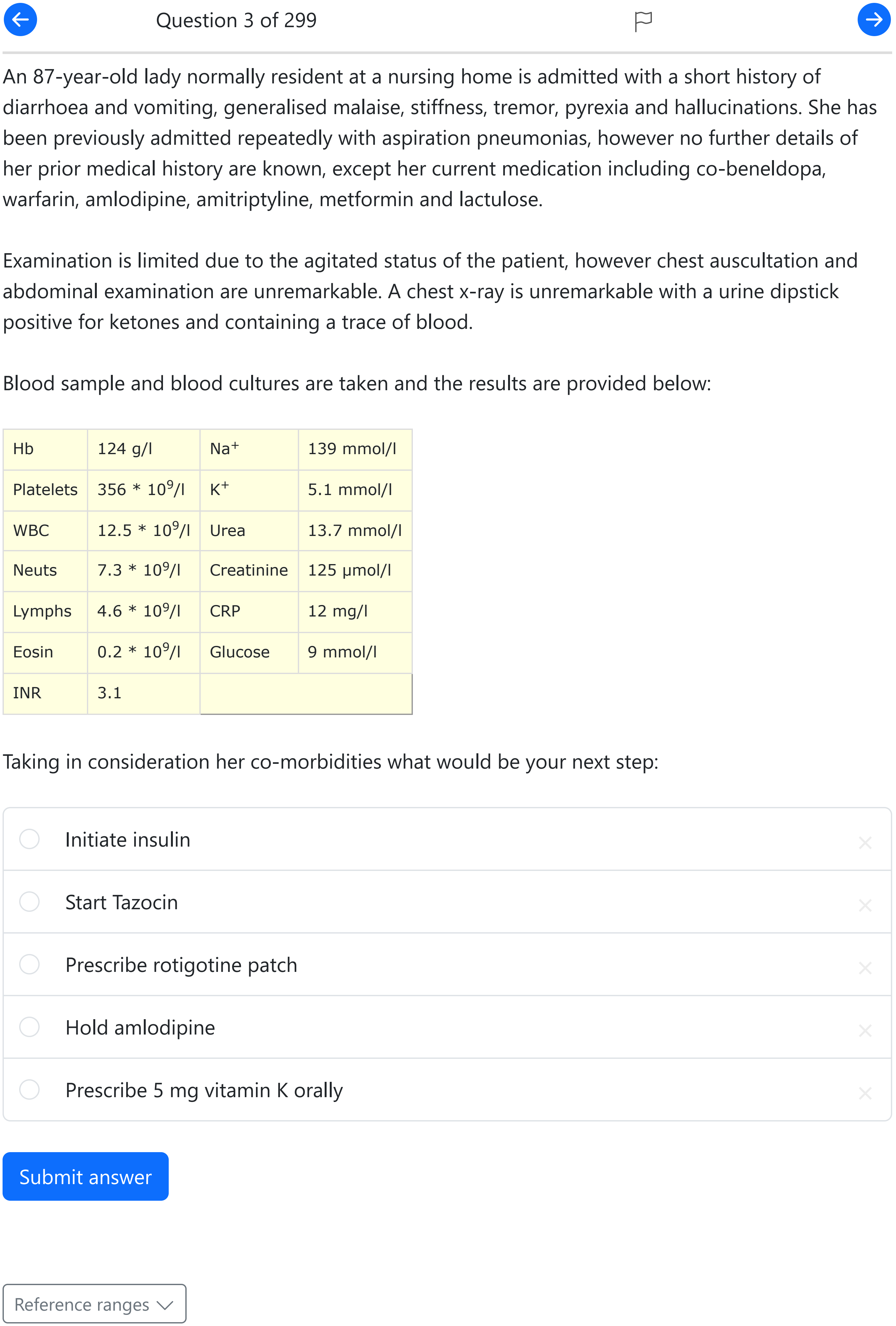

Question 3 of 299

An 87-year-old lady normally resident at a nursing home is admitted with a short history of diarrhoea and vomiting, generalised malaise, stiffness, tremor, pyrexia and hallucinations. She has been previously admitted repeatedly with aspiration pneumonias, however no further details of her prior medical history are known, except her current medication including co-beneldopa, warfarin, amlodipine, amitriptyline, metformin and lactulose.

Examination is limited due to the agitated status of the patient, however chest auscultation and abdominal examination are unremarkable. A chest x-ray is unremarkable with a urine dipstick positive for ketones and containing a trace of blood.

Blood sample and blood cultures are taken and the results are provided below:

Hb	124 g/l	Na^+	139 mmol/l
Platelets	356 * 10^9/l	K^+	5.1 mmol/l
WBC	12.5 * 10^9/l	Urea	13.7 mmol/l
Neuts	7.3 * 10^9/l	Creatinine	125 μmol/l
Lymphs	4.6 * 10^9/l	CRP	12 mg/l
Eosin	0.2 * 10^9/l	Glucose	9 mmol/l
INR	3.1		

Taking in consideration her co-morbidities what would be your next step:

- ○ Initiate insulin
- ○ Start Tazocin
- ○ Prescribe rotigotine patch
- ○ Hold amlodipine
- ○ Prescribe 5 mg vitamin K orally

Score: **0%**

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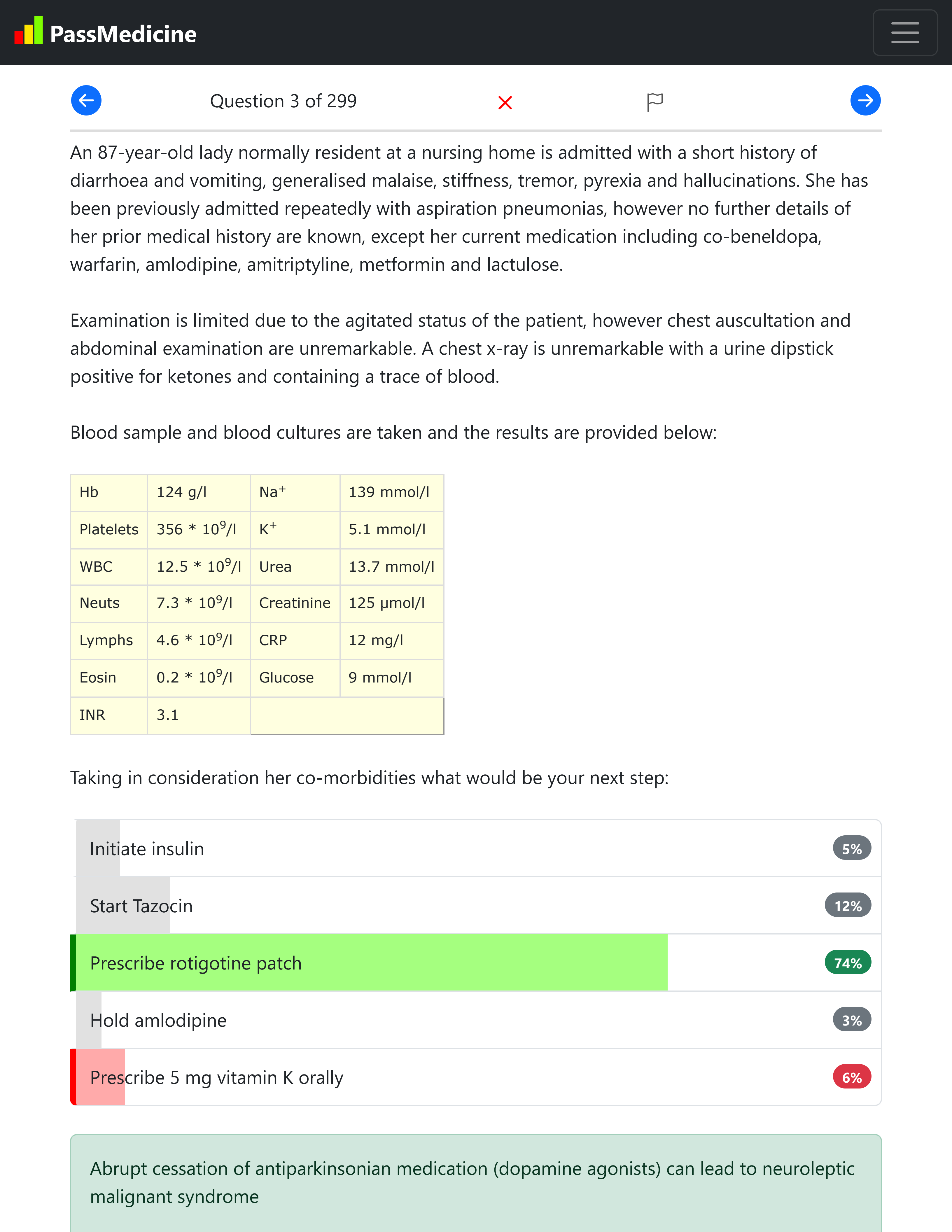

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Lymphs	4.6 * 10^9/l	CRP	12 mg/l
Eosin	0.2 * 10^9/l	Glucose	9 mmol/l
INR	3.1		

Taking in consideration her co-morbidities what would be your next step:

- Initiate insulin 5%
- Start Tazocin 12%
- Prescribe rotigotine patch 74%
- Hold amlodipine 3%
- Prescribe 5 mg vitamin K orally 6%

Abrupt cessation of antiparkinsonian medication (dopamine agonists) can lead to neuroleptic malignant syndrome

Important for me Less important

This is a common situation in hospital medicine. A lady that suffers from Parkinson's disease (even though not explicitly stated - but can be assumed given the frequent aspiration pneumonias and her medication) presents dehydrated with diarrhoea and vomiting. It is possible that this lady may have gastroenteritis and it hence important to rehydrate her. It is also however important to ensure that she continues to take her anti-parkinsonian medication. Given her inability to eat and drink at the moment the most appropriate alternative would be a parenteral or topical solution. Given her blood picture and inability to isolate a source of infection, one should be cautious about starting broad spectrum antibiotics.

👍 👎 Discuss (4) Improve

Next question ›

Neuroleptic malignant syndrome ★

Neuroleptic malignant syndrome is a rare but dangerous condition seen in patients taking antipsychotic medication. It carries a mortality of up to 10% and can also occur with atypical antipsychotics. It may also occur with dopaminergic drugs (such as levodopa) for Parkinson's disease, usually when the drug is suddenly stopped or the dose reduced.

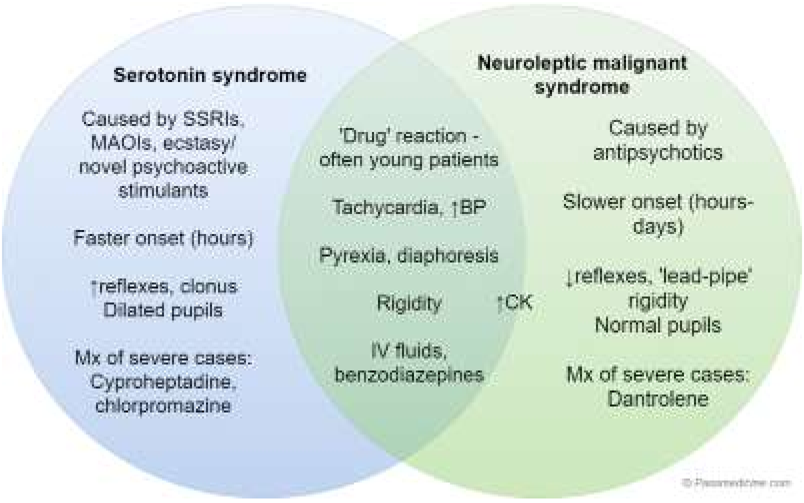
The pathophysiology is unknown but one theory is that the dopamine blockade induced by antipsychotics triggers massive glutamate release and subsequent neurotoxicity and muscle damage.

It occurs within hours to days of starting an antipsychotic (antipsychotics are also known as neuroleptics, hence the name) and the typical features are:
- pyrexia
- muscle rigidity
- autonomic lability: typical features include hypertension, tachycardia and tachypnoea
- agitated delirium with confusion

A raised creatine kinase is present in most cases. Acute kidney injury (secondary to rhabdomyolysis) may develop in severe cases. A leukocytosis may also be seen

Management
- stop antipsychotic

- patients should be transferred to a medical ward if they are on a psychiatric ward and often they are nursed in intensive care units
- IV fluids to prevent renal failure
- dantrolene may be useful in selected cases
  - thought to work by decreasing excitation-contraction coupling in skeletal muscle by binding to the ryanodine receptor, and decreasing the release of calcium from the sarcoplasmic reticulum
- bromocriptine, dopamine agonist, may also be used


Serotonin syndrome
Caused by SSRIs, MAOIs, ecstasy/ novel psychoactive stimulants
Faster onset (hours)
↑reflexes, clonus
Dilated pupils
Mx of severe cases: Cyproheptadine, chlorpromazine
Neuroleptic malignant syndrome
Caused by antipsychotics
Slower onset (hours-days)
↓reflexes, 'lead-pipe' rigidity
Normal pupils
Mx of severe cases: Dantrolene
'Drug' reaction - often young patients
Tachycardia, ↑BP
Pyrexia, diaphoresis
Rigidity
↑CK
IV fluids, benzodiazepines
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Venn diagram showing contrasting serotonin syndrome with neuroleptic malignant syndrome. Note that both conditions can cause a raised creatine kinase (CK) but it tends to be more associated with NMS.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Medsafe 👍 3 👎 0

Neuroleptice malignant syndrome Vs serotonin syndrome

Patient.info 👍 1 👎 1

Neuroleptic Malignant Syndrome

Suggest link

Report broken link

Media

DOPAMINE ANTAGONISM
AUTONOMIC INSTABILITY
NEUROLEPTIC MALIGNANT SYNDROME
FEVER
ANTIPSYCHOTICS

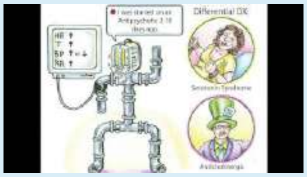
Neuroleptic malignant syndrome

Rhesus Medicine - YouTube 👍 1 👎 0

NEUROLEPTIC MALIGNANT SYNDROME

Neuroleptic malignant syndrome

Townsend Teaching - YouTube 👍 0 👎 0


Differential DX
Serotonin Syndrome

Neuroleptic malignant syndrome

Kloss and Bruce - YouTube 👍 0 👎 1

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Score: **16%**

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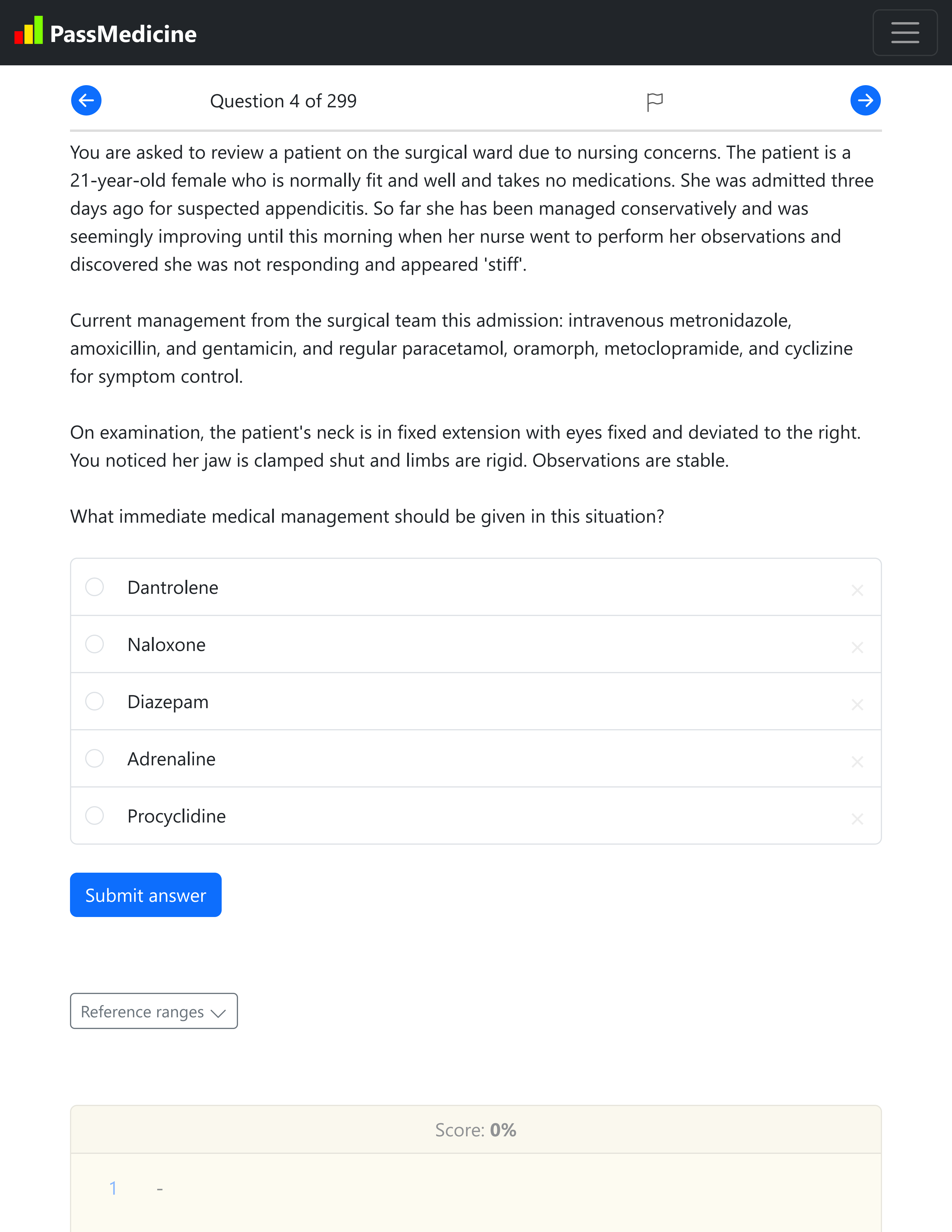

Question 4 of 299

You are asked to review a patient on the surgical ward due to nursing concerns. The patient is a 21-year-old female who is normally fit and well and takes no medications. She was admitted three days ago for suspected appendicitis. So far she has been managed conservatively and was seemingly improving until this morning when her nurse went to perform her observations and discovered she was not responding and appeared 'stiff'.

Current management from the surgical team this admission: intravenous metronidazole, amoxicillin, and gentamicin, and regular paracetamol, oramorph, metoclopramide, and cyclizine for symptom control.

On examination, the patient's neck is in fixed extension with eyes fixed and deviated to the right. You noticed her jaw is clamped shut and limbs are rigid. Observations are stable.

What immediate medical management should be given in this situation?

○ Dantrolene
○ Naloxone
○ Diazepam
○ Adrenaline
○ Procyclidine

Score: **0%**

1 -

2 -

3 -

4 -

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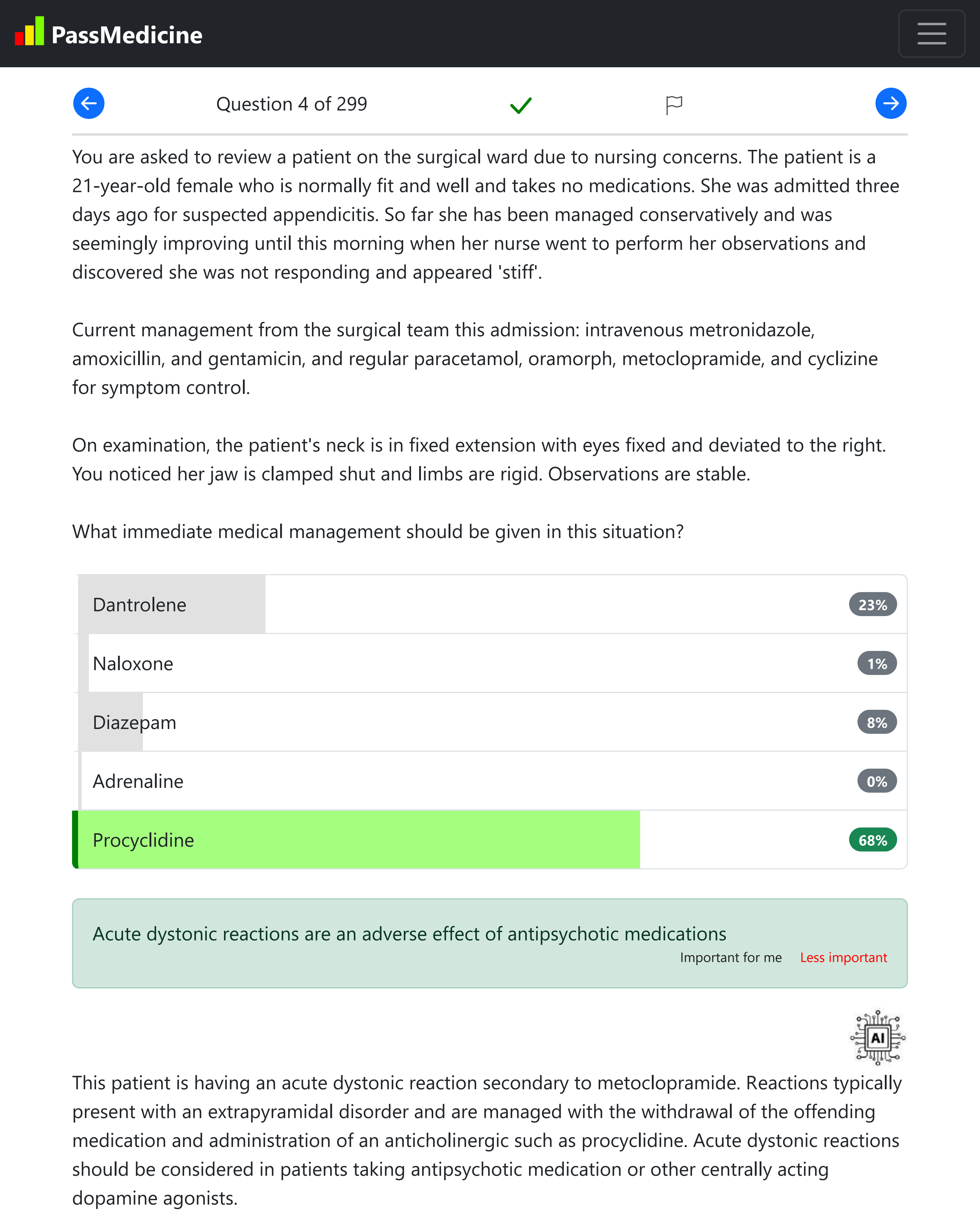

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On examination, the patient's neck is in fixed extension with eyes fixed and deviated to the right. You noticed her jaw is clamped shut and limbs are rigid. Observations are stable.

What immediate medical management should be given in this situation?

Dantrolene	23%
Naloxone	1%
Diazepam	8%
Adrenaline	0%
Procyclidine	68%

Acute dystonic reactions are an adverse effect of antipsychotic medications

Important for me Less important

This patient is having an acute dystonic reaction secondary to metoclopramide. Reactions typically present with an extrapyramidal disorder and are managed with the withdrawal of the offending medication and administration of an anticholinergic such as procyclidine. Acute dystonic reactions should be considered in patients taking antipsychotic medication or other centrally acting dopamine agonists.

Discuss (2) | Improve

Next question

Antipsychotics

Antipsychotics are a group of drugs used in the management of schizophrenia and other forms of psychosis, mania and agitation. They are usually divided into typical and atypical antipsychotics. The atypical antipsychotics were developed due to the problematic extrapyramidal side-effects which are associated with the first generation of typical antipsychotics.

	Typical antipsychotics	Atypical antipsychotics
Mechanism of action	Dopamine D2 receptor antagonists, blocking dopaminergic transmission in the mesolimbic pathways	Act on a variety of receptors (D2, D3, D4, 5-HT)
Adverse effects	Extrapyramidal side-effects and hyperprolactinaemia common	Extrapyramidal side-effects and hyperprolactinaemia less common Metabolic effects
Examples	Haloperidol Chlorpromazine	Clozapine Risperidone Olanzapine

The rest of this section will focus on typical antipsychotics, with atypical antipsychotics covered elsewhere.

Extrapyramidal side-effects (EPSEs)
- Parkinsonism
- acute dystonia
  - sustained muscle contraction (e.g. torticollis, oculogyric crisis)
  - may be managed with procyclidine
- akathisia (severe restlessness)
- tardive dyskinesia (late onset of choreoathetoid movements, abnormal, involuntary, may occur in 40% of patients, may be irreversible, most common is chewing and pouting of jaw)

The Medicines and Healthcare products Regulatory Agency has issued specific warnings when antipsychotics are used in elderly patients:

- increased risk of stroke
- increased risk of venous thromboembolism

Other side-effects

- antimuscarinic: dry mouth, blurred vision, urinary retention, constipation
- sedation, weight gain
- raised prolactin
  - may result in galactorrhoea
  - due to inhibition of the dopaminergic tuberoinfundibular pathway
- impaired glucose tolerance
- neuroleptic malignant syndrome: pyrexia, muscle stiffness
- reduced seizure threshold (greater with atypicals)
- prolonged QT interval (particularly haloperidol)

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 1 👎 0

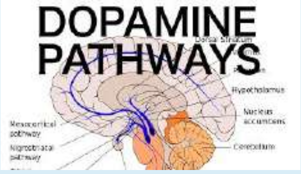
Schizophrenia > Prescribing Information > Adverse effects

BNF 👍 1 👎 2

Psychoses and related disorders

Suggest link Report broken link

Media

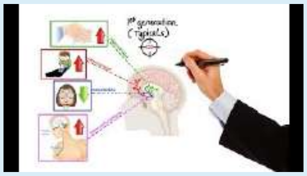

DOPAMINE PATHWAYS
Dorsal Striatum
Hypothalamus
Nucleus accumbens
Mesocortical pathway
Nigrostriatal pathway
Cerebellum

Antipsychotics, Dopamine, and EPS

Dirty USMLE - YouTube 👍 2 👎 0

Video of patients with tardive dyskinesia

Abnormal Involuntary Movement Scale training video - YouTube 👍 0 👎 0


1st generation (Typicals)

Antipsychotics

Speed Pharmacology - YouTube 👍 1 👎 1

Report broken media

Score: **16%**

1 ✗
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3 ✗
4 ✓
5 ✗
6 ✗

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33	-

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A 72-year-old man presents to the emergency department. He had experienced slurring of his speech and right-sided facial drooping earlier that day following his lunch. He reports that the symptoms were mostly resolved ten minutes following their onset but took another five minutes to fully resolve.

On examination, there is no residual dysphasia or a facial droop and he has a normal neurological examination.

What imaging modality would be preferred for this patient?

- ○ CT angiogram head
- ○ Diffusion-weighted MRI head
- ○ Fluid attenuation inversion recovery (FLAIR) MRI head
- ○ T1-weighted MRI head
- ○ T2-weighted MRI head

Submit answer

Reference ranges

Score: **0%**

1 -
2 -
3 -
4 -
5 -

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A 72-year-old man presents to the emergency department. He had experienced slurring of his speech and right-sided facial drooping earlier that day following his lunch. He reports that the symptoms were mostly resolved ten minutes following their onset but took another five minutes to fully resolve.

On examination, there is no residual dysphasia or a facial droop and he has a normal neurological examination.

What imaging modality would be preferred for this patient?

CT angiogram head	12%
Diffusion-weighted MRI head	68%
Fluid attenuation inversion recovery (FLAIR) MRI head	10%
T1-weighted MRI head	3%
T2-weighted MRI head	8%

MRI brain with diffusion-weighted imaging is the preferred modality in patients with suspected TIA who require brain imaging

Important for me   Less important

The patient has focal neurological symptoms that lasted for only 10 minutes, consistent with a transient ischemic attack. The preferred imaging modality for TIA is a **diffusion-weighted MRI head** which is very sensitive to small ischaemic lesions.

CT angiogram is most commonly used to image an acute ischaemic stroke (where symptoms are ongoing). It is useful as a quick method to detect an occlusion which can then be treated. It is also used to detect intracranial aneurysms when subarachnoid hemorrhage is suspected.

FLAIR MRI head is a T2 weighted technique that dampens CSF signal and can be a superior method of detecting a white matter brain lesion and can be used to assess for leptomeningeal diseases.

T1-weighted MRI head is a sequence of MRI that provides the best contrast for paramagnetic contrast agents such as gadolinium-containing compounds. The sequence also is good for demonstrating anatomy and shows white matter as bright whilst fluid-filled compartments appear dark. It would not be the most effective imaging sequence to investigate a TIA.

T2-weighted MRI head is a sequence of MRI that lights up fluid-filled compartments, whilst white matter appears dark. Hence lesions that are associated with increased water content such as oedema will show up bright making this a good sequence to detect these lesions. It again would not be the most effective imaging sequence to investigate a TIA.

👍 👎 Discuss (2) Improve

Next question >

Transient ischaemic attack ★

A transient ischaemic attack (TIA), as the name suggests, is a brief period of neurological deficit due to a vascular cause, typically lasting less than an hour. The National Clinical Guideline for Stroke was published in 2023 and made a number of updated recommendations, including the use of dual antiplatelet therapy (DAPT). It should be noted that DAPT is not currently recommended for 'major' ischaemic stroke as the risk of haemorrhagic transformation is too high.

The original definition of a TIA was time-based: a sudden onset of a focal neurologic symptom and/or sign lasting less than 24 hours, brought on by a transient decrease in blood flow. However, this has now changed as it is recognised that even short periods of ischaemia can result in pathological changes to the brain. Therefore, a new '**tissue-based**' definition is now used: a transient episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischaemia, without acute infarction. REF

Patients often use the term 'mini-stroke' for TIAs.

Clinical features

The clinical features are similar to those of a stroke, i.e. sudden onset, focal neurological deficit but, rather than persisting, the features resolve, typically within 1 hour.

Possible features include

- unilateral weakness or sensory loss.
- aphasia or dysarthria

- ataxia, vertigo, or loss of balance
- visual problems
    - sudden transient loss of vision in one eye (amaurosis fugax)
    - diplopia
    - homonymous hemianopia

Assessment and referral

Patients with acute focal neurological symptoms that resolve completely within 24 hours of onset (i.e. suspected TIA) should:
- be given aspirin 300 mg immediately unless contraindicated
- assessed urgently within 24 hours by a stroke specialist clinician

Examples of TIA mimics that require exclusion
- hypoglycaemia
- intracranial haemorrhage
    - all patients on anticoagulants or with similar risk factors should be admitted for urgent imaging to exclude haemorrhage

If a patient presents more than 7 days ago they should be seen by a stroke specialist clinician as soon as possible within 7 days.

The ABCD2 prognostic score has previously been used to risk stratify patients who present with a suspected TIA. However, data from studies have suggested it performs poorly and it is therefore no longer recommended.

Patients with suspected TIA should be assessed by a stroke specialist clinician before a decision on brain imaging NICE
- NICE recommend that CT brains should not be done '*unless there is clinical suspicion of an alternative diagnosis that CT could detect*'
    - an example exception would be when there is a concern about haemorrhage as the patient is taking anticoagulants
- MRI (including diffusion-weighted and blood-sensitive sequences) is preferred to determine the territory of ischaemia, or to detect haemorrhage or alternative pathologies
    - it should be done on the same day as the specialist assessment if possible

Management of TIA

It should be remembered that patients who've had a TIA are at high risk of further vascular events, particularly in the first few days. This section covers management following a diagnosis, i.e. after being seen by a stroke specialist clinician.

Medication

Immediate antithrombotic therapy: REF
- patients with TIA or minor ischaemic stroke should be given antiplatelet therapy provided there is neither a contraindication nor a high risk of bleeding
- for patients within 24 hours of onset of TIA or minor ischaemic stroke and with a low risk of bleeding, the following DAPT regimes should be considered:
  - clopidogrel (initial dose 300 mg followed by 75 mg od) + aspirin (initial dose 300 mg followed by 75 mg od for 21 days) followed by monotherapy with clopidogrel 75 mg od
  - ticagrelor + clopidogrel is an alternative
- if not appropriate for DAPT:
  - clopidogrel 300 mg loading dose followed by 75 mg od should be given
- proton pump inhibitor therapy should be considered for DAPT

The different antithrombotic recommendations may seem confusing - it is helpful to think of 3 different phases for a typical patient:

Resolved TIA symptoms, awaiting specialist review within 24 hours	**Reviewed by specialist, initial 21 days when at high risk of further events**	**Long-term secondary prevention after 21 days**
Aspirin	Aspirin + Clopidogrel	Clopidogrel

If a patient has atrial fibrillation they should be anticoagulated as soon as intracranial haemorrhage has been excluded.

Lipid modification
- high-intensity statin (such as atorvastatin 20-80 mg daily) - the aim of statin therapy is to reduce non-HDL cholesterol by more than 40%

Further investigation

Carotid imaging
- atherosclerosis in the carotid artery may be a source of emboli in some patients
- patients who are considered candidates for carotid intervention should have carotid imaging performed within 24 hours of assessment
  - carotid duplex ultrasound or either CT angiography or MR angiography
- carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled NICE

    - should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

+ it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

+ Q 123

Next question >

B *I*

Textbooks

High-yield textbook

Extended textbook

Links

RCP 👍 13 👎 13

2016 Stroke guidelines

National Clinical Guideline for Stroke 👍 7 👎 3

2023 National Clinical Guideline for Stroke

NICE 👍 17 👎 6

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Clinical Knowledge Summaries 👍 8 👎 3

Transient ischaemic attack

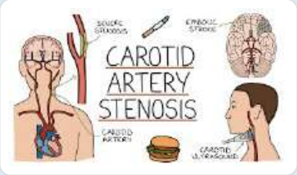
Radiopaedia 👍 7 👎 1

Transient ischaemic attack

Suggest link

Report broken link

Media


SEVERE STENOSIS
EMBOLIC STROKE
CAROTID ARTERY STENOSIS
CAROTID ARTERY
CAROTID ULTRASOUND

Understanding Carotid Artery Stenosis

Zero To Finals - YouTube 👍 2 👎 1

Report broken media

Score: **16%**

1	✗
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A 57-year-old woman is seen on the acute medical take with a worsening frontal headache, nausea and distal right upper limb pins and needles.

She was born in India and emigrated 3 years ago to the United Kingdom (UK). Her past medical history includes tuberculosis treated 10 years ago and rheumatoid arthritis for which she has recently been started on methotrexate and sulfasalazine alongside a short course of oral prednisolone. Her chest X-ray was normal and quantiFERON test negative on entry to the UK.

On examination, she looks well and is alert and orientated. Her temperature is 36.8ºC, oxygen saturation 97% on air, heart rate 80 beats per minute, respiratory rate 18 breaths per minute and blood pressure 143/75 mmHg. There is symmetrical swelling in the small joints in both hands and neurological examination reveals 4/5 MRC grade of power in the distal right upper limb. Examination of the cardiovascular, respiratory and abdominal system reveals no additional abnormalities.

Her investigation results are as follows:

Hb	124 g/L
Platelets	398* 10^9/L (150 - 400)
WBC	12 * 10^9/L (4.0 - 11.0)
Na	135 mmol/L (135 - 145)
K	3.6 mmol/L (3.5 - 5.0)
Urea	5 mmol/L (2.0 - 7.0)
Creatinine	63 µmol/L (55 - 120)
CRP	24 (< 5)

You arrange a contrast CT head which shows the following- A 3-4cm diameter homogeneous contrast enhancing round lesion adjacent to the meningeal membrane situated in the left frontal lobe with evidence of surrounding oedema and mass effect. No additional lesions noted. No evidence of acute ischemia, haemorrhage or collection.

What is the most likely diagnosis?

○ Tuberculosis ×

○ Aspergilloma ×

○ Meningioma ×

○ Neuroblastoma ×

○ Lung metastasis ×

Submit answer

Reference ranges

Score: **0%**

1 -
2 -
3 -
4 -
5 -
6 -

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What is the most likely diagnosis?

Tuberculosis	16%

Aspergilloma	11%
Meningioma	68%
Neuroblastoma	4%
Lung metastasis	2%

Meningioma primary brain tumors show contrast enhancement on a CT scan

Important for me Less important

The key to this question is the interpretation of the CT head findings. A meningioma will show homogeneous contrast enhancement on a CT scan.

The description and location of this space occupying lesion fit better with a meningioma than neuroblastoma.

Similarly, you may expect local or systemic signs and symptoms with metastatic lung cancer and CT findings fits better with the diagnosis of a meningioma. However, a screening CT scan of the chest, abdomen and pelvis may be appropriate to rule out this possibility.

We are told that this lady had active tuberculosis while living in India. However, on entry to the UK, there was no evidence of an active infection or a latent infection which could have been re-activated when initiated on immunosuppressive medications.

Although aspergilloma would be a risk with immunosuppression, this would be rarer and the CT head findings are more concordant with the diagnosis of a meningioma.

Discuss (7) Improve

Next question >

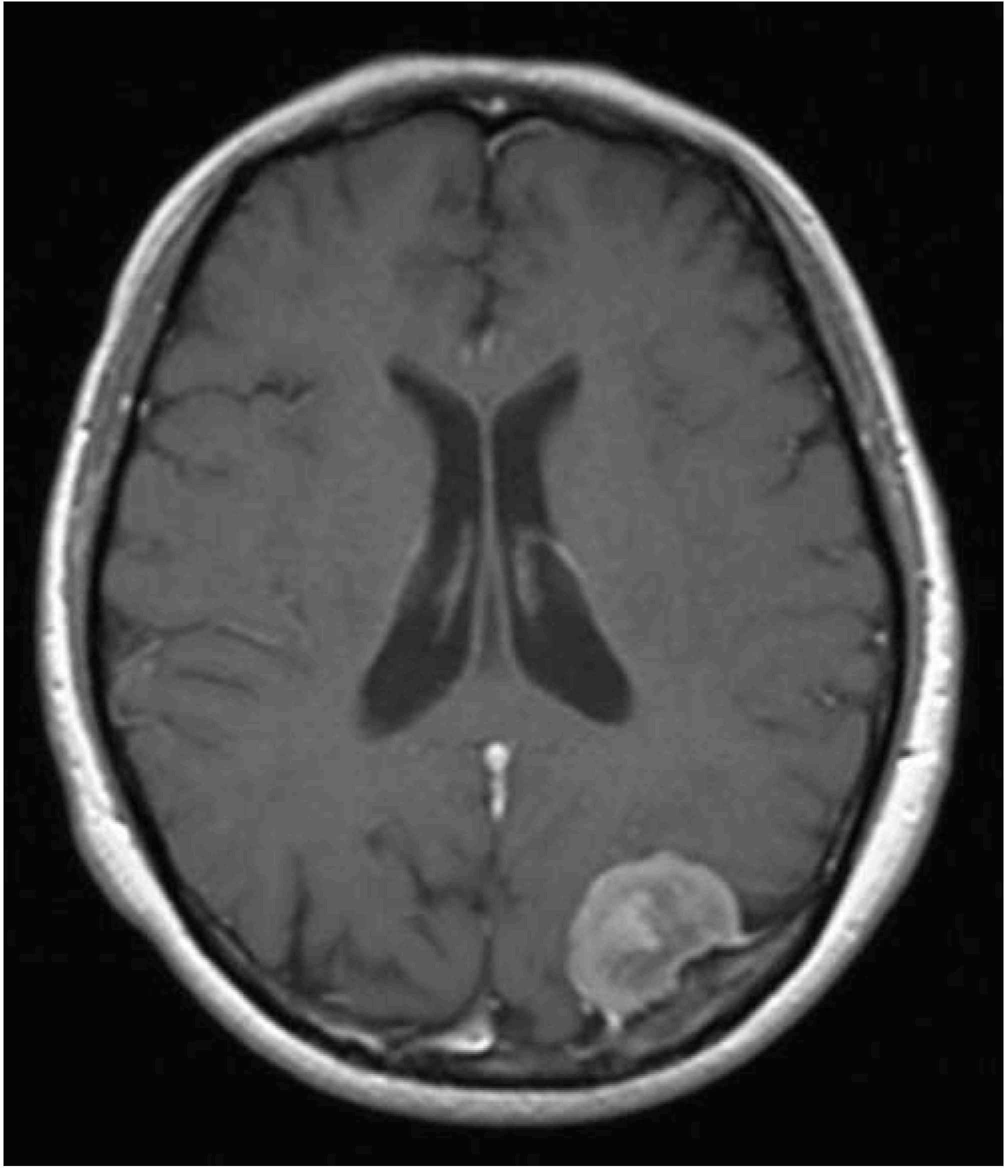
Brain tumours

The majority of adult tumours are supratentorial, where as the majority of childhood tumours are infratentorial.

Type of tumour	**Features**
Metastases	Metastatic brain cancer is the most common form of brain tumours. They are often multiple and not treatable with surgical intervention. Tumours that most commonly spread to the brain include: • lung (most common) • breast • bowel • skin (namely melanoma) • kidney
Glioblastoma multiforme	• Glioblastoma is the most common primary tumour in adults and is associated with a poor prognosis (~ 1yr). • On imaging they are solid tumours with central necrosis and a rim that enhances with contrast. Disruption of the blood-brain barrier and therefore are associated with vasogenic oedema. • Histology: Pleomorphic tumour cells border necrotic areas • Treatment is surgical with postoperative chemotherapy and/or radiotherapy. Dexamethasone is used to treat the oedema.
Meningioma	• The second most common primary brain tumour in adults • Meningiomas are typically benign, extrinsic tumours of the central nervous system. They arise from the arachnoid cap cells of the meninges and are typically located next to the dura and cause symptoms by compression rather than invasion. • They typically are located at the falx cerebri, superior sagittal sinus, convexity or skull base. • Histology: Spindle cells in concentric whorls and calcified psammoma bodies • Investigation is with CT (will show contrast enhancement) and MRI, and treatment will involve either observation, radiotherapy or surgical resection.
Vestibular schwannoma	• A vestibular schwannoma (previously termed acoustic neuroma) is a benign tumour arising from the eighth cranial nerve (vestibulocochlear nerve). Often seen in the cerebellopontine angle. It presents with

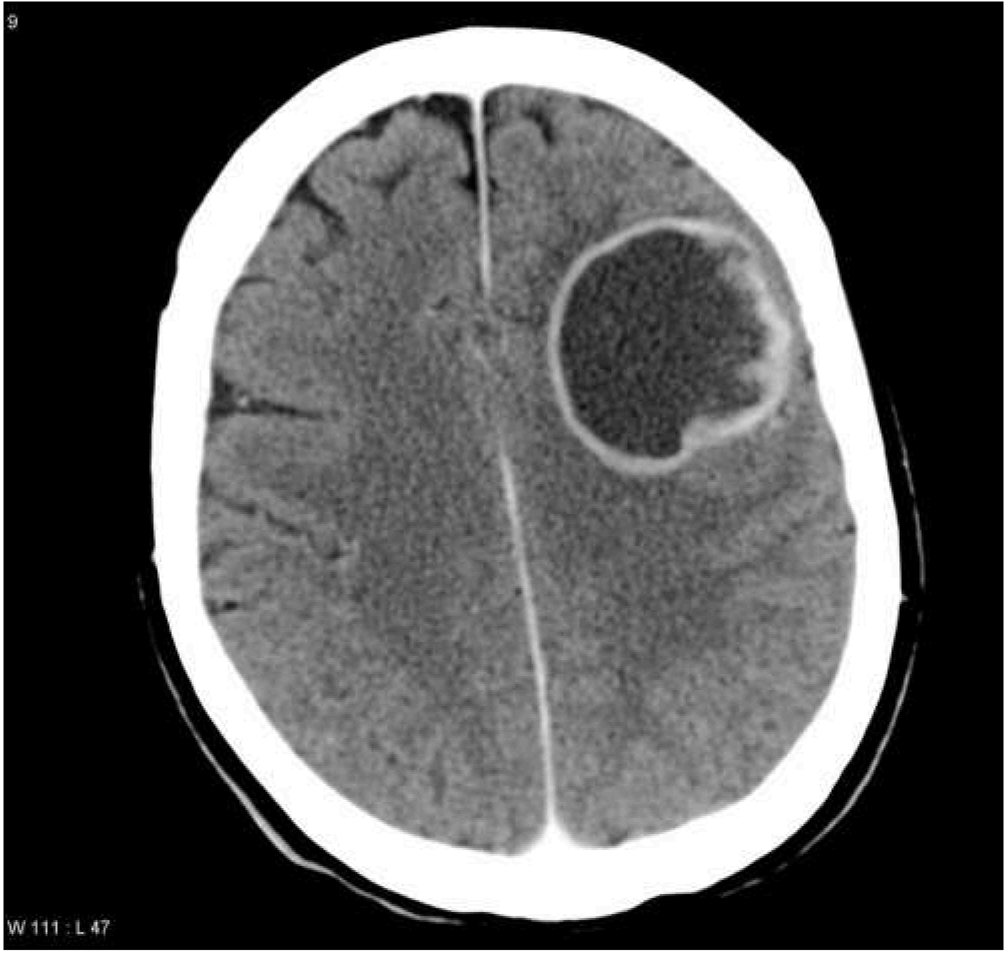
Type of tumour	Features
	hearing loss, facial nerve palsy (due to compression of the nearby facial nerve) and tinnitus. • Neurofibromatosis type 2 is associated with bilateral vestibular schwannomas. • Histology: Antoni A or B patterns are seen. Verocay bodies (acellular areas surrounded by nuclear palisades) • Treatment may involve observation, radiotherapy or surgery.
Pilocytic astrocytoma	• The most common primary brain tumour in children • Histology: Rosenthal fibres (corkscrew eosinophilic bundle)
Medulloblastoma	• A medulloblastoma is an aggressive paediatric brain tumour that arises within the infratentorial compartment. It spreads through the CSF system. Treatment is surgical resection and chemotherapy. • Histology: Small, blue cells. Rosette pattern of cells with many mitotic figures
Ependymoma	• Commonly seen in the 4th ventricle • May cause hydrocephalus • Histology: perivascular pseudorosettes
Oligodendroma	• Benign, slow-growing tumour common in the frontal lobes • Histology: Calcifications with 'fried-egg' appearance
Haemangioblastoma	• Vascular tumour of the cerebellum • Associated with von Hippel-Lindau syndrome • Histology: foam cells and high vascularity

Type of tumour	Features
Pituitary adenoma	• Pituitary adenomas are benign tumours of the pituitary gland. They are either secretory (producing a hormone in excess) or non-secretory. They may be divided into microadenomas (smaller than 1cm) or macroadenoma (larger than 1cm). • Patients will present with the consequences of hormone excess (e.g. Cushing's due to ACTH, or acromegaly due to GH) or depletion. Compression of the optic chiasm will cause a bitemporal hemianopia due to the crossing nasal fibers. • Investigation requires a pituitary blood profile and MRI. Treatment can either be hormonal or surgical (e.g. transphenoidal resection).
Craniopharyngioma	• Most common paediatric supratentorial tumour • A craniopharyngioma is a solid/cystic tumour of the sellar region that is derived from the remnants of Rathke's pouch. It is common in children, but can present in adults also. It may present with hormonal disturbance, symptoms of hydrocephalus or bitemporal hemianopia. • Histology: Derived from remnants of Rathke pouch • Investigation requires pituitary blood profile and MRI. Treatment is typically surgical with or without postoperative radiotherapy.



© Image used on license from Radiopaedia

Meningioma - MRI showing the typical well-circumscribed appearance. A dural tail can be where the tumour 'connects' to the dura. It is seen in around 65% of meningiomas.



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Glioblastoma multiforme - CT showing a peripherally enhancing lesion within the left frontal lobe. Note the contrast to the more homogenous meningioma above.

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

ADULT
BRAIN TUMORS
Osmosis.org

Adult brain tumours

Osmosis - YouTube 👍 4 👎 0

GLIOBLASTOMA
OSMOSIS KNOWLEDGE SHOT

Glioblastoma

Osmosis - YouTube 👍 2 👎 1

PEDIATRIC
BRAIN TUMORS
Osmosis.org

Paediatric brain tumours

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **16%**

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A 56-year-old man is brought into the emergency department of a major stroke unit by his wife who reports that for the last 2-hours her husband has been unable to move his right side and is struggling to speak. He is known to have type 2 diabetes mellitus and hypercholesterolemia, for which he takes metformin and atorvastatin respectively. In addition, he has had a recent provoked lower limb deep vein thrombosis (DVT) for which he was started on apixaban. He works as an accountant, is an ex-smoker and drinks occasionally.

On examination, there is a dense right hemiparesis with expressive dysphasia. He scores 22 on the National Institute of Health Stroke Score (NIHSS).

Following rapid assessment, a CT brain and CT intracranial angiogram are performed, showing a proximal occlusion within the M2 segment of the middle cerebral artery with no major new ischaemic changes. An MRI head with MR angiography confirms these appearances.

Given this patient's presentation, which of the following would be the best intervention?

- ○ Thrombolysis immediately ×
- ○ Thrombectomy within 6 hours ×
- ○ Thrombolysis within 6 hours ×
- ○ Thrombectomy within 12 hours ×
- ○ Thrombolysis within 12 hours ×

Submit answer

Reference ranges

Score: **0%**

1 -

2 -
3 -
4 -
5 -
6 -
7 -

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Given this patient's presentation, which of the following would be the best intervention?

Thrombolysis immediately	45%
Thrombectomy within 6 hours	40%
Thrombolysis within 6 hours	3%
Thrombectomy within 12 hours	12%
Thrombolysis within 12 hours	0%

The standard target time for thrombectomy in acute ischaemic stroke is 6 hours

Important for me Less important

This patient meets the criteria for a mechanical thrombectomy:

- thrombectomy can be achieved within 6 hours of symptom onset (if this cannot be achieved, consideration for thrombectomy is given in patients with sufficient salvageable brain tissue identified on imaging for up to 24 hours after symptom onset, although this is assessed on a case-by-case basis).
- he is anticoagulated, thus thrombolysis is contraindicated
- Imaging demonstrates a proximal occlusion
- There are no new major ischaemic changes on imaging

- his NIHSS is more than 5
- he was independent prior to symptom onset

👍 👎 Discuss (7) Improve

Next question >

Stroke: management of acute ischaemic stroke ★

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:
- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
  - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
  - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:

- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.

- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
    - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
    - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 11 👎 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 16

Stroke and TIA guidelines

Age and Ageing 👍 21 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

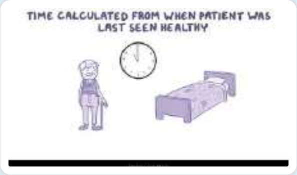
2023 National Clinical Guideline for Stroke

NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia
Acute Ischaemic Stroke

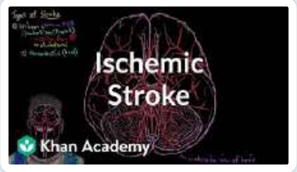
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

Khan Academy - YouTube

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Score: **16%**

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You are asked to see a 57 year-old female who has presented acutely to eye clinic with deteriorating vision. On questioning she has experienced sudden blurring of the temporal field in her right eye. Her past medical history is noteworthy for hypertension and type 2 diabetes mellitus.

What is the likely diagnosis?

- ○ Branch retinal artery occlusion
- ○ Nasal retinal detachment
- ○ Central retinal vein occlusion
- ○ Temporal branch retinal vein occlusion
- ○ Nasal branch retinal vein occlusion

Submit answer

Reference ranges

Score: **0%**

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You are asked to see a 57 year-old female who has presented acutely to eye clinic with deteriorating vision. On questioning she has experienced sudden blurring of the temporal field in her right eye. Her past medical history is noteworthy for hypertension and type 2 diabetes mellitus.

What is the likely diagnosis?

Branch retinal artery occlusion	18%
Nasal retinal detachment	14%
Central retinal vein occlusion	6%
Temporal branch retinal vein occlusion	20%
Nasal branch retinal vein occlusion	42%

This case describes visual blurring and not total visual loss. Retinal artery occlusions lead to amaurosis fugax, often describes as a 'black curtain' descending over the vision. The most likely explanation for visual blurring is a retinal vein occlusion. The temporal field corresponds to the nasal retina. Both hypertension and diabetes are risk factors for a retinal vein occlusion.

Discuss (5) Improve

Next question >

Sudden loss of vision

Sudden loss is a frightening symptom for patients. It may represent an ongoing issue or only be temporary. The term transient monocular visual loss (TMVL) describes a sudden, transient loss of vision that lasts less than 24 hours.

The most common causes of a sudden painless loss of vision are as follows:

- ischaemic/vascular (e.g. thrombosis, embolism, temporal arteritis etc). This includes recognised syndromes e.g. occlusion of central retinal vein and occlusion of central retinal artery

- vitreous haemorrhage
- retinal detachment
- retinal migraine

Ischaemic/vascular

- often referred to as 'amaurosis fugax'
- wide differential including large artery disease (atherothrombosis, embolus, dissection), small artery occlusive disease (anterior ischemic optic neuropathy, vasculitis e.g. temporal arteritis), venous disease and hypoperfusion
- may represent a form of transient ischaemic attack (TIA). It should therefore be treated in a similar fashion, with aspirin 300mg being given
- altitudinal field defects are often seen: 'curtain coming down'
- ischaemic optic neuropathy is due to occlusion of the short posterior ciliary arteries, causing damage to the optic nerve

Central retinal vein occlusion

- incidence increases with age, more common than arterial occlusion
- causes: glaucoma, polycythaemia, hypertension
- severe retinal haemorrhages are usually seen on fundoscopy

Central retinal artery occlusion

- due to thromboembolism (from atherosclerosis) or arteritis (e.g. temporal arteritis)
- features include afferent pupillary defect, 'cherry red' spot on a pale retina

Vitreous haemorrhage

- causes: diabetes, bleeding disorders, anticoagulants
- features may include sudden visual loss, dark spots

Retinal detachment

- features of vitreous detachment, which may precede retinal detachment, include flashes of light or floaters (see below)

Differentiating posterior vitreous detachment, retinal detachment and vitreous haemorrhage

Posterior vitreous detachment	**Retinal detachment**	**Vitreous haemorrhage**
Flashes of light (photopsia) - in the peripheral field of vision Floaters, often on the temporal side of the central vision	Dense shadow that starts peripherally progresses towards the central vision A veil or curtain over the field of vision Straight lines appear curved Central visual loss	Large bleeds cause sudden visual loss Moderate bleeds may be described as numerous dark spots Small bleeds may cause floaters

123

Next question

Textbooks

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Extended textbook

Links

The Royal Australian College of General Practitioners 16 20

Sudden loss of vision review

Patient.info 14 11

Retinal artery occlusions

American Academy of Ophthalmology 16 10

Central Retinal Vein Occlusion

American Academy of Ophthalmology 👍 15 👎 7

Central Retinal Artery Occlusion

Suggest link Report broken link

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A 55-year-old man presents to the emergency department with a four-hour history of an acutely painful left eye. He also describes photophobia and reduced visual acuity associated with the pain.

On examination, diffuse redness is noted throughout the left eye. The left pupil is smaller than the left and he is unable to count fingers in the left eye although visual acuity in the right eye is unimpaired. It is also noted that there is some pus in the anterior chamber.

What treatment is most likely to be indicated?

○ Intravenous antibiotics ×

○ Intravenous diuretic ×

○ Steroid + mydriatic eye drops ×

○ Topical antibiotics ×

○ Topical beta blocker ×

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Reference ranges

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Question 9 of 299 ✗

A 55-year-old man presents to the emergency department with a four-hour history of an acutely painful left eye. He also describes photophobia and reduced visual acuity associated with the pain.

On examination, diffuse redness is noted throughout the left eye. The left pupil is smaller than the left and he is unable to count fingers in the left eye although visual acuity in the right eye is unimpaired. It is also noted that there is some pus in the anterior chamber.

What treatment is most likely to be indicated?

Intravenous antibiotics	23%
Intravenous diuretic	3%
Steroid + mydriatic eye drops	50%
Topical antibiotics	17%
Topical beta blocker	6%

Anterior uveitis is most likely to be treated with a steroid + cycloplegic (mydriatic) drops

Important for me Less important

This man has presented with a case of anterior uveitis, as confirmed by the typical findings on examination. As per NICE guidance, the management of anterior uveitis is dependant on whether an infective or non-infective cause is established. As a result, all cases must be diagnosed and managed by ophthalmology in secondary care. The most common therapy is steroid + mydriatic drops and therefore this is the correct answer.

Intravenous antibiotics may be indicated in cases of infective anterior uveitis as directed by ophthalmology however treatment is more likely to be topical steroids and cycloplegics.

Carbonic anhydrase diuretics (for example acetazolamide) are of use in acute angle-closure glaucoma however as this is not the diagnosis here, this answer is incorrect.

Topical antibiotics again may be indicated in infective cases but are not the most likely treatment.

Topical beta-blockers are again indicated in cases of acute angle-closure glaucoma which is not the diagnosis here.

👍 👎 Discuss (5) Improve

Next question >

Anterior uveitis ★

Anterior uveitis is one of the important differentials of a red eye. It is also referred to as iritis. Anterior uveitis describes inflammation of the anterior portion of the uvea - iris and ciliary body. It is associated with HLA-B27 and may be seen in association with other HLA-B27 linked conditions (see below).

Features
- acute onset
- ocular discomfort & pain (may increase with use)
- pupil may be small +/- irregular due to sphincter muscle contraction
- photophobia (often intense)
- blurred vision
- red eye
- lacrimation
- ciliary flush: a ring of red spreading outwards
- hypopyon; describes pus and inflammatory cells in the anterior chamber, often resulting in a visible fluid level
- visual acuity initially normal → impaired

Associated conditions
- ankylosing spondylitis
- reactive arthritis
- ulcerative colitis, Crohn's disease
- Behcet's disease
- sarcoidosis: bilateral disease may be seen

Management
- urgent review by ophthalmology
- cycloplegics (dilates the pupil which helps to relieve pain and photophobia) e.g. Atropine, cyclopentolate

- steroid eye drops

123

Next question ›

Textbooks

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Score: **16%**

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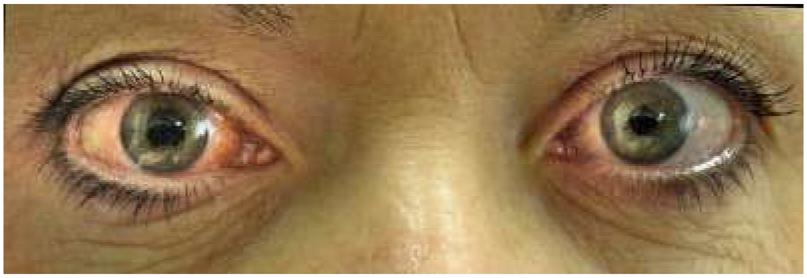
Question 10 of 299

A 48-year-old woman presents to the eye casualty department with several hours of blurred vision in her right eye, accompanied by seeing 'haloes' around bright lights. She also describes nausea and numerous episodes of vomiting.

On examination, the eye appears as shown below:



What is an appropriate initial choice of treatment?

○ Topical brinzolamide

○ Topical dexamethasone

○ Topical ganciclovir

○ Topical gentamicin

○ Topical latanoprost

Submit answer

Reference ranges

Score: **0%**

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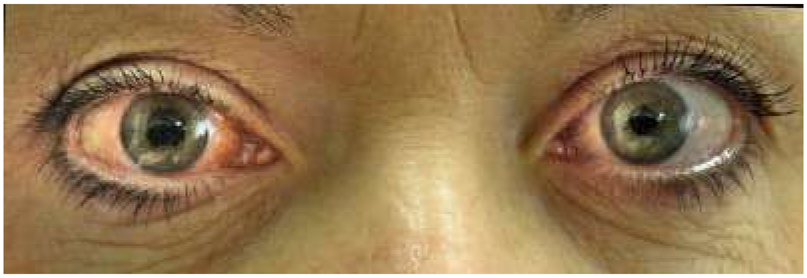


What is an appropriate initial choice of treatment?

Topical brinzolamide	42%
Topical dexamethasone	9%
Topical ganciclovir	2%
Topical gentamicin	2%
Topical latanoprost	45%

This patient's presentation of blurred vision, the description of 'haloes' around lights and nausea is suggestive of acute angle-closure glaucoma (AACG). This is supported by the photo of the right eye, showing erythema and a semi-dilated pupil, relative to the left eye. Immediate management involves a combination of eye drops, typically including a carbonic anhydrase inhibitor such as **topical brinzolamide**.

Topical dexamethasone is a corticosteroid used to reduce inflammation in a number of eye diseases. It does not routinely play a role in AACG.

Topical ganciclovir is used in the management of dendritic/herpetic keratitis. Rather than a fixed, dilated pupil, fluorescein staining would reveal a branching pattern.

Topical gentamicin is used to manage bacterial infections of the eye, such as bacterial keratitis. The pupil would not be fixed and dilated as seen here, but the eye would be generally red in appearance.

Topical latanoprost may be used to treat residual angle-closure glaucoma after laser peripheral iridotomy. It is not used in the acute setting.

👍 👎 Discuss (5) Improve

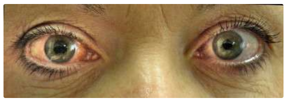
Next question >

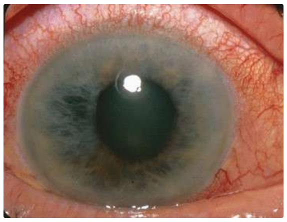
Acute angle-closure glaucoma ★

Glaucoma is a group of disorders characterised by optic neuropathy due, in the majority of patients, to raised intraocular pressure (IOP). It is now recognised that a minority of patients with raised IOP do not have glaucoma and vice versa.

In acute angle-closure glaucoma (AACG) there is a rise in IOP secondary to an impairment of aqueous outflow. Factors predisposing to AACG include:
- hypermetropia (long-sightedness)
- pupillary dilatation
- lens growth associated with age

Features
- severe pain: may be ocular or headache
- decreased visual acuity
- symptoms worse with mydriasis (e.g. watching TV in a dark room)
- hard, red-eye
- haloes around lights
- semi-dilated non-reacting pupil
- corneal oedema results in dull or hazy cornea
- systemic upset may be seen, such as nausea and vomiting and even abdominal pain





Investigations

- tonometry to assess for elevated IOP
- gonioscopy (literally looking, *oscopy*, at the angle, *gonio*): a special lens for the slit lamp that allows visualisation of the angle

Management

The management of AACG is an emergency and should prompt urgent referral to an ophthalmologist. Emergency medical treatment is required to lower the IOP with more definitive surgical treatment given once the acute attack has settled.

There are no guidelines for the initial medical treatment emergency treatment. An example regime would be:

- combination of eye drops, for example:
    - a direct parasympathomimetic (e.g. pilocarpine, causes contraction of the ciliary muscle → opening the trabecular meshwork → increased outflow of the aqueous humour)
    - a beta-blocker (e.g. timolol, decreases aqueous humour production)
    - an alpha-2 agonist (e.g. apraclonidine, dual mechanism, decreasing aqueous humour production and increasing uveoscleral outflow)
- intravenous acetazolamide
    - reduces aqueous secretions
- some guidelines also recommend the use of topical steroids to reduce inflammation

Definitive management

- laser peripheral iridotomy
    - creates a tiny hole in the peripheral iris → aqueous humour flowing to the angle

Next question >

Textbooks

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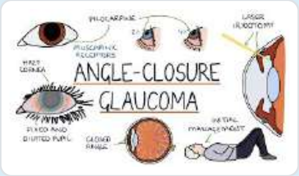
Links

Patient.info 👍 26 👎 9

Acute angle closure glaucoma review

Suggest link Report broken link

Media


ANGLE-CLOSURE
GLAUCOMA

Acute angle closure glaucoma

Zero To Finals - YouTube 👍 4 👎 0

GLAUCOMA
Osmosis.org

Glaucoma (open-angle, closed-angle, and normal-tension)

Osmosis - YouTube 👍 6 👎 3

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Score: **16%**

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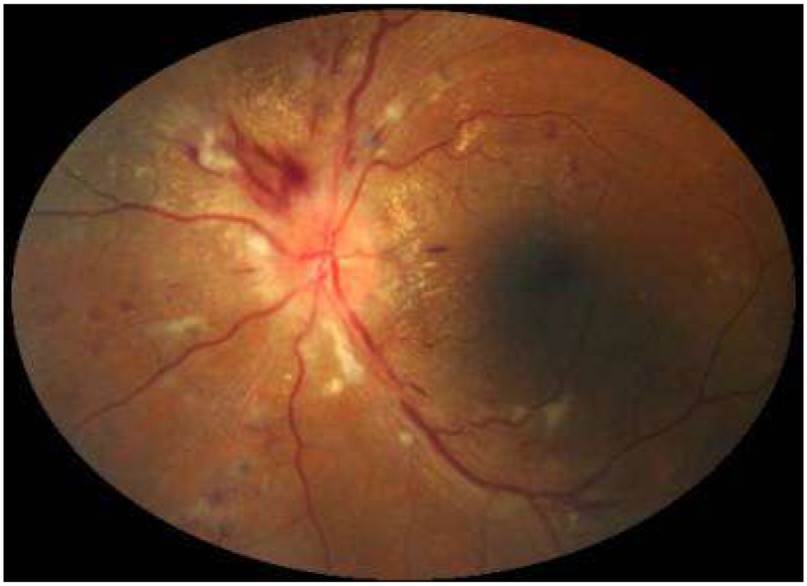
Question 11 of 299

A 76-year-old man is routinely reviewed as part of a health checkup. During this process, his eyes are examined using retinal photography and the following image is noted:



He reports generally reduced visual acuity and sometimes double vision.

He takes a number of medications, including amlodipine, losartan, salbutamol, metformin, paracetamol and codeine.

Given the photo above, what is the most likely diagnosis?

- ○ Central retinal artery occlusion
- ○ Central retinal vein occlusion
- ○ Diabetic retinopathy
- ○ Hypertensive retinopathy
- ○ Vitreous haemorrhage

Submit answer

Reference ranges

Score: **0%**

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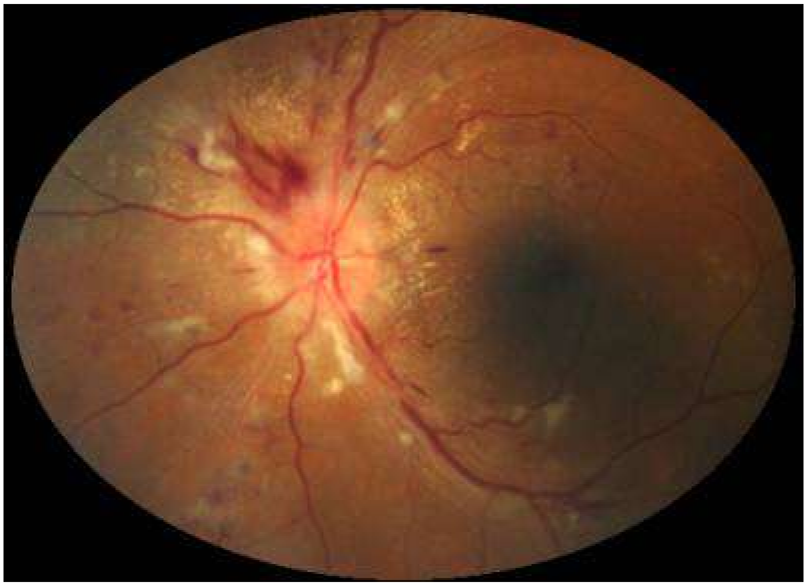
A 76-year-old man is routinely reviewed as part of a health checkup. During this process, his eyes are examined using retinal photography and the following image is noted:



He reports generally reduced visual acuity and sometimes double vision.

He takes a number of medications, including amlodipine, losartan, salbutamol, metformin, paracetamol and codeine.

Given the photo above, what is the most likely diagnosis?

Central retinal artery occlusion	9%
Central retinal vein occlusion	14%
Diabetic retinopathy	28%
Hypertensive retinopathy ✓	35%
Vitreous haemorrhage ✗	13%

The findings on the photo above are indicative of hypertensive retinopathy. The photo shows some generalised arterial narrowing and some evidence of silver wiring - a silvery/white appearance of retinal arterioles if they have become occluded. A flame haemorrhage is also visible in the top left area, as well as exudates scattered across the image. Together, these are all features of hypertensive retinopathy.

Central retinal artery occlusion classically presents with retinal whitening and a cherry-red spot - opacification of the nerve fibre layer as it becomes oedematous due to ischaemia, with the cherry-red spot being due to fovea sparing (no nerve fibre layer over the fovea).

Central retinal vein occlusion would also include features such as flame haemorrhages, as well as cotton-wool spots - fluffy white patches on the retina caused by damage to nerve fibres and the resulting accumulation of material within the nerve fibre layer. However, the other findings visible here, such as silver wiring and arteriovenous (AV) nicking, are more indicative of hypertensive retinopathy.

Diabetic retinopathy may have some similar findings as hypertensive retinopathy on fundoscopy and can include haemorrhages and cotton-wool spots. Hypertensive retinopathy can be distinguished by the presence of silver wiring and AV nicking, which are both present in this case. Furthermore, with advanced proliferative retinopathy, neovascularisation would be observed.

The haemorrhage visible in this case is a flame haemorrhage, rather than a vitreous haemorrhage. Vitreous haemorrhages appear as a well-defined accumulation of blood within the vitreous body, obscuring the retina, rather than on the retina itself. They normally present with a sudden, painless loss or blurring of vision.

👍 👎 Discuss (4) Improve

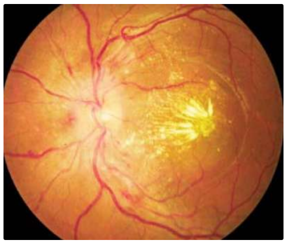
Next question >

Hypertensive retinopathy

The table below shows the Keith-Wagener classification of hypertensive retinopathy

Stage	Features
I	Arteriolar narrowing and tortuosity Increased light reflex - silver wiring
II	Arteriovenous nipping
III	Cotton-wool exudates Flame and blot haemorrhages • These may collect around the fovea resulting in a 'macular star'

Stage	Features
IV	Papilloedema



123

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Media

HYPERTENSIVE
RETINOPATHY

Hypertensive retinopathy stages

Last Second Medicine - YouTube 14 3

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Score: **16%**

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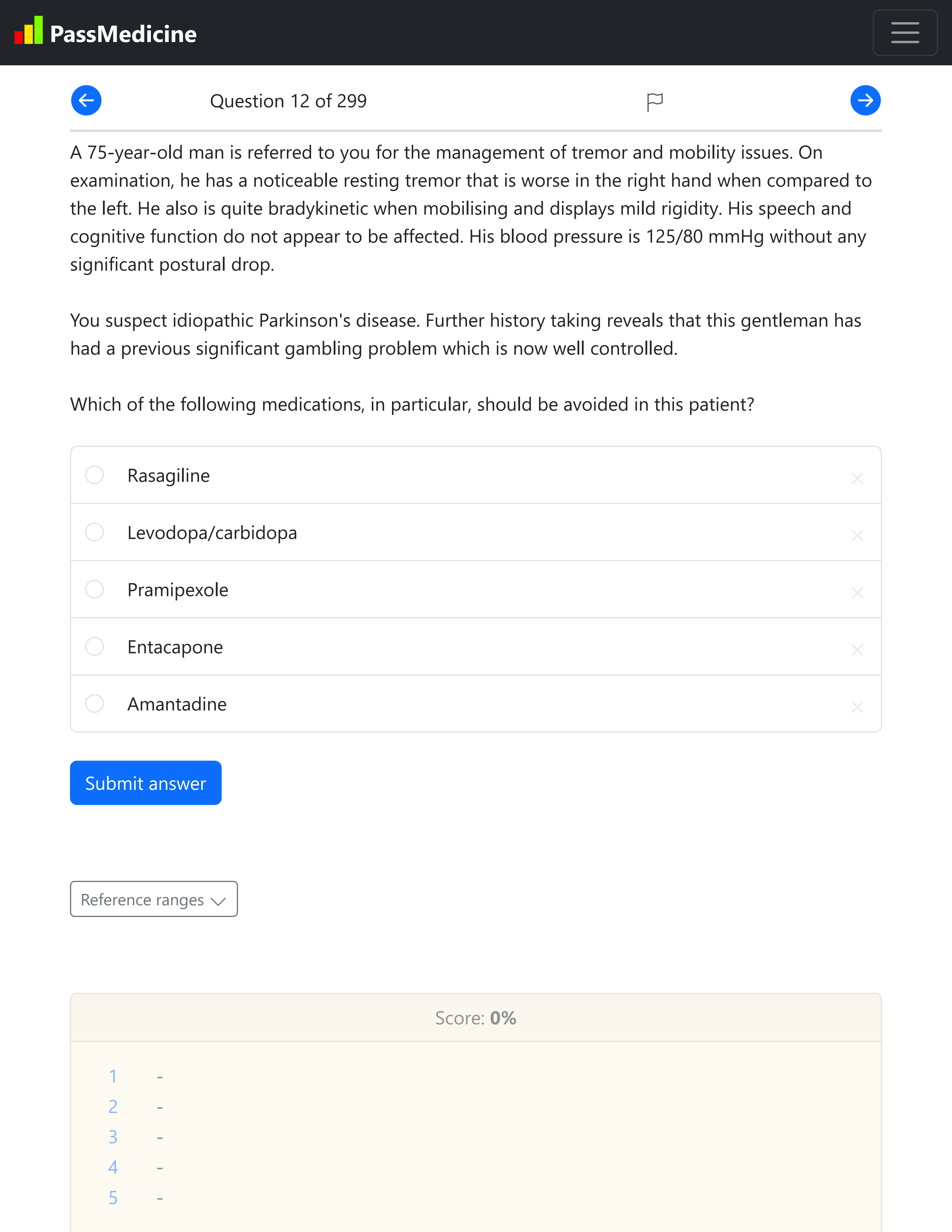

A 75-year-old man is referred to you for the management of tremor and mobility issues. On examination, he has a noticeable resting tremor that is worse in the right hand when compared to the left. He also is quite bradykinetic when mobilising and displays mild rigidity. His speech and cognitive function do not appear to be affected. His blood pressure is 125/80 mmHg without any significant postural drop.

You suspect idiopathic Parkinson's disease. Further history taking reveals that this gentleman has had a previous significant gambling problem which is now well controlled.

Which of the following medications, in particular, should be avoided in this patient?

○ Rasagiline

○ Levodopa/carbidopa

○ Pramipexole

○ Entacapone

○ Amantadine

Score: **0%**

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Question 12 of 299

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You suspect idiopathic Parkinson's disease. Further history taking reveals that this gentleman has had a previous significant gambling problem which is now well controlled.

Which of the following medications, in particular, should be avoided in this patient?

Rasagiline	18%
Levodopa/carbidopa	18%
Pramipexole	43%
Entacapone	11%
Amantadine	9%

There is an established link between the use of dopamine receptor agonists and serious impulse control disorders. For patients who have a history of habitual behaviours associated with increased risk, this group of medications should be avoided.

Discuss (3) Improve

Next question ›

Parkinson's disease: management ★

Parkinsons disease should only be diagnosed, and management initiated, by a specialist with expertise in movement disorders. However, it is important for all doctors to be aware of the medications used in Parkinson's given the prevalence of this condition. NICE published guidelines in 2017 regarding the management of Parkinson's disease.

For first-line treatment:

- if the motor symptoms are affecting the patient's quality of life: levodopa
- if the motor symptoms are not affecting the patient's quality of life: dopamine agonist (non-ergot derived), levodopa or monoamine oxidase B (MAO-B) inhibitor

Whilst all drugs used to treat Parkinson's can cause a wide variety of side-effects NICE produced tables to help with decision making:

	Levodopa	Dopamine agonists	MAO-B inhibitors
Motor symptoms	More improvement in motor symptoms	Less improvement in motor symptoms	Less improvement in motor symptoms
Activities of daily living	More improvement in activities of daily living	Less improvement in activities of daily living	Less improvement in activities of daily living
Motor complications	More motor complications	Fewer motor complications	Fewer motor complications
Adverse events	Fewer specified adverse events*	More specified adverse events*	Fewer specified adverse events*

* excessive sleepiness, hallucinations and impulse control disorders

If a patient continues to have symptoms despite optimal levodopa treatment or has developed dyskinesia then NICE recommend the addition of a dopamine agonist, MAO-B inhibitor or catechol-O-methyl transferase (COMT) inhibitor as an adjunct. Again, NICE summarise the main points in terms of decision making:

	Dopamine agonists	MAO-B inhibitors	COMT inhibitors	Amantadine
Motor symptoms	Improvement in motor symptoms	Improvement in motor symptoms	Improvement in motor symptoms	No evidence of improvement in motor symptoms
Activities of daily living	Improvement in activities of daily living	Improvement in activities of daily living	Improvement in activities of daily living	No evidence of improvement in activities of daily living
Off time	More off-time reduction	Off-time reduction	Off-time reduction	No studies reporting this

	Dopamine agonists	MAO-B inhibitors	COMT inhibitors	Amantadine
				outcome
Adverse events	Intermediate risk of adverse events	Fewer adverse events	More adverse events	No studies reporting this outcome
Hallucinations	More risk of hallucinations	Lower risk of hallucinations	Lower risk of hallucinations	No studies reporting this outcome

Specific points regarding Parkinson's medication

NICE reminds us of the risk of acute akinesia or neuroleptic malignant syndrome if medication is not taken/absorbed (for example due to gastroenteritis) and advise against giving patients a 'drug holiday' for the same reason.

Impulse control disorders have become a significant issue in recent years. These can occur with any dopaminergic therapy but are more common with:

- dopamine agonist therapy
- a history of previous impulsive behaviours
- a history of alcohol consumption and/or smoking

If excessive daytime sleepiness develops then patients should not drive. Medication should be adjusted to control symptoms. Modafinil can be considered if alternative strategies fail.

If orthostatic hypotension develops then a medication review looking at potential causes should be done. If symptoms persist then midodrine (acts on peripheral alpha-adrenergic receptors to increase arterial resistance) can be considered.

Consider glycopyrronium bromide to manage drooling of saliva in people with Parkinson's disease.

Further information regarding specific anti-Parkinson's medication

Levodopa

- nearly always combined with a decarboxylase inhibitor (e.g. carbidopa or benserazide)

    - this prevents the peripheral metabolism of levodopa to dopamine outside of the brain and hence can reduce side effects
- common adverse effects:
    - dry mouth
    - anorexia
    - palpitations
    - postural hypotension
    - psychosis
- some adverse effects are due to the difficulty in achieving a steady dose of levodopa
    - end-of-dose wearing off: symptoms often worsen towards the end of dosage interval. This results in a decline of motor activity
    - 'on-off' phenomenon: large variations in motor performance, with normal function during the 'on' period, and weakness and restricted mobility during the 'off' period
    - dyskinesias at peak dose: dystonia, chorea and athetosis (involuntary writhing movements)
    - these effects may worsen over time with - clinicians therefore may limit doses until necessary
- it is important not to acutely stop levodopa, for example, if a patient is admitted to hospital
    - if a patient with Parkinson's disease cannot take levodopa orally, they can be given a dopamine agonist patch as rescue medication to prevent acute dystonia

Dopamine receptor agonists

- e.g. bromocriptine, ropinirole, cabergoline, apomorphine
- ergot-derived dopamine receptor agonists (bromocriptine, cabergoline) have been associated with pulmonary, retroperitoneal and cardiac fibrosis. The Committee on Safety of Medicines advice that an echocardiogram, ESR, creatinine and chest x-ray should be obtained prior to treatment and patients should be closely monitored
- patients should be warned about the potential for dopamine receptor agonists to cause impulse control disorders and excessive daytime somnolence
- more likely than levodopa to cause hallucinations in older patients. Nasal congestion and postural hypotension are also seen in some patients

MAO-B (Monoamine Oxidase-B) inhibitors

- e.g. selegiline
- inhibits the breakdown of dopamine secreted by the dopaminergic neurons

Amantadine

- mechanism is not fully understood, probably increases dopamine release and inhibits its uptake at dopaminergic synapses
- side-effects include ataxia, slurred speech, confusion, dizziness and livedo reticularis

COMT (Catechol-O-Methyl Transferase) inhibitors

- e.g. entacapone, tolcapone
- COMT is an enzyme involved in the breakdown of dopamine, and hence may be used as an adjunct to levodopa therapy
- used in conjunction with levodopa in patients with established PD

Antimuscarinics

- block cholinergic receptors
- now used more to treat drug-induced parkinsonism rather than idiopathic Parkinson's disease
- help tremor and rigidity
- e.g. procyclidine, benzotropine, trihexyphenidyl (benzhexol)

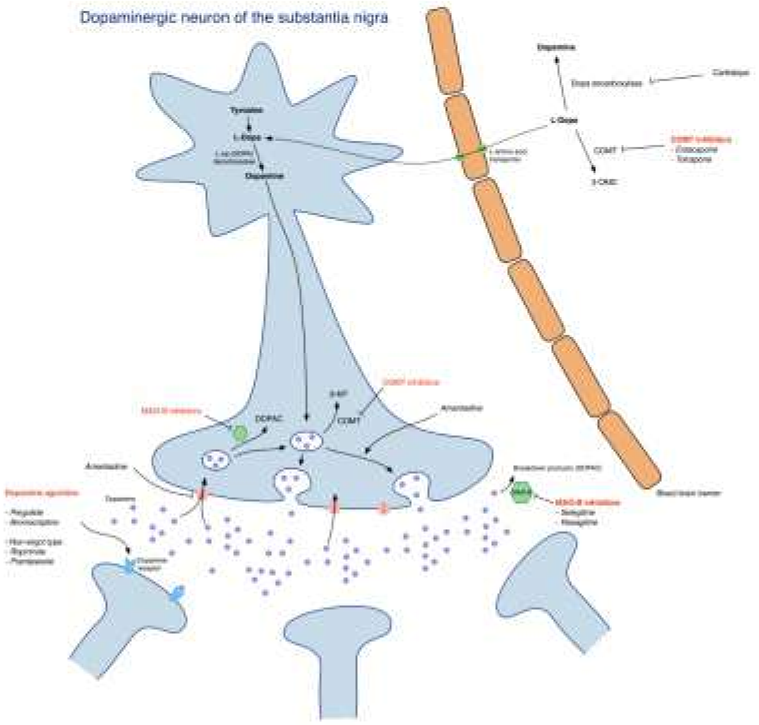

Dopaminergic neuron of the substantia nigra
Tyrosine
L-Dopa
Dopamine
Dopamine
L-Dopa
COMT
3-OMD
DOPAC
3-MT
COMT
Amantadine
Amantadine

Image sourced from Wikipedia

Diagram showing the mechanism of action of Parkinson's drugs

Next question

Textbooks

High-yield textbook

Extended textbook

Links

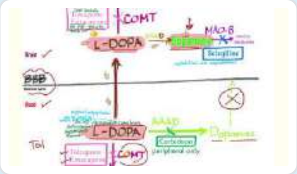
NICE 👍 23 👎 12

2017 Parkinson's disease guidelines

Suggest link

Report broken link

Media


COMT
L-DOPA
MAO-B
L-DOPA
COMT

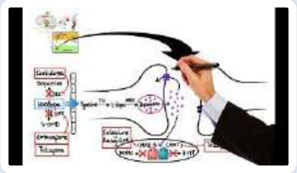
The use of Levodopa/ Carbidopa in the treatment of Parkinson's disease

Medicosis Perfectionalis - YouTube 👍 13 👎 0

PARKINSON'S
DISEASE
PHARMACOLOGY

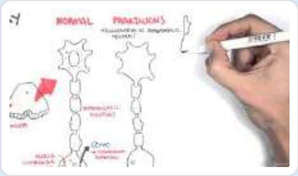
Parkinson's disease pharmacology

Soton Brain Hub - YouTube 👍 4 👎 0



Pharmacology - DRUGS FOR PARKINSON'S DISEASE

Speed Pharmacology - YouTube 👍 8 👎 1


NORMAL
PARKINSONS

Pharmacology - Parkinson's Disease

Armando Hasudungan - YouTube 👍 16 👎 2

PARKINSON'S
DISEASE
Osmosis.org

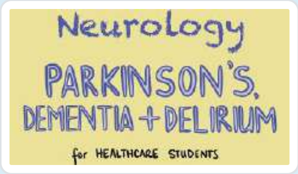
Parkinson's disease

Osmosis - YouTube 👍 4 👎 1

PARKINSON'S
DISEASE
SHUFFLING GAIT
α-SYNUCLEIN
PILL ROLLING TREMOR
COG-WHEEL RIGIDITY
DaT SCAN

Understanding Parkinson's Disease

Rhesus Medicine - YouTube 👍 2 👎 1


Neurology
PARKINSON'S,
DEMENTIA + DELIRIUM
for HEALTHCARE STUDENTS

Parkinson's disease

MedFlix - YouTube 👍 1 👎 2

Report broken media

Score: **16%**

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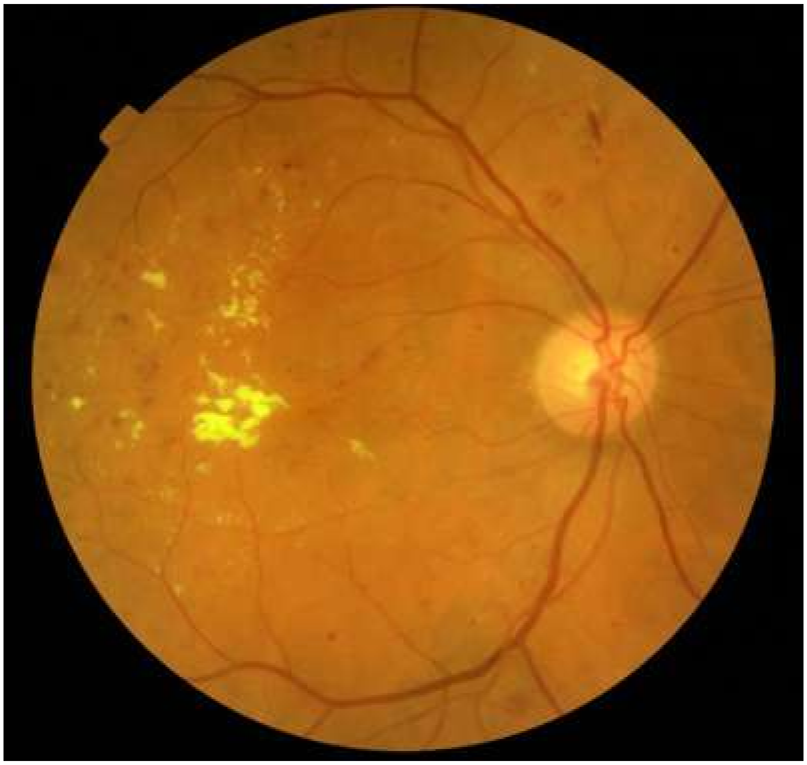
A 48-year-old man with a history of type II diabetes mellitus attends the routine diabetic eye screening. He reports good compliance with his diabetes medications. His fundoscopy showed:



What is the most appropriate management for this patient?

○ Intra-vitreal VEGF inhibitor therapy

○ Optimise glycaemic control

○ Routine 2-yearly eye screening

○ Urgent trabeculectomy

○ Vitreoretinal surgery

Submit answer

Reference ranges

Score: **0%**

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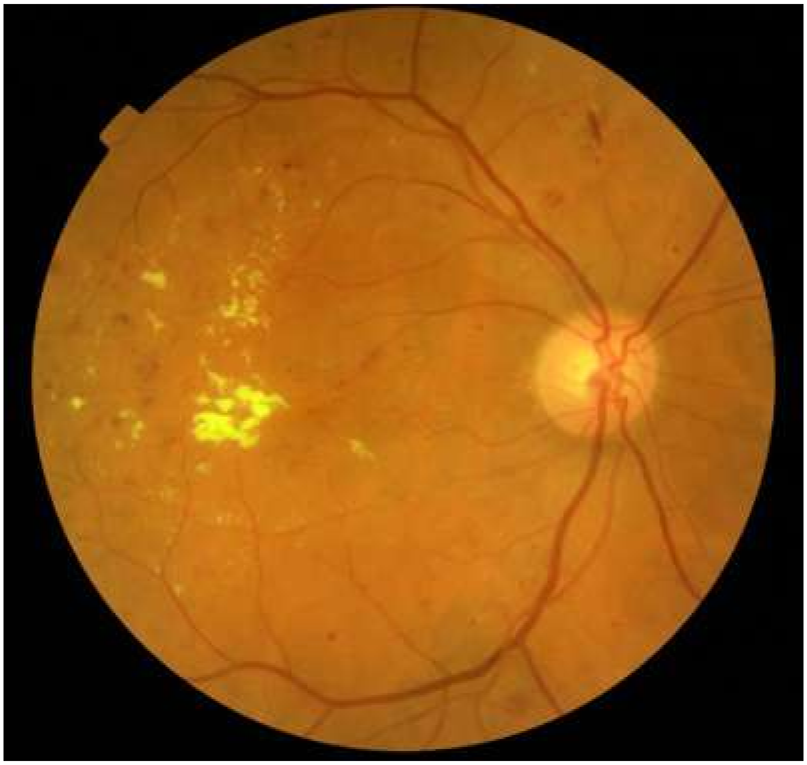
Question 13 of 299

A 48-year-old man with a history of type II diabetes mellitus attends the routine diabetic eye screening. He reports good compliance with his diabetes medications. His fundoscopy showed:



What is the most appropriate management for this patient?

Intra-vitreal VEGF inhibitor therapy	41%
Optimise glycaemic control	45%
Routine 2-yearly eye screening	11%
Urgent trabeculectomy	1%
Vitreoretinal surgery	2%

The fundoscopic findings of microaneurysms, hard exudates, and blot haemorrhages suggest severe non-proliferative diabetic retinopathy (NPDR). Besides optimising glycaemic control, antihypertensives and statin therapy, pan-retinal laser photocoagulation should be considered in severe NPDR.

Optimise glycaemic control should be considered in all patients with non-proliferative or proliferative diabetic retinopathy. Furthermore, tight control of blood pressure and lipid levels may help slow the progression of diabetic retinopathy.

Intra-vitreal VEGF inhibitor therapy is recommended for patients with maculopathy or may be considered in combination with pan-retinal laser photocoagulation to treat severe proliferative diabetic retinopathy which may present with extensive neovascularisation around the disc or elsewhere.

Routine 2-yearly eye screening is incorrect. All patients aged 15 or over with diabetes are advised to have routine diabetic eye screening at least once a year.

Urgent trabeculectomy , the surgical intervention used to reduce intraocular pressure in acute angle-closure glaucoma. Glaucoma leads to the classical fundoscopic appearance of cupping or increased vertical cup-to-disc ratio, which is not seen in this patient.

Vitreoretinal surgery is recommended for severe or vitreous haemorrhage, which is often a complication of extensive neovascularisation seen in proliferative diabetic retinopathy (PDR). However, this patient does not show any PDR or vitreous haemorrhage features, which may be seen as a red haze of haemorrhage into the vitreous associated with dot-blot haemorrhages, neovascularisation elsewhere or boat-shaped pre-retinal haemorrhages.

👍 👎 Discuss (1) Improve

Next question ›

Diabetic retinopathy ★

Diabetic retinopathy is the most common cause of blindness in adults aged 35-65 years-old.

Pathophysiology

Hyperglycaemia is thought to cause increased retinal blood flow and abnormal metabolism in the retinal vessel walls. This precipitates damage to endothelial cells and pericytes

Endothelial dysfunction leads to increased vascular permeability which causes the characteristic exudates seen on fundoscopy. Pericyte dysfunction predisposes to the formation of microaneurysms. Neovasculization is thought to be caused by the production of growth factors in response to retinal ischaemia

Classification

Patients are typically classified into those with non-proliferative diabetic retinopathy (NPDR), proliferative retinopathy (PDR) and maculopathy.

Non-proliferative diabetic retinopathy

Mild NPDR

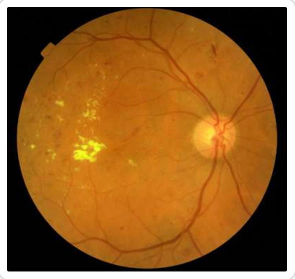
- 1 or more microaneurysm

Moderate NPDR

- microaneurysms
- blot haemorrhages
- hard exudates
- cotton wool spots ('soft exudates' - represent areas of retinal infarction), venous beading/looping and intraretinal microvascular abnormalities (IRMA) less severe than in severe NPDR

Severe NPDR

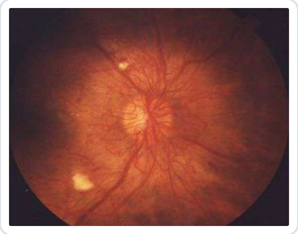
- blot haemorrhages and microaneurysms in 4 quadrants
- venous beading in at least 2 quadrants
- IRMA in at least 1 quadrant



Proliferative diabetic retinopathy

Key features

- retinal neovascularisation - may lead to vitrous haemorrhage
- fibrous tissue forming anterior to retinal disc
- more common in Type I DM, 50% blind in 5 years



Maculopathy

Key features

- based on location rather than severity, anything is potentially serious
- hard exudates and other 'background' changes on macula
- check visual acuity
- more common in Type II DM

Management

All patients

- optimise glycaemic control, blood pressure and hyperlipidemia
- regular review by ophthalmology

Maculopathy

- if there is a change in visual acuity then intravitreal vascular endothelial growth factor (VEGF) inhibitors

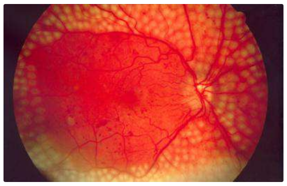
Non-proliferative retinopathy

- regular observation
- if severe/very severe consider panretinal laser photocoagulation

Proliferative retinopathy

- panretinal laser photocoagulation
  - following treatment around 50% of patients develop a noticeable reduction in their visual fields due to the scarring of peripheral retinal tissue
  - other complications include a decrease in night vision (rods are predominantly responsible for vision in low light conditions, the majority of rod cells are located in the peripheral retina), a generalised decrease in visual acuity and macular oedema
- intravitreal VEGF inhibitors
  - often now used in combination with panretinal laser photocoagulation
  - examples include ranibizumab
  - strong evidence base suggests they both slow progression of proliferative diabetic retinopathy and improve visual acuity

- if severe or vitreous haemorrhage: vitreoretinal surgery



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Next question >

Textbooks

High-yield textbook

Extended textbook

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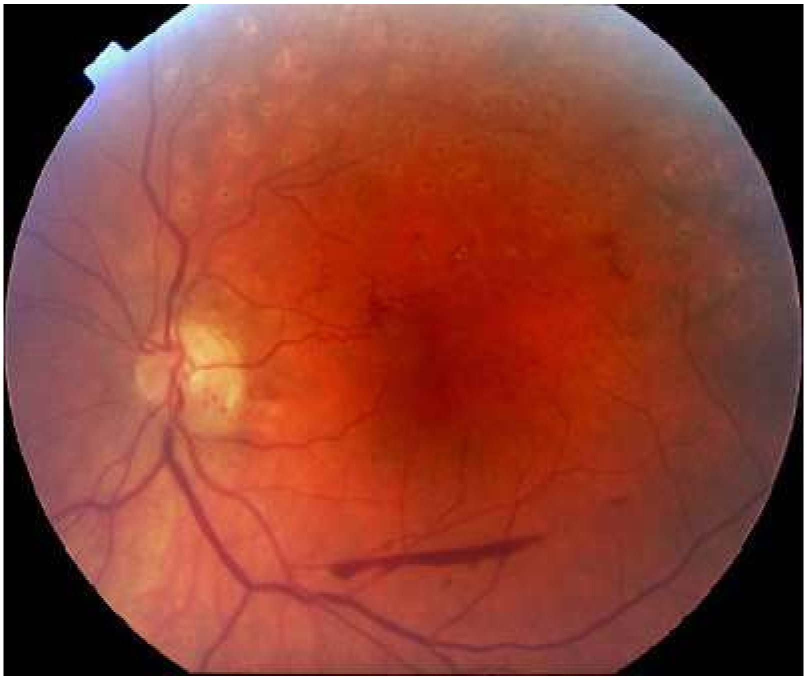
A 60-year-old woman with history of proliferative diabetic retinopathy presents to the emergency department with a sudden painless loss of vision in the left eye. On examination, there is reduced visual acuity in the left eye.

Fundoscopy shows:



Given the fundoscopy findings, what is the most likely diagnosis?

○ Central retinal artery occlusion

○ Central retinal vein occlusion

○ Hypertensive retinopathy

○ Retinal detachment

○ Vitreous haemorrhage

Submit answer

Reference ranges

Score: **0%**

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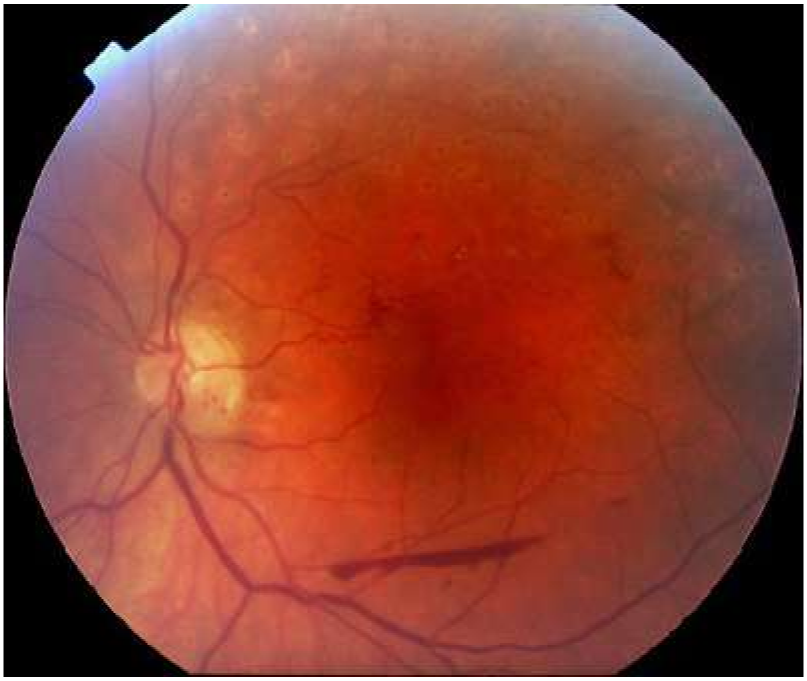


Given the fundoscopy findings, what is the most likely diagnosis?

Central retinal artery occlusion	12%
Central retinal vein occlusion	38%
Hypertensive retinopathy	8%
Retinal detachment	7%
Vitreous haemorrhage	35%

The central red haze of haemorrhage into the vitreous is characteristic of vitreous haemorrhage. Looking closely at this patient's retina, there are dot and blot haemorrhages (seen as round blots of haemorrhages with indistinct margins) in the upper periphery of the retina. There is also evidence of neovascularisation which is seen best in the mid-zone with multiple fine vessels leaving the optic disc into the vitreous. Additionally, in the lower quadrant of the retina, there is a pre-retinal haemorrhage. Together, these features are suggestive of proliferative diabetic retinopathy.

Vitreous haemorrhage is characterised by a red haze of haemorrhage into the vitreous. The associated findings of dot blot haemorrhages, neovascularisation and pre-retinal haemorrhages suggest proliferative diabetic retinopathy, the most common cause of vitreous haemorrhage in adults.

Central retinal artery occlusion is characterised by retinal whitening and thin arteries. Associated findings may include sluggish blood flow (boxcar shape of the arteries), Hollenhorst plaque-cholesterol (refractile lesions within the arteries) or large calcific plaques (white lesions within the arteries).

Central retinal vein occlusion results in diffuse retinal 'blood and thunder' haemorrhages. However, this is less common and is not usually associated with other features of proliferative diabetic retinopathy seen in this patient. Risk factors for retinal vein occlusion include advancing age (>55y), hypertension, diabetes, glaucoma, sickle cell anaemia, oral contraceptives and smoking.

Hypertensive retinopathy can cause retinal vascular damage, resulting in flame-shaped haemorrhages associated with arteriolar constriction, arteriovenous nicking, cotton-wool spots, yellow hard exudates, and optic disk oedema, which are not seen in this patient.

Retinal detachment appears as the retina detaches from the typical structure of the eye. In the case of rhegmatogenous retinal detachment, a large bullous separation of the retina may be seen.

👍 👎 Discuss (3) Improve

Next question >

Vitreous haemorrage ★

Vitreous haemorrhage is bleeding into the vitreous humour. It is one of the most common causes of sudden painless loss of vision. It causes disruption to vision to a variable degree, ranging from floaters to complete visual loss. The source of bleeding can be from disruption of any vessel in the retina as well as the extension through the retina from other areas. Once the bleeding stops, the blood is typically cleared from the retina at an approximate rate of 1% per day.

Epidemiology

- Spontaneous vitreous haemorrhage has an incidence of around 7 cases per 100000 patient-years[1]
- Incidence by age and sex varies according to the incidence of the underlying causes

Common causes (collectively account for 90% of cases):

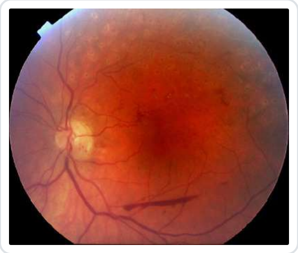
- proliferative diabetic retinopathy (over 50%)
- posterior vitreous detachment
- ocular trauma: the most common cause in children and young adults

Patients typically present with an acute or subacute onset of:

- painless visual loss or haze (commonest)
- red hue in the vision
- floaters or shadows/dark spots in the vision

Signs:

- decreased visual acuity: variable depending on the location, size and degree of vitreous haemorrhage
- visual field defect if severe haemorrhage



Investigations:

- dilated fundoscopy: may show haemorrhage in the vitreous cavity
- slit-lamp examination: red blood cells in the anterior vitreous
- ultrasound: useful to rule out retinal tear/detachment and if haemorrhage obscures the retina
- fluorescein angiography: to identify neovascularization
- orbital CT: used if open globe injury

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16%**

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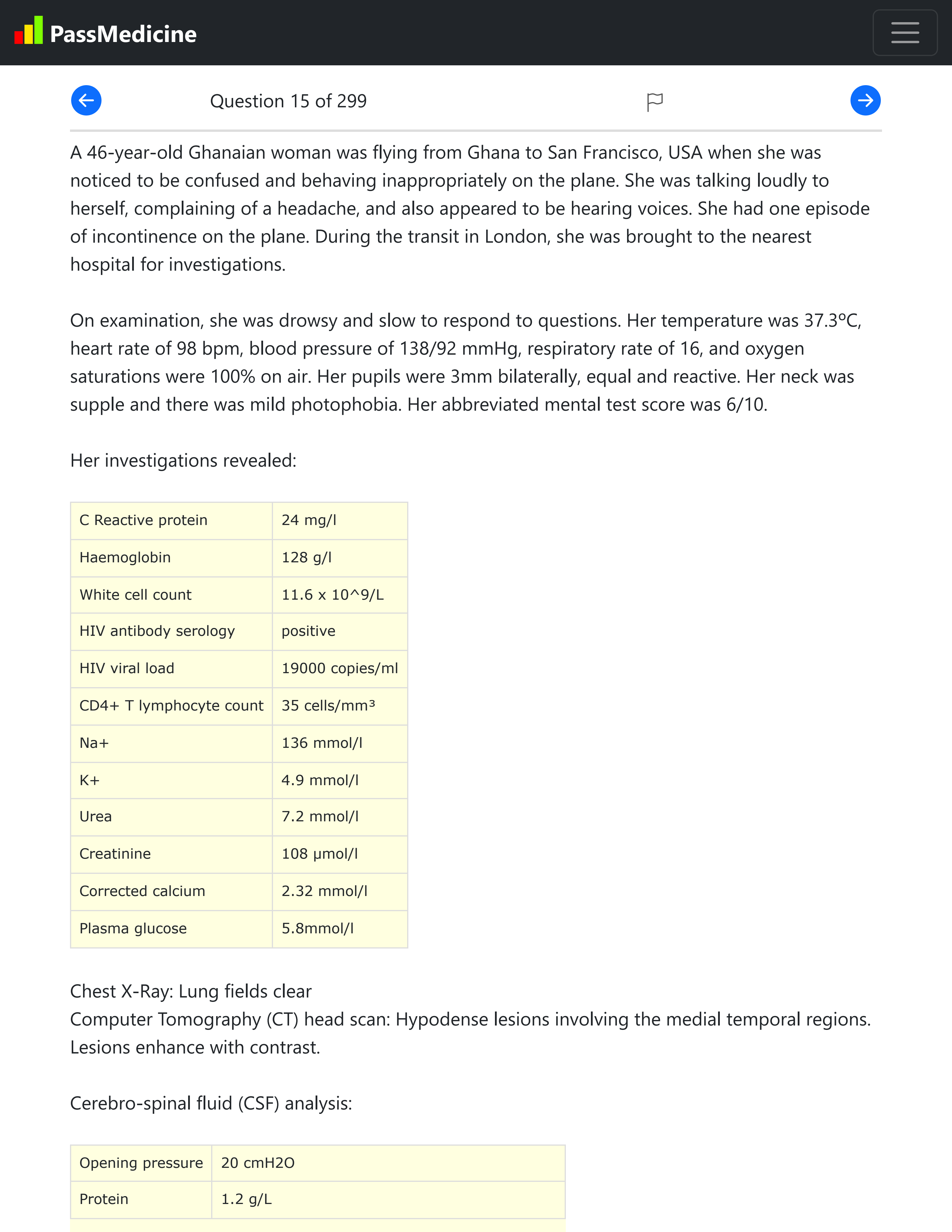

A 46-year-old Ghanaian woman was flying from Ghana to San Francisco, USA when she was noticed to be confused and behaving inappropriately on the plane. She was talking loudly to herself, complaining of a headache, and also appeared to be hearing voices. She had one episode of incontinence on the plane. During the transit in London, she was brought to the nearest hospital for investigations.

On examination, she was drowsy and slow to respond to questions. Her temperature was 37.3ºC, heart rate of 98 bpm, blood pressure of 138/92 mmHg, respiratory rate of 16, and oxygen saturations were 100% on air. Her pupils were 3mm bilaterally, equal and reactive. Her neck was supple and there was mild photophobia. Her abbreviated mental test score was 6/10.

Her investigations revealed:

C Reactive protein	24 mg/l
Haemoglobin	128 g/l
White cell count	11.6 x 10^9/L
HIV antibody serology	positive
HIV viral load	19000 copies/ml
CD4+ T lymphocyte count	35 cells/mm³
Na+	136 mmol/l
K+	4.9 mmol/l
Urea	7.2 mmol/l
Creatinine	108 µmol/l
Corrected calcium	2.32 mmol/l
Plasma glucose	5.8mmol/l

Chest X-Ray: Lung fields clear
Computer Tomography (CT) head scan: Hypodense lesions involving the medial temporal regions. Lesions enhance with contrast.

Cerebro-spinal fluid (CSF) analysis:

Opening pressure	20 cmH2O
Protein	1.2 g/L

White cell count	50 per mm³ (predominantly mononuclear cells)
Red cell count	5 per mm³
Glucose	4.8 mmol/l
Gram stain	No organisms seen

What is the next most appropriate management step?

○ Intravenous acyclovir ×

○ Oral prednisolone ×

○ Highly Active Anti-Retroviral Therapy (HAART) ×

○ Intravenous fluconazole ×

○ Pyrimethamine and Sulfadiazine ×

Submit answer

Reference ranges

Score: **0%**

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A 46-year-old Ghanaian woman was flying from Ghana to San Francisco, USA when she was noticed to be confused and behaving inappropriately on the plane. She was talking loudly to herself, complaining of a headache, and also appeared to be hearing voices. She had one episode of incontinence on the plane. During the transit in London, she was brought to the nearest hospital for investigations.

On examination, she was drowsy and slow to respond to questions. Her temperature was 37.3ºC, heart rate of 98 bpm, blood pressure of 138/92 mmHg, respiratory rate of 16, and oxygen saturations were 100% on air. Her pupils were 3mm bilaterally, equal and reactive. Her neck was supple and there was mild photophobia. Her abbreviated mental test score was 6/10.

Her investigations revealed:

C Reactive protein	24 mg/l
Haemoglobin	128 g/l
White cell count	11.6 x 10^9/L
HIV antibody serology	positive
HIV viral load	19000 copies/ml
CD4+ T lymphocyte count	35 cells/mm³
Na+	136 mmol/l
K+	4.9 mmol/l
Urea	7.2 mmol/l
Creatinine	108 µmol/l
Corrected calcium	2.32 mmol/l
Plasma glucose	5.8mmol/l

Chest X-Ray: Lung fields clear
Computer Tomography (CT) head scan: Hypodense lesions involving the medial temporal regions. Lesions enhance with contrast.

Cerebro-spinal fluid (CSF) analysis:

Opening pressure	20 cmH2O
Protein	1.2 g/L

White cell count	50 per mm^3 (predominantly mononuclear cells)
Red cell count	5 per mm^3
Glucose	4.8 mmol/l
Gram stain	No organisms seen

What is the next most appropriate management step?

Intravenous acyclovir	45%
Oral prednisolone	3%
Highly Active Anti-Retroviral Therapy (HAART)	18%
Intravenous fluconazole	4%
Pyrimethamine and Sulfadiazine	30%

This lady is immunocompromised with a new diagnosis of HIV, and has an altered conscious level, with psychotic symptoms suggestive of encephalitis. Her CT head scan shows the involvement of the medial temporal region, which is highly suggestive of herpes encephalitis, and this is supported by the LP results with raised protein levels, and a predominantly mononuclear white cell picture. Viral polymerase chain reaction (PCR) is the gold standard in the diagnosis of herpes encephalitis, and management for Herpes encephalitis is high-dose intravenous acyclovir to achieve central nervous system penetrance.

Although this lady has advanced HIV infection, there is some evidence that starting HAART may cause an immune reconstitution syndrome, resulting in an initial deterioration in the patient's condition as the immune system is boosted. Thus while she would require HAART, the initial management should be to decrease the burden of infection with acyclovir to prevent a deterioration in her condition.

There is limited evidence for steroid use in HSV encephalitis. There is no evidence of cryptococcal or toxoplasmosis infection from the CT and CSF results, which rules out options (d) and (e) respectively.

👍 👎 Discuss (7) Improve

Next question ›

Herpes simplex encephalitis

Herpes simplex (HSV) encephalitis is a common topic in the exam. The virus characteristically affects the temporal lobes - questions may give the result of imaging or describe temporal lobe signs e.g. aphasia.

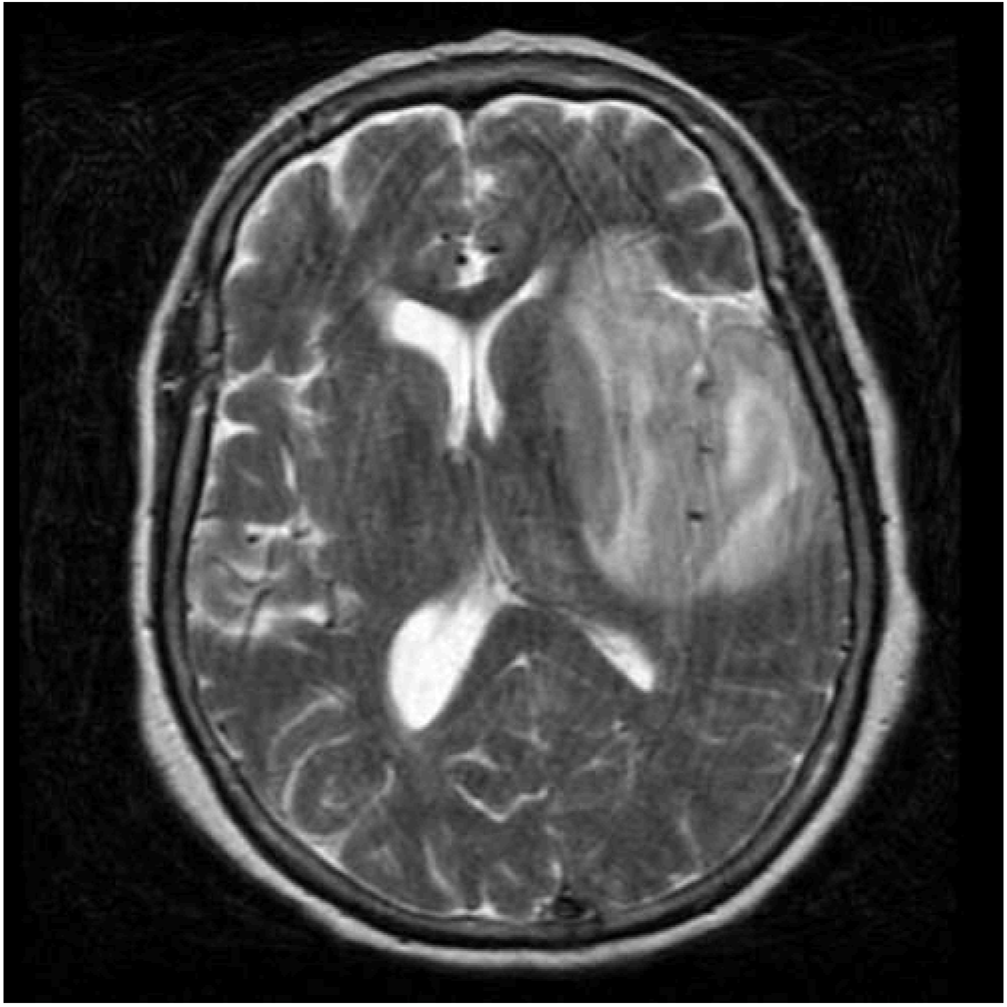
Features
- fever, headache, psychiatric symptoms, seizures, vomiting
- focal features e.g. aphasia
- peripheral lesions (e.g. cold sores) have no relation to the presence of HSV encephalitis

Pathophysiology
- HSV-1 is responsible for 95% of cases in adults
- typically affects temporal and inferior frontal lobes

Investigation
- CSF: lymphocytosis, elevated protein
- PCR for HSV
- CT: medial temporal and inferior frontal changes (e.g. petechial haemorrhages) - normal in one-third of patients
- MRI is better
- EEG pattern: lateralised periodic discharges at 2 Hz

Treatment
- intravenous aciclovir

The prognosis is dependent on whether aciclovir is commenced early. If treatment is started promptly the mortality is 10-20%. Left untreated the mortality approaches 80%



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MRI of a patient with HSV encephalitis. There is hyperintensity of the affected white matter and cortex in the medial temporal lobes and insular cortex.

+ Q 123

Next question >

Textbooks

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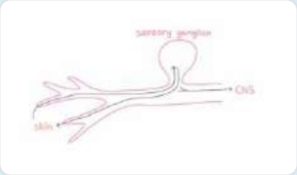
Media


sensory ganglion
CNS
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Herpes simplex encephalitis

macrophage - YouTube 👍 2 👎 0

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Score: **16%**

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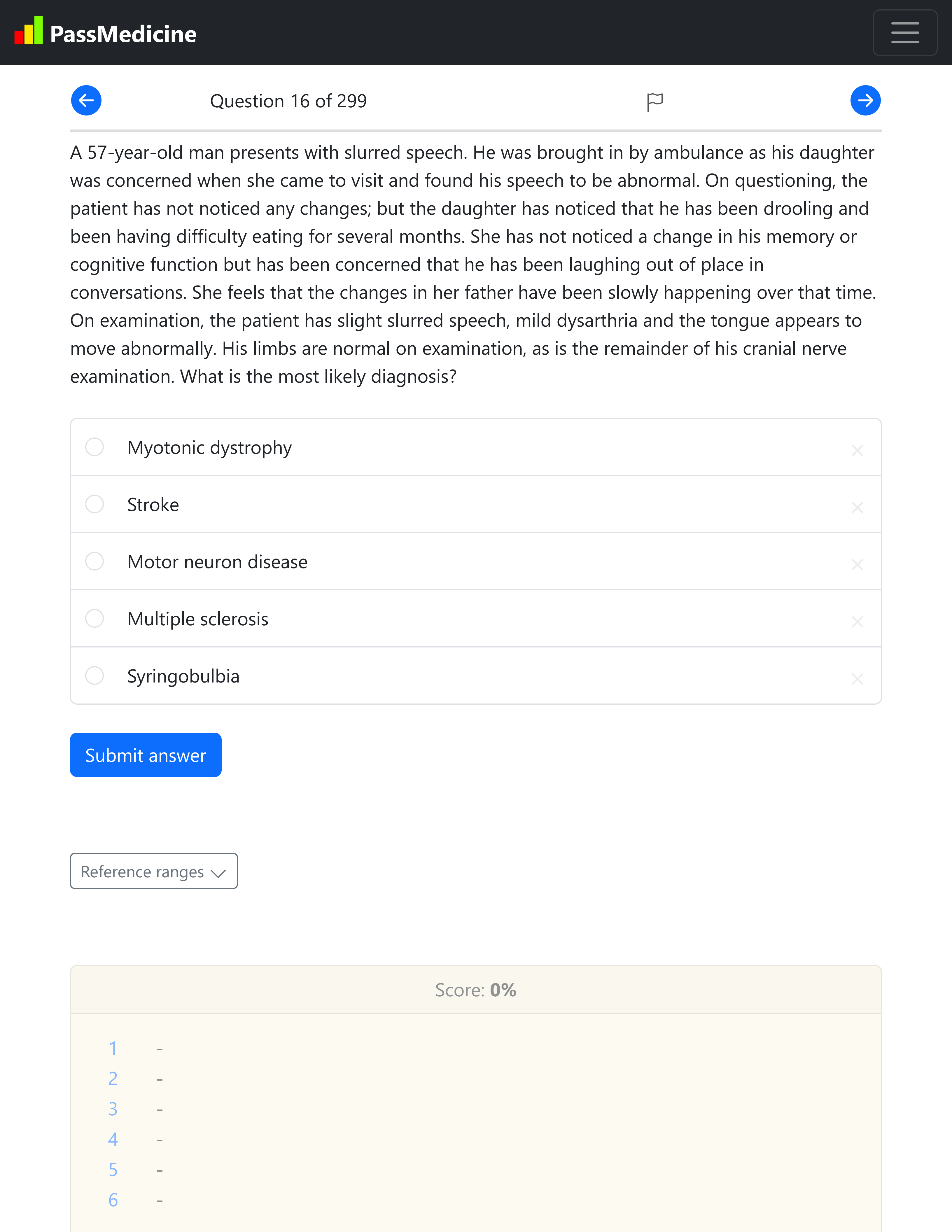

Question 16 of 299

A 57-year-old man presents with slurred speech. He was brought in by ambulance as his daughter was concerned when she came to visit and found his speech to be abnormal. On questioning, the patient has not noticed any changes; but the daughter has noticed that he has been drooling and been having difficulty eating for several months. She has not noticed a change in his memory or cognitive function but has been concerned that he has been laughing out of place in conversations. She feels that the changes in her father have been slowly happening over that time. On examination, the patient has slight slurred speech, mild dysarthria and the tongue appears to move abnormally. His limbs are normal on examination, as is the remainder of his cranial nerve examination. What is the most likely diagnosis?

○ Myotonic dystrophy

○ Stroke

○ Motor neuron disease

○ Multiple sclerosis

○ Syringobulbia

Score: **0%**

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Question 16 of 299 ✗

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Myotonic dystrophy	14%
Stroke	6%
Motor neuron disease	37%
Multiple sclerosis	3%
Syringobulbia	40%

The correct answer is motor neuron disease. This a male patient in his 50s who has developed evidence of bulbar signs without any sensory or peripheral involvement. The chronic history makes a stroke less likely, whilst the progressive nature, his age and sex makes multiple sclerosis less likely. The absence of sensory loss or weakness of the arms makes syringobulbia less likely whilst myotonic dystrophy would have more peripheral signs. This is motor neuron disease presenting with bulbar features.

Discuss (16) | Improve

Next question >

Motor neuron disease: features

Motor neuron disease is a neurological condition of unknown cause which can present with both upper and lower motor neuron signs. It rarely presents before 40 years and various patterns of disease are recognised including amyotrophic lateral sclerosis (ALS), progressive muscular atrophy and bulbar palsy.

There are a number of clues which point towards a diagnosis of motor neuron disease:
- asymmetric limb weakness is the most common presentation of ALS
- the mixture of lower motor neuron and upper motor neuron signs
- wasting of the small hand muscles/tibialis anterior is common
- fasciculations
- the absence of sensory signs/symptoms
    - vague sensory symptoms may occur early in the disease (e.g. limb pain) but 'never' sensory signs

Other features
- doesn't affect external ocular muscles
- no cerebellar signs
- abdominal reflexes are usually preserved and sphincter dysfunction if present is a late feature

The diagnosis of motor neuron disease is clinical, but nerve conduction studies will show normal motor conduction and can help exclude a neuropathy. Electromyography shows a reduced number of action potentials with increased amplitude. MRI is usually performed to exclude the differential diagnosis of cervical cord compression and myelopathy

AI + Q 123

Next question >

Textbooks

High-yield textbook

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Links

Postgraduate Medical Journal 👍 15 👎 11

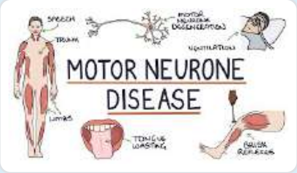
Review of MND

Royal College of Physicians 👍 17 👎 10

Motor neuron disease: diagnostic pitfalls

Suggest link Report broken link

Media



Motor neuron disease

Zero To Finals - YouTube 👍 0 👎 0

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Score: **16%**

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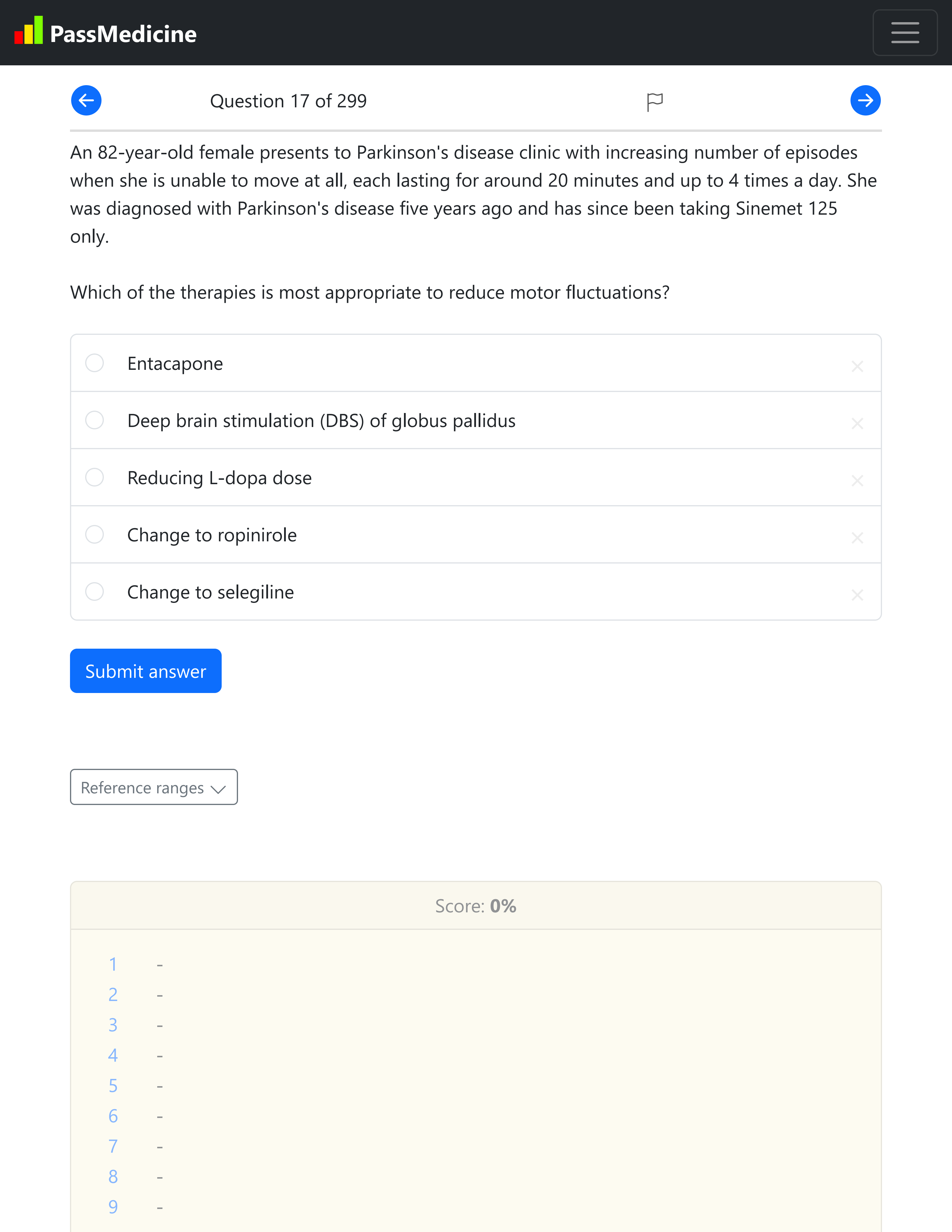

Question 17 of 299

An 82-year-old female presents to Parkinson's disease clinic with increasing number of episodes when she is unable to move at all, each lasting for around 20 minutes and up to 4 times a day. She was diagnosed with Parkinson's disease five years ago and has since been taking Sinemet 125 only.

Which of the therapies is most appropriate to reduce motor fluctuations?

- ○ Entacapone ×
- ○ Deep brain stimulation (DBS) of globus pallidus ×
- ○ Reducing L-dopa dose ×
- ○ Change to ropinirole ×
- ○ Change to selegiline ×

Score: **0%**

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Question 17 of 299 ✗

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Which of the therapies is most appropriate to reduce motor fluctuations?

🟩 Entacapone	46%
Deep brain stimulation (DBS) of globus pallidus	7%
Reducing L-dopa dose	18%
Change to ropinirole	23%
🟥 Change to selegiline	5%

The patient describes increasing frequency of 'on-off' fluctuations, a common complaint in patients who have been diagnosed with PD and medicated with L-dopa for a long time. Selegiline, a monoamine oxidase B inhibitor, while useful as an adjunct, has a very poor evidence base as a monotherapy. There is little evidence that in a more elderly patient, changing to a dopamine agonist has any benefit. The patients L-dopa load should be kept as low as possible but the main problem is the lack of a smooth profile. DBS is premature. Entacapone is a catechol-O-methyltransferase (COMT) inhibitor, which reduces the peripheral breakdown of L-dopa and increases the serum half-life of L-dopa. It has little therapeutic benefit when used alone but is a successful adjunct in smoothing out motor fluctuations.

Discuss (8) Improve

Next question ›

Parkinson's disease: management ★

Parkinsons disease should only be diagnosed, and management initiated, by a specialist with expertise in movement disorders. However, it is important for all doctors to be aware of the medications used in Parkinson's given the prevalence of this condition. NICE published guidelines in 2017 regarding the management of Parkinson's disease.

For first-line treatment:

- if the motor symptoms are affecting the patient's quality of life: levodopa
- if the motor symptoms are not affecting the patient's quality of life: dopamine agonist (non-ergot derived), levodopa or monoamine oxidase B (MAO-B) inhibitor

Whilst all drugs used to treat Parkinson's can cause a wide variety of side-effects NICE produced tables to help with decision making:

	Levodopa	**Dopamine agonists**	**MAO-B inhibitors**
Motor symptoms	More improvement in motor symptoms	Less improvement in motor symptoms	Less improvement in motor symptoms
Activities of daily living	More improvement in activities of daily living	Less improvement in activities of daily living	Less improvement in activities of daily living
Motor complications	More motor complications	Fewer motor complications	Fewer motor complications
Adverse events	Fewer specified adverse events*	More specified adverse events*	Fewer specified adverse events*

* excessive sleepiness, hallucinations and impulse control disorders

If a patient continues to have symptoms despite optimal levodopa treatment or has developed dyskinesia then NICE recommend the addition of a dopamine agonist, MAO-B inhibitor or catechol-O-methyl transferase (COMT) inhibitor as an adjunct. Again, NICE summarise the main points in terms of decision making:

	Dopamine agonists	**MAO-B inhibitors**	**COMT inhibitors**	**Amantadine**
Motor symptoms	Improvement in motor symptoms	Improvement in motor symptoms	Improvement in motor symptoms	No evidence of improvement in motor symptoms
Activities of daily living	Improvement in activities of daily living	Improvement in activities of daily living	Improvement in activities of daily living	No evidence of improvement in activities of daily living

	Dopamine agonists	**MAO-B inhibitors**	**COMT inhibitors**	**Amantadine**
Off time	More off-time reduction	Off-time reduction	Off-time reduction	No studies reporting this outcome
Adverse events	Intermediate risk of adverse events	Fewer adverse events	More adverse events	No studies reporting this outcome
Hallucinations	More risk of hallucinations	Lower risk of hallucinations	Lower risk of hallucinations	No studies reporting this outcome

Specific points regarding Parkinson's medication

NICE reminds us of the risk of acute akinesia or neuroleptic malignant syndrome if medication is not taken/absorbed (for example due to gastroenteritis) and advise against giving patients a 'drug holiday' for the same reason.

Impulse control disorders have become a significant issue in recent years. These can occur with any dopaminergic therapy but are more common with:
- dopamine agonist therapy
- a history of previous impulsive behaviours
- a history of alcohol consumption and/or smoking

If excessive daytime sleepiness develops then patients should not drive. Medication should be adjusted to control symptoms. Modafinil can be considered if alternative strategies fail.

If orthostatic hypotension develops then a medication review looking at potential causes should be done. If symptoms persist then midodrine (acts on peripheral alpha-adrenergic receptors to increase arterial resistance) can be considered.

Consider glycopyrronium bromide to manage drooling of saliva in people with Parkinson's disease.

Further information regarding specific anti-Parkinson's medication

Levodopa

- nearly always combined with a decarboxylase inhibitor (e.g. carbidopa or benserazide)
    - this prevents the peripheral metabolism of levodopa to dopamine outside of the brain and hence can reduce side effects
- common adverse effects:
    - dry mouth
    - anorexia
    - palpitations
    - postural hypotension
    - psychosis
- some adverse effects are due to the difficulty in achieving a steady dose of levodopa
    - end-of-dose wearing off: symptoms often worsen towards the end of dosage interval. This results in a decline of motor activity
    - 'on-off' phenomenon: large variations in motor performance, with normal function during the 'on' period, and weakness and restricted mobility during the 'off' period
    - dyskinesias at peak dose: dystonia, chorea and athetosis (involuntary writhing movements)
    - these effects may worsen over time with - clinicians therefore may limit doses until necessary
- it is important not to acutely stop levodopa, for example, if a patient is admitted to hospital
    - if a patient with Parkinson's disease cannot take levodopa orally, they can be given a dopamine agonist patch as rescue medication to prevent acute dystonia

Dopamine receptor agonists

- e.g. bromocriptine, ropinirole, cabergoline, apomorphine
- ergot-derived dopamine receptor agonists (bromocriptine, cabergoline) have been associated with pulmonary, retroperitoneal and cardiac fibrosis. The Committee on Safety of Medicines advice that an echocardiogram, ESR, creatinine and chest x-ray should be obtained prior to treatment and patients should be closely monitored
- patients should be warned about the potential for dopamine receptor agonists to cause impulse control disorders and excessive daytime somnolence
- more likely than levodopa to cause hallucinations in older patients. Nasal congestion and postural hypotension are also seen in some patients

MAO-B (Monoamine Oxidase-B) inhibitors

- e.g. selegiline
- inhibits the breakdown of dopamine secreted by the dopaminergic neurons

Amantadine

- mechanism is not fully understood, probably increases dopamine release and inhibits its uptake at dopaminergic synapses
- side-effects include ataxia, slurred speech, confusion, dizziness and livedo reticularis

COMT (Catechol-O-Methyl Transferase) inhibitors

- e.g. entacapone, tolcapone
- COMT is an enzyme involved in the breakdown of dopamine, and hence may be used as an adjunct to levodopa therapy
- used in conjunction with levodopa in patients with established PD

Antimuscarinics

- block cholinergic receptors
- now used more to treat drug-induced parkinsonism rather than idiopathic Parkinson's disease
- help tremor and rigidity
- e.g. procyclidine, benzotropine, trihexyphenidyl (benzhexol)

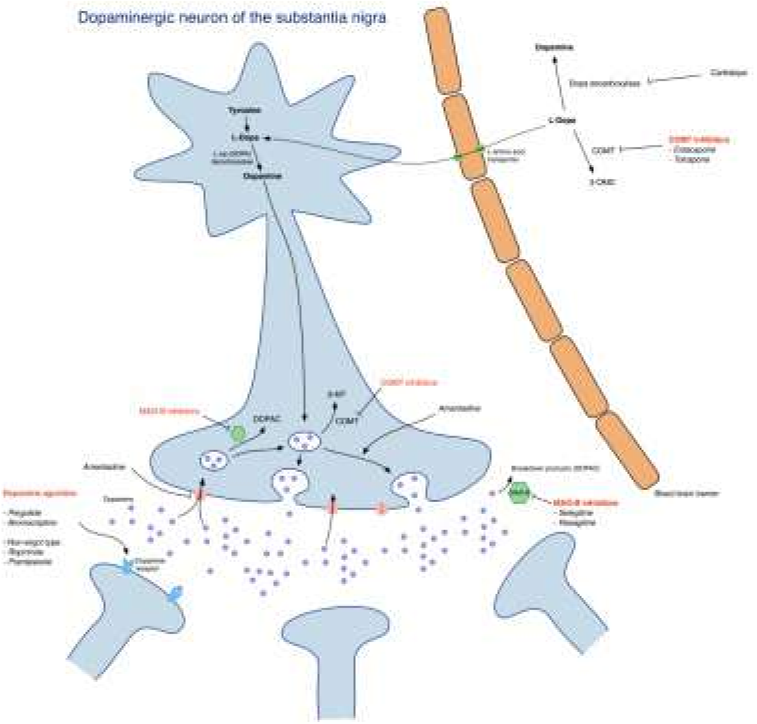
Dopaminergic neuron of the substantia nigra
Tyrosine
L-Dopa
Dopamine
Dopamine
L-Dopa
Carbidopa
COMT
Entacapone
Tolcapone
3-OMD
DOPAC
3-MT
COMT
Amantadine
Amantadine
Selegiline
Rasagiline
Blood brain barrier

Image sourced from Wikipedia

Diagram showing the mechanism of action of Parkinson's drugs

123

Next question

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Links

NICE 👍 23 👎 12

2017 Parkinson's disease guidelines

Suggest link

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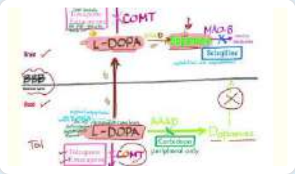
Media


COMT
L-DOPA
MAO-B
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The use of Levodopa/ Carbidopa in the treatment of Parkinson's disease

Medicosis Perfectionalis - YouTube 👍 13 👎 0

PARKINSON'S
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PHARMACOLOGY

Parkinson's disease pharmacology

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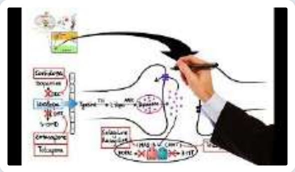

Pharmacology - DRUGS FOR PARKINSON'S DISEASE

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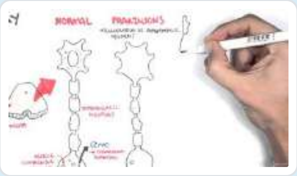
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PARKINSON'S
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[Parkinson's disease]

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PARKINSON'S
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[Understanding Parkinson's Disease]

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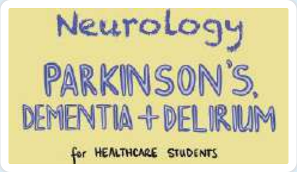
Neurology
PARKINSON'S,
DEMENTIA + DELIRIUM
for HEALTHCARE STUDENTS

[Parkinson's disease]

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Score: **16%**

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Question 18 of 299

An 80-year-old male presents to the Parkinson's disease (PD) clinic complaining of jerking and flailing movements of his arm. He says he has no control of them and finds them embarrassing and disabling. However, he reports very few other PD symptoms, with minimal problems with 'off' periods. His past medical history includes PD, diagnosed 8 years ago, and type 2 diabetes. He currently takes Sinemet 250 for Parkinson's disease 5 times a day and metformin 500mg TDS, he has been on the same dose of Sinemet for past 18 months. What is your most appropriate management?

- ○ Reduce dose of Sinemet
- ○ Increase dose of Sinemet
- ○ Start entacapone
- ○ Start ropinirole
- ○ Deep brain stimulation (DBS)

Submit answer

Reference ranges

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Reduce dose of Sinemet	34%
Increase dose of Sinemet	7%
Start entacapone	27%
Start ropinirole	28%
Deep brain stimulation (DBS)	3%

The patient describes involuntary movements on a background of chronic and substantial use of L-dopa, a phenomenon known as L-dopa dyskinesias (LID). The pathophysiology of LIDs remains unclear but the key is to minimise L-dopa dosage if at all possible. As the patient reports minimal other motor symptoms, this is possible in this case. Entacapone smooths the motor profile of L-dopa but does not help with LIDs. DBS is a possible treatment for LIDs changes to L-dopa dose is not possible or if symptoms remain refractory despite L-dopa dose changes.

Discuss (9) | Improve

Next question

Parkinson's disease: management

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For first-line treatment:

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	Dopamine agonists	MAO-B inhibitors	COMT inhibitors	Amantadine
Off time	More off-time reduction	Off-time reduction	Off-time reduction	No studies reporting this outcome
Adverse events	Intermediate risk of adverse events	Fewer adverse events	More adverse events	No studies reporting this outcome
Hallucinations	More risk of hallucinations	Lower risk of hallucinations	Lower risk of hallucinations	No studies reporting this outcome

Specific points regarding Parkinson's medication

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Levodopa

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    - palpitations
    - postural hypotension
    - psychosis
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    - dyskinesias at peak dose: dystonia, chorea and athetosis (involuntary writhing movements)
    - these effects may worsen over time with - clinicians therefore may limit doses until necessary
- it is important not to acutely stop levodopa, for example, if a patient is admitted to hospital
    - if a patient with Parkinson's disease cannot take levodopa orally, they can be given a dopamine agonist patch as rescue medication to prevent acute dystonia

Dopamine receptor agonists

- e.g. bromocriptine, ropinirole, cabergoline, apomorphine
- ergot-derived dopamine receptor agonists (bromocriptine, cabergoline) have been associated with pulmonary, retroperitoneal and cardiac fibrosis. The Committee on Safety of Medicines advice that an echocardiogram, ESR, creatinine and chest x-ray should be obtained prior to treatment and patients should be closely monitored
- patients should be warned about the potential for dopamine receptor agonists to cause impulse control disorders and excessive daytime somnolence
- more likely than levodopa to cause hallucinations in older patients. Nasal congestion and postural hypotension are also seen in some patients

MAO-B (Monoamine Oxidase-B) inhibitors

- e.g. selegiline
- inhibits the breakdown of dopamine secreted by the dopaminergic neurons

Amantadine

- mechanism is not fully understood, probably increases dopamine release and inhibits its uptake at dopaminergic synapses
- side-effects include ataxia, slurred speech, confusion, dizziness and livedo reticularis

COMT (Catechol-O-Methyl Transferase) inhibitors

- e.g. entacapone, tolcapone
- COMT is an enzyme involved in the breakdown of dopamine, and hence may be used as an adjunct to levodopa therapy
- used in conjunction with levodopa in patients with established PD

Antimuscarinics

- block cholinergic receptors
- now used more to treat drug-induced parkinsonism rather than idiopathic Parkinson's disease
- help tremor and rigidity
- e.g. procyclidine, benzotropine, trihexyphenidyl (benzhexol)

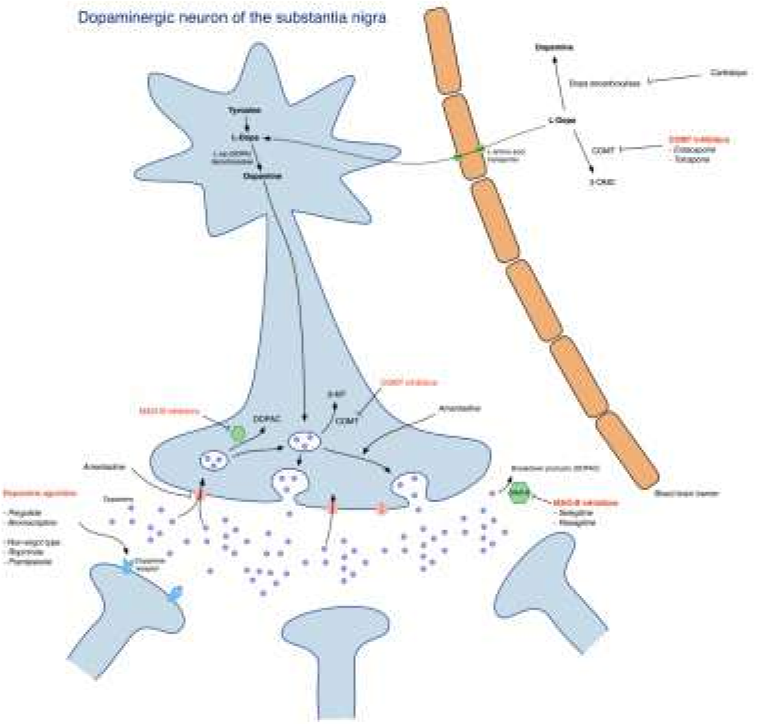

Dopaminergic neuron of the substantia nigra
Tyrosine
L-Dopa
Dopamine
L-Dopa
Dopamine
COMT
3-OMD
DOPAC
3-MT
COMT
Amantadine
Amantadine
Blood brain barrier

Image sourced from Wikipedia

Diagram showing the mechanism of action of Parkinson's drugs

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

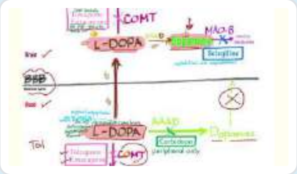
NICE 👍 23 👎 12

2017 Parkinson's disease guidelines

Suggest link

Report broken link

Media


COMT
L-DOPA
MAO-B
L-DOPA

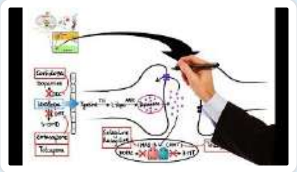
The use of Levodopa/ Carbidopa in the treatment of Parkinson's disease

Medicosis Perfectionalis - YouTube 👍 13 👎 0

PARKINSON'S
DISEASE
PHARMACOLOGY

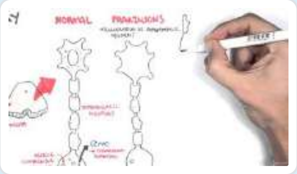
Parkinson's disease pharmacology

Soton Brain Hub - YouTube 👍 4 👎 0



Pharmacology - DRUGS FOR PARKINSON'S DISEASE

Speed Pharmacology - YouTube 👍 8 👎 1


NORMAL
PARKINSON'S

Pharmacology - Parkinson's Disease

Armando Hasudungan - YouTube 👍 16 👎 2

PARKINSON'S DISEASE
Osmosis.org

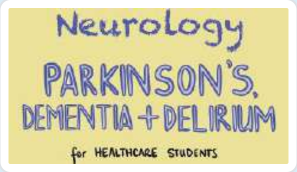
Parkinson's disease

Osmosis - YouTube 👍 4 👎 1

PARKINSON'S DISEASE
SHUFFLING GAIT
α-SYNUCLEIN
PILL ROLLING TREMOR
COG-WHEEL RIGIDITY
DaT SCAN

Understanding Parkinson's Disease

Rhesus Medicine - YouTube 👍 2 👎 1


Neurology
PARKINSON'S, DEMENTIA + DELIRIUM
for HEALTHCARE STUDENTS

Parkinson's disease

MedFlix - YouTube 👍 1 👎 2

Report broken media

Score: **16%**

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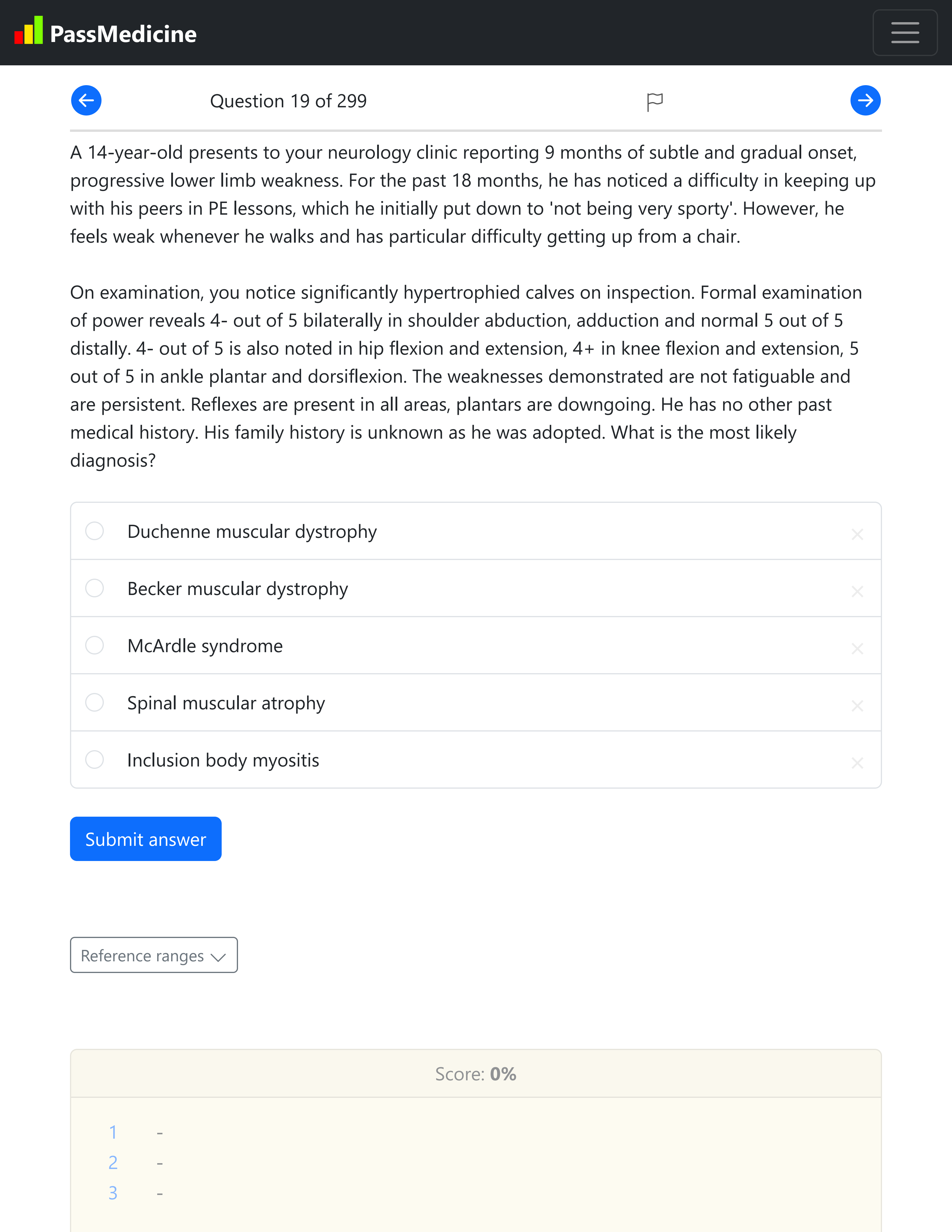

Question 19 of 299

A 14-year-old presents to your neurology clinic reporting 9 months of subtle and gradual onset, progressive lower limb weakness. For the past 18 months, he has noticed a difficulty in keeping up with his peers in PE lessons, which he initially put down to 'not being very sporty'. However, he feels weak whenever he walks and has particular difficulty getting up from a chair.

On examination, you notice significantly hypertrophied calves on inspection. Formal examination of power reveals 4- out of 5 bilaterally in shoulder abduction, adduction and normal 5 out of 5 distally. 4- out of 5 is also noted in hip flexion and extension, 4+ in knee flexion and extension, 5 out of 5 in ankle plantar and dorsiflexion. The weaknesses demonstrated are not fatiguable and are persistent. Reflexes are present in all areas, plantars are downgoing. He has no other past medical history. His family history is unknown as he was adopted. What is the most likely diagnosis?

○ Duchenne muscular dystrophy

○ Becker muscular dystrophy

○ McArdle syndrome

○ Spinal muscular atrophy

○ Inclusion body myositis

Submit answer

Reference ranges

Score: **0%**

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On examination, you notice significantly hypertrophied calves on inspection. Formal examination of power reveals 4- out of 5 bilaterally in shoulder abduction, adduction and normal 5 out of 5 distally. 4- out of 5 is also noted in hip flexion and extension, 4+ in knee flexion and extension, 5 out of 5 in ankle plantar and dorsiflexion. The weaknesses demonstrated are not fatiguable and are persistent. Reflexes are present in all areas, plantars are downgoing. He has no other past medical history. His family history is unknown as he was adopted. What is the most likely diagnosis?

Duchenne muscular dystrophy	41%
Becker muscular dystrophy	49%
McArdle syndrome	6%
Spinal muscular atrophy	2%
Inclusion body myositis	2%

Progressive proximal muscle weakness appears to be the most important symptoms in this teenage patient. The key differentials can be separated by the age of the patient. Duchenne muscular dystrophy (DMD) is a more severe form of Becker muscular dystrophy (BMD) and patients are normally wheelchair bound by early teens. Similarly, spinal muscular atrophy produces a flaccid paralysis of proximal musculature with absent reflexes, presenting from newborn to under the age of 3.

McArdle disease is a metabolic disorder affecting proximal muscles caused by myophosphorylase deficiency. Patients frequently report a 'second wind' caused by a change in fatty acid metabolism, weakness is hence classically not persistent. Inclusion body myositis classically produces distal to proximal weakness in middle-aged patients. BMD is hence the most appropriate answer.

BMD is an X-linked disorder of dystrophin, a component of skeletal muscle that reinforces the sarcolemma-glycoprotein complex and prevents degradation. Respiratory, cardiac and bulbar manifestations are common. Diagnosis is made on clinical suspicion, raised CK, EMG suggestive of small polyphasic potentials, a muscle biopsy suggestive of dystrophinopathy and subsequent genetic testing.

Discuss (3) | Improve

Next question >

Dystrophinopathies

Overview
- X-linked recessive
- due to mutation in the gene encoding dystrophin, dystrophin gene on Xp21
- dystrophin is part of a large membrane associated protein in muscle which connects the muscle membrane to actin, part of the muscle cytoskeleton
- in Duchenne muscular dystrophy there is a frameshift mutation resulting in one or both of the binding sites are lost leading to a severe form
- in Becker muscular dystrophy there is a non-frameshift insertion in the dystrophin gene resulting in both binding sites being preserved leading to a milder form

Duchenne muscular dystrophy
- progressive proximal muscle weakness from 5 years
- calf pseudohypertrophy
- Gower's sign: child uses arms to stand up from a squatted position
- 30% of patients have intellectual impairment

Becker muscular dystrophy
- develops after the age of 10 years
- intellectual impairment much less common

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

DUCHENNE & BECKER
MUSCULAR
DYSTROPHY

Duchenne & Becker muscular dystrophy

Osmosis - YouTube 👍 2 👎 0

Gower's sign

Individual - YouTube 👍 2 👎 0

Report broken media

Score: **16%**

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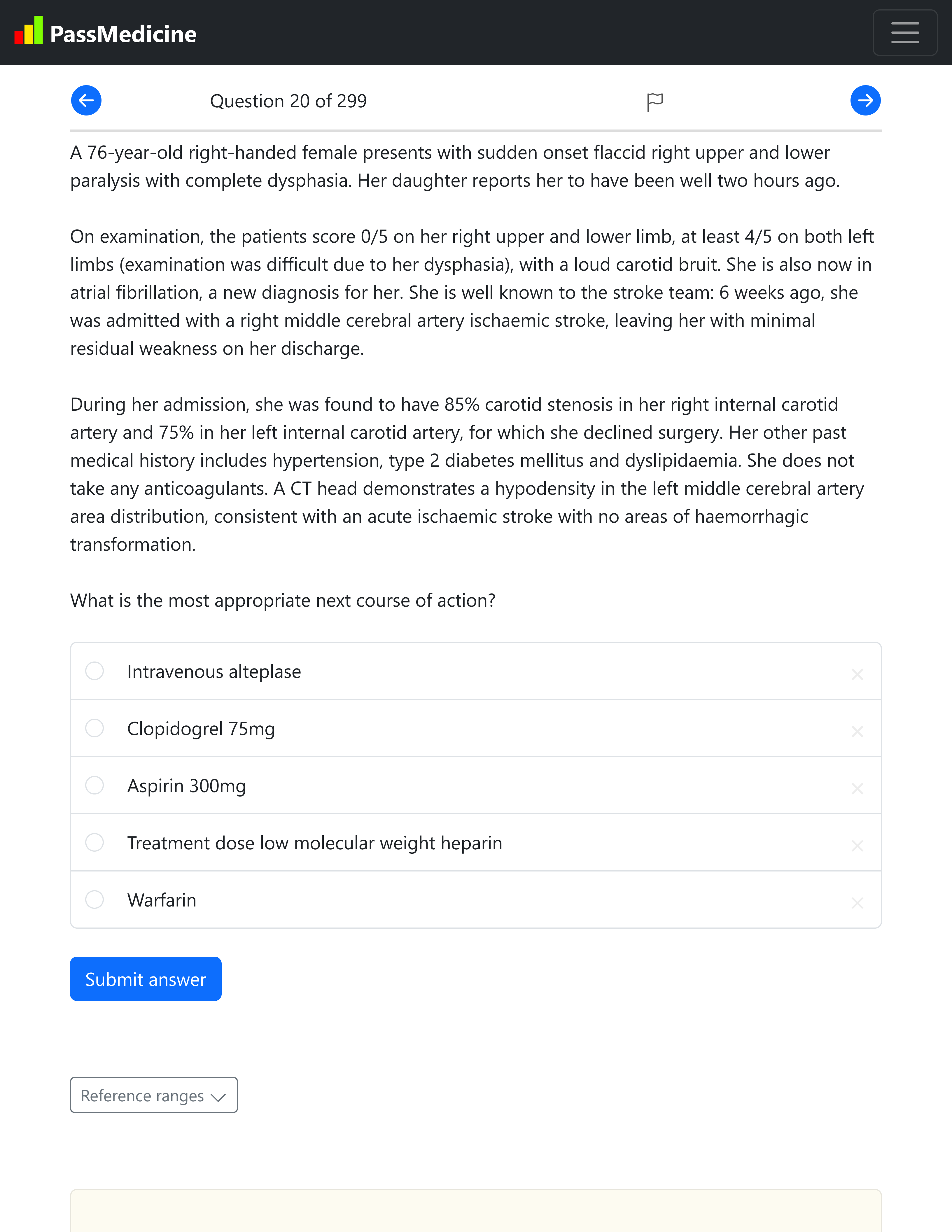

A 76-year-old right-handed female presents with sudden onset flaccid right upper and lower paralysis with complete dysphasia. Her daughter reports her to have been well two hours ago.

On examination, the patients score 0/5 on her right upper and lower limb, at least 4/5 on both left limbs (examination was difficult due to her dysphasia), with a loud carotid bruit. She is also now in atrial fibrillation, a new diagnosis for her. She is well known to the stroke team: 6 weeks ago, she was admitted with a right middle cerebral artery ischaemic stroke, leaving her with minimal residual weakness on her discharge.

During her admission, she was found to have 85% carotid stenosis in her right internal carotid artery and 75% in her left internal carotid artery, for which she declined surgery. Her other past medical history includes hypertension, type 2 diabetes mellitus and dyslipidaemia. She does not take any anticoagulants. A CT head demonstrates a hypodensity in the left middle cerebral artery area distribution, consistent with an acute ischaemic stroke with no areas of haemorrhagic transformation.

What is the most appropriate next course of action?

○ Intravenous alteplase

○ Clopidogrel 75mg

○ Aspirin 300mg

○ Treatment dose low molecular weight heparin

○ Warfarin

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What is the most appropriate next course of action?

Intravenous alteplase	46%
Clopidogrel 75mg	3%
Aspirin 300mg	48%
Treatment dose low molecular weight heparin	2%
Warfarin	1%

This patient has clearly had a new ischaemic stroke. Her NIHSS score and presentation within 4.5 hours would be sufficient to warrant consideration of thrombolysis. However, the key here is to identify the contraindication to thrombolysis: refusing carotid surgery recently is NOT a contraindication. However, a recent stroke or head trauma within 3 months is. The appropriate management would be to load the patient with 300mg aspirin. Clopidogrel 75mg is preferred 14 days after an ischaemic stroke. Anticoagulation is typically started 14 days after the acute stroke, possibly sooner in patients with small infarct sizes and hence a low risk of haemorrhagic transformation.

Discuss (8) Improve

Next question

Stroke: management of acute ischaemic stroke

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
    - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
    - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:

- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.

- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
    - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
    - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 11 👎 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 16

Stroke and TIA guidelines

Age and Ageing 👍 21 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

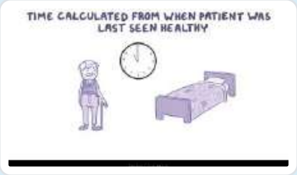
2023 National Clinical Guideline for Stroke

NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia
Acute Ischaemic Stroke

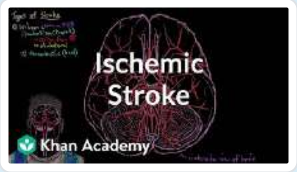
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

Khan Academy - YouTube 👍 1 👎 1

Report broken media

Score: **16%**

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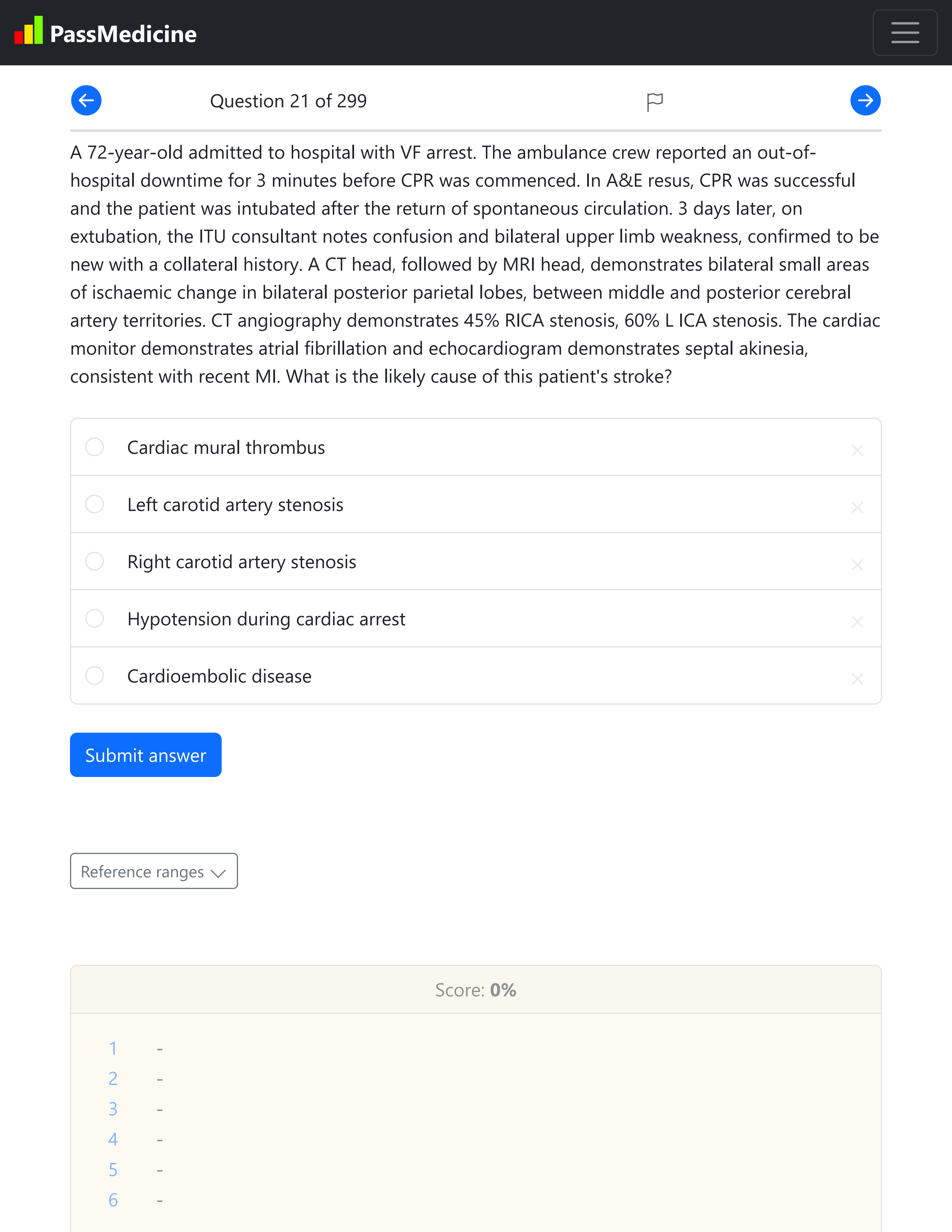

Question 21 of 299

A 72-year-old admitted to hospital with VF arrest. The ambulance crew reported an out-of-hospital downtime for 3 minutes before CPR was commenced. In A&E resus, CPR was successful and the patient was intubated after the return of spontaneous circulation. 3 days later, on extubation, the ITU consultant notes confusion and bilateral upper limb weakness, confirmed to be new with a collateral history. A CT head, followed by MRI head, demonstrates bilateral small areas of ischaemic change in bilateral posterior parietal lobes, between middle and posterior cerebral artery territories. CT angiography demonstrates 45% RICA stenosis, 60% L ICA stenosis. The cardiac monitor demonstrates atrial fibrillation and echocardiogram demonstrates septal akinesia, consistent with recent MI. What is the likely cause of this patient's stroke?

- ○ Cardiac mural thrombus
- ○ Left carotid artery stenosis
- ○ Right carotid artery stenosis
- ○ Hypotension during cardiac arrest
- ○ Cardioembolic disease

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Question 21 of 299 ✗

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Cardiac mural thrombus	17%
Left carotid artery stenosis	3%
Right carotid artery stenosis	2%
🟩 Hypotension during cardiac arrest	44%
🟥 Cardioembolic disease	35%

The neuroimaging describes a posterior watershed infarct, in which the areas of ischaemia lie in the most vulnerable regions of the brain in between two cerebral artery territories. Embolic disease and cardiac thrombi can produce bilateral symptoms. However, the specific areas involved suggest regions most prone to ischaemia during the period of systemic hypoperfusion. The two broad categories of differentials to consider include cardiac pump failure and hypovolaemia. In this case, hypoperfusion due to cardiac arrest is most likely.

Discuss (3) Improve

Next question ›

Hypoperfusion strokes

Hypoperfusion tends to cause brain injuries at 'watershed' areas that are border zones between the major cerebral arteries.

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **16%**

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Question 22 of 299

A 52-year-old male is brought to the emergency department by his concerned wife after tripping and falling down a flight of 12 stairs at home, hitting his head on the way down. The patient himself is not concerned and believes he could have stayed at home.

He denies having a headache, reports no nausea or vomiting, no seizures and did not lose consciousness between the fall and when you examine him. He is not taking any regular medications including anticoagulants and remembers the whole episode except for about 20 seconds after landing at the bottom of the floor. On examination, he has no limb weakness or loss of sensation. His pupils are equal and reactive bilaterally. What is the most appropriate management?

- ○ Discharge, no further investigations required
- ○ Discharge, outpatient CT head within 72 hours
- ○ Observe for 24 hours and discharge if no deterioration
- ○ CT head immediately
- ○ CT head within 8 hours of injury

Submit answer

Reference ranges

Score: **0%**

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He denies having a headache, reports no nausea or vomiting, no seizures and did not lose consciousness between the fall and when you examine him. He is not taking any regular medications including anticoagulants and remembers the whole episode except for about 20 seconds after landing at the bottom of the floor. On examination, he has no limb weakness or loss of sensation. His pupils are equal and reactive bilaterally. What is the most appropriate management?

Discharge, no further investigations required	13%
Discharge, outpatient CT head within 72 hours	5%
Observe for 24 hours and discharge if no deterioration	16%
CT head immediately	23%
CT head within 8 hours of injury	44%

CT head within 8 hours of injury is the correct answer according to NICE guidelines for head injury assessment. This patient has experienced a dangerous mechanism of injury (fall down >3 steps) and has retrograde amnesia (loss of memory for 20 seconds after the event), which are both risk factors that warrant a CT head scan within 8 hours, even in the absence of other concerning features.

CT head immediately would be indicated if the patient had additional concerning features such as GCS <13, focal neurological deficit, seizure, vomiting, or was on anticoagulation. As none of these are present, scanning within 8 hours rather than immediately is appropriate.

Observe for 24 hours and discharge if no deterioration would be excessive for this patient. In-patient observation is typically reserved for patients with more concerning features or those who have had abnormal imaging. This would be an inefficient use of resources for this patient.

Discharge, outpatient CT head within 72 hours is inappropriate as the timeframe is too long. If imaging is indicated in head injury, it should be performed within 8 hours to identify potentially serious pathology that may require intervention.

Discharge, no further investigations required would be unsafe. While the patient appears well, the mechanism of injury and presence of retrograde amnesia mandate further investigation as per NICE guidelines. Missing an intracranial injury could have serious consequences.

The key principle in head injury management is to identify patients who require imaging based on risk factors, even if they appear well, as early intervention for intracranial pathology leads to better outcomes.

👍 👎 Discuss (5) Improve

Next question >

Head injury: NICE guidance on investigation

NICE has strict and clear guidance regarding which adult patients are safe to discharge and which need further CT head imaging. The latter group are also divided into two further cohorts, those who require an immediate CT head and those requiring CT head within 8 hours of injury:

CT head within 1 hour
- GCS < 13 on initial assessment
- GCS < 15 at 2 hours post-injury
- suspected open or depressed skull fracture
- any sign of basal skull fracture (haemotympanum, 'panda' eyes, cerebrospinal fluid leakage from the ear or nose, Battle's sign).
- post-traumatic seizure.
- focal neurological deficit.
- more than 1 episode of vomiting

CT head scan within 8 hours of the head injury - for adults with any of the following risk factors who have experienced some loss of consciousness or amnesia since the injury:
- age 65 years or older
- any history of bleeding or clotting disorders including anticogulants
- dangerous mechanism of injury (a pedestrian or cyclist struck by a motor vehicle, an occupant ejected from a motor vehicle or a fall from a height of greater than 1 metre or 5 stairs)
- more than 30 minutes' retrograde amnesia of events immediately before the head injury

If a patient is on warfarin who have sustained a head injury with no other indications for a CT head scan, perform a CT head scan within 8 hours of the injury.

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

NICE ✓ 12 ✗ 8

2014 Head injury guidelines

Suggest link Report broken link

Score: **16%**

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A 19 year old woman presents to the general medical clinic for review. She has been referred due to drooping of her eyelids, first on the right and now bilaterally. On further questioning she reveals that she has been struggling to read in dim light and recalls that two family members have required insertion of cardiac pacemakers. Examination reveals partial bilateral ptosis and a generalised ophthalmoplegia in all directions of gaze. Fundoscopy demonstrates central areas of dark pigmentation on a pale fundus.

From the options given which investigation is most likely to reveal the diagnosis?

- ○ Serum for anti-acetylcholine receptor antibodies
- ○ Serum for anti-voltage gated calcium channel receptor antibodies
- ○ Muscle biopsy
- ○ Karotyping
- ○ Thyroid function tests

Submit answer

Reference ranges

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A 19 year old woman presents to the general medical clinic for review. She has been referred due to drooping of her eyelids, first on the right and now bilaterally. On further questioning she reveals that she has been struggling to read in dim light and recalls that two family members have required insertion of cardiac pacemakers. Examination reveals partial bilateral ptosis and a generalised ophthalmoplegia in all directions of gaze. Fundoscopy demonstrates central areas of dark pigmentation on a pale fundus.

From the options given which investigation is most likely to reveal the diagnosis?

Serum for anti-acetylcholine receptor antibodies	10%
Serum for anti-voltage gated calcium channel receptor antibodies	8%
Muscle biopsy	47%
Karotyping	34%
Thyroid function tests	1%

A triad of chronic progressive external ophthalmoplegia, pigmentary retinopathy and cardiac conduction defects makes Kearns-Sayer syndrome likely.

Kearns-Sayer syndrome is an example of a mitochondrial disease, a group of rare conditions which many find confusing. Despite their rarity, those candidates who do a rotation in neurology are likely to encounter mitochondrial disease in one form or another and so a basic knowledge is useful.

Kearns-Sayer syndrome is characterised by ptosis and external ophthalmoplegia occurring in the first or second decades of life. Cardiac conduction defects usually occur later and are usually in the form of atrio-ventricular nodal block, often requiring cardiac pacing. Multiple endocrinopathies may be associated and a proximal myopathy may be evident. Serum elevation of lactate and pyruvate is frequently seen, as is the case with many mitochondrial conditions.

The diagnosis should be suspected from the history and clinical features as listed above and diagnosis is confirmed by muscle biopsy which demonstrates characteristic 'ragged red fibres', this is often supplemented by polymerase chain reaction analysis of mitochondrial DNA for mutation detection.

Anti-acetylcholine receptor antibodies are seen in myasthenia gravis, and would be involved in the work-up of any patient with an unexplained bilateral ptosis, but this wouldn't explain the pigmented retinopathy or the family history of pacemaker insertion. This is also the case for anti-voltage gated calcium channel antibodies, found in the Lambert-Eaton myasthenic syndrome.

Discuss (3) | Improve

Next question ›

Mitochondrial diseases

Whilst most DNA is found in the cell nucleus, a small amount of double-stranded DNA is present in the mitochondria. It encodes protein components of the respiratory chain and some special types of RNA

Mitochondrial inheritance has the following characteristics:

- inheritance is only via the maternal line as the sperm contributes no cytoplasm to the zygote
- none of the children of an affected male will inherit the disease
- all of the children of an affected female will inherit the disease
- generally, encode rare neurological diseases
- poor genotype:phenotype correlation - within a tissue or cell there can be different mitochondrial populations - this is known as heteroplasmy

Histology

- muscle biopsy classically shows 'red, ragged fibres' due to increased number of mitochondria

Examples include:

- Leber's optic atrophy
  - symptoms typically develop at around the age of 30 years
  - central scotoma → loss of colour vision → rapid onset of significant visual impairment
- MELAS syndrome: mitochondrial encephalomyopathy lactic acidosis and stroke-like episodes
- MERRF syndrome: myoclonus epilepsy with ragged-red fibres
- Kearns-Sayre syndrome: onset in patients < 20 years old, external ophthalmoplegia, retinitis pigmentosa. Ptosis may be seen
- sensorineural hearing loss

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 5 👎 6

2013 How to spot mitochondrial disease in adults

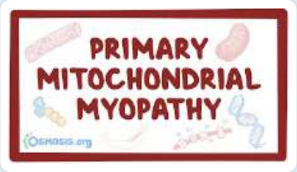
Rare Disease Video Library - NORD 👍 3 👎 4

Primary Mitochondrial Myopathy

Suggest link

Report broken link

Media


PRIMARY
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Osmosis.org

Primary mitochondrial myopathy

Osmosis - YouTube 👍 6 👎 1

Report broken media

Score: **16%**

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A 70-year-old man with a history of hypertension and benign prostatic hypertrophy is brought in by ambulance after a fall. He reports he felt dizzy after standing up from his arm chair, stumbled and tripped over his cat. His wife, who witnessed the fall, reports that he then hit his head on the coffee table a lost consciousness for around 1 minute.

She describes no abnormal movements or incontinence. On regaining consciousness he was oriented immediately. He remembers regaining consciousness. He has no headache, dizziness, nausea or vomiting.

On examination, he has a small laceration on his forehead. His pupils were equal and reactive to light. He had no focal neurological deficits. He was a 15 on the Glasgow Coma Scale. His abbreviated mental test score was 10/10.

ECG: Sinus rhythm. 70 beats per minute. No T wave or ST segment changes.

Blood pressure (lying): 135/75 mmHg
Blood pressure (standing): 110/60 mmHg

Haemoglobin	135 g/dl
Troponin T	1 ng/L

Urine dip: trace of protein

What is the most appropriate course of action?

- ○ Admit for CT head within 1 hour
- ○ Admit for CT head within 8 hours
- ○ Admit for CT head within 24 hours
- ○ Discharge to return for CT head next day
- ○ Discharge with outpatient follow-up, no imaging required

Submit answer

Reference ranges

Score: **0%**

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Question 24 of 299

A 70-year-old man with a history of hypertension and benign prostatic hypertrophy is brought in by ambulance after a fall. He reports he felt dizzy after standing up from his arm chair, stumbled and tripped over his cat. His wife, who witnessed the fall, reports that he then hit his head on the coffee table a lost consciousness for around 1 minute.

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On examination, he has a small laceration on his forehead. His pupils were equal and reactive to light. He had no focal neurological deficits. He was a 15 on the Glasgow Coma Scale. His abbreviated mental test score was 10/10.

ECG: Sinus rhythm. 70 beats per minute. No T wave or ST segment changes.

Blood pressure (lying): 135/75 mmHg
Blood pressure (standing): 110/60 mmHg

Haemoglobin	135 g/dl
Troponin T	1 ng/L

Urine dip: trace of protein

What is the most appropriate course of action?

Admit for CT head within 1 hour	24%
Admit for CT head within 8 hours	46%
Admit for CT head within 24 hours	11%
Discharge to return for CT head next day	1%
Discharge with outpatient follow-up, no imaging required	18%

Nice Guidelines on head injury state that:

'For adults who have sustained a head injury and have any of the following risk factors, perform a CT head scan within 1 hour of the risk factor being identified:

- GCS less than 13 on initial assessment in the emergency department.
- GCS less than 15 at 2 hours after the injury on assessment in the Emergency Department.
- Suspected open or depressed skull fracture.
- Any sign of basal skull fracture (haemotympanum, 'panda' eyes, cerebrospinal fluid leakage from the ear or nose, Battle's sign).
- Post-traumatic seizure.
- Focal neurological deficit.
- More than 1 episode of vomiting.'

The patient does not meet any of these criteria, so CT head within 1 hour is not mandatory.

The guideline then also states:

'For adults with any of the following risk factors who have experienced some loss of consciousness or amnesia since the injury, perform a CT head scan within 8 hours of the head injury:

- Age 65 years or older.
- Any history of bleeding or clotting disorders.
- Dangerous mechanism of injury (a pedestrian or cyclist struck by a motor vehicle, an occupant ejected from a motor vehicle or a fall from a height of greater than 1 metre or 5 stairs).
- More than 30 minutes' retrograde amnesia of events immediately before the head injury.'

As this gentleman had a period of unconsciousness and is over 65, he should have a CT head within 8 hours.

👍 👎 Discuss (4) Improve

Next question >

Head injury: NICE guidance on investigation

NICE has strict and clear guidance regarding which adult patients are safe to discharge and which need further CT head imaging. The latter group are also divided into two further cohorts, those who require an immediate CT head and those requiring CT head within 8 hours of injury:

CT head within 1 hour

- GCS < 13 on initial assessment
- GCS < 15 at 2 hours post-injury
- suspected open or depressed skull fracture

- any sign of basal skull fracture (haemotympanum, 'panda' eyes, cerebrospinal fluid leakage from the ear or nose, Battle's sign).
- post-traumatic seizure.
- focal neurological deficit.
- more than 1 episode of vomiting

CT head scan within 8 hours of the head injury - for adults with any of the following risk factors who have experienced some loss of consciousness or amnesia since the injury:

- age 65 years or older
- any history of bleeding or clotting disorders including anticogulants
- dangerous mechanism of injury (a pedestrian or cyclist struck by a motor vehicle, an occupant ejected from a motor vehicle or a fall from a height of greater than 1 metre or 5 stairs)
- more than 30 minutes' retrograde amnesia of events immediately before the head injury

If a patient is on warfarin who have sustained a head injury with no other indications for a CT head scan, perform a CT head scan within 8 hours of the injury.

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 12 👎 8

2014 Head injury guidelines

Suggest link Report broken link

Score: **16%**

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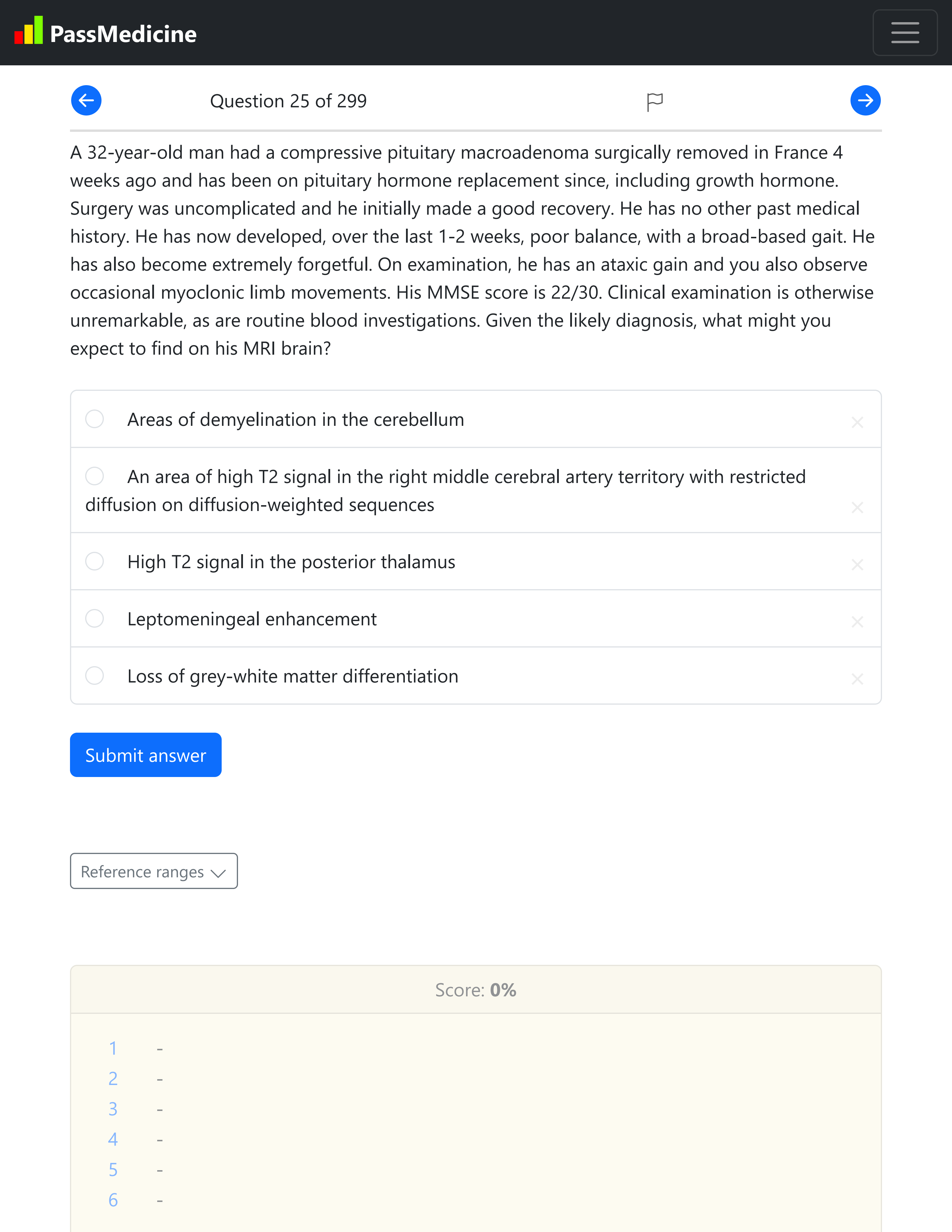

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Question 25 of 299

A 32-year-old man had a compressive pituitary macroadenoma surgically removed in France 4 weeks ago and has been on pituitary hormone replacement since, including growth hormone. Surgery was uncomplicated and he initially made a good recovery. He has no other past medical history. He has now developed, over the last 1-2 weeks, poor balance, with a broad-based gait. He has also become extremely forgetful. On examination, he has an ataxic gain and you also observe occasional myoclonic limb movements. His MMSE score is 22/30. Clinical examination is otherwise unremarkable, as are routine blood investigations. Given the likely diagnosis, what might you expect to find on his MRI brain?

- ○ Areas of demyelination in the cerebellum
- ○ An area of high T2 signal in the right middle cerebral artery territory with restricted diffusion on diffusion-weighted sequences
- ○ High T2 signal in the posterior thalamus
- ○ Leptomeningeal enhancement
- ○ Loss of grey-white matter differentiation

Score: **0%**

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Areas of demyelination in the cerebellum	24%
An area of high T2 signal in the right middle cerebral artery territory with restricted diffusion on diffusion-weighted sequences	15%
High T2 signal in the posterior thalamus	37%
Leptomeningeal enhancement	13%
Loss of grey-white matter differentiation	11%

The diagnosis is Creutzfeldt-Jakob disease (CJD) from an iatrogenic source.

The use of prion-contaminated medication, grafts and instruments may result in iatrogenic CJD. The current worldwide total of growth-hormone-associated cases of CJD is 226. Most cases occurred in France (119 cases/1,880 recipients; attack rate 6.3%), the United Kingdom (65 cases/1,800 recipients; attack rate 3.6%), and the United States (29 cases/7,700 recipients; attack rate 0.4%). No new case has been identified since 2008 but would be important to recognise this condition and how it presents if it did, as it is invariably fatal -so patients need to be made aware of their prognosis.

The clinical features of the disease vary according to the route of inoculation.

Central inoculation, eg from infected neurosurgical instruments, results in a rapidly progressive neurodegenerative disease similar to sporadic CJD, characterised by ataxia, dementia, myoclonus, rigidity, and akinetic mutism. Survival is 2-12 months.

Peripheral inoculation, eg from the use of the old human growth hormone products, results in a slightly less acute disease -characterised by progressive cerebellar ataxia and dementia. Survival is 8-18 months.

Discuss (10) | Improve

Next question

Creutzfeldt-Jakob disease

Creutzfeldt-Jakob disease (CJD) is rapidly progressive neurological condition caused by prion proteins. These proteins induce the formation of amyloid folds resulting in tightly packed beta-pleated sheets resistant to proteases.

Features
- dementia (rapid onset)
- myoclonus

Investigation
- CSF is usually normal
- EEG: biphasic, high amplitude sharp waves (only in sporadic CJD)
- MRI: hyperintense signals in the basal ganglia and thalamus

Sporadic CJD
- accounts for 85% of cases
- 10-15% of cases are familial
- mean age of onset is 65 years

New variant CJD
- younger patients (average age of onset = 25 years)
- psychological symptoms such as anxiety, withdrawal and dysphonia are the most common presenting features
- the 'prion protein' is encoded on chromosome 20 - it's role is not yet understood
- methionine homozygosity at codon 129 of the prion protein is a risk factor for developing CJD - all patients who have so far died have had this
- median survival = 13 months

Other prion diseases
- kuru

- fatal familial insomnia
- Gerstmann Straussler-Scheinker disease

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

PRIONS
(SPONGIFORM
ENCEPHALOPATHY)

Prions (Spongiform encephalopathy)

Osmosis - YouTube 👍 7 👎 0

Report broken media

Score: **17.6%**

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Question 26 of 299

A 75 year old man with Parkinson's disease is brought to see you in clinic by his son. He has become increasingly concerned that his neighbours have been watching him, and have put wiring throughout his walls to monitor his movements. He has also been describing visual hallucinations of animals climbing up his walls. His son is concerned that he has become increasingly anxious. He is currently on co-careldopa, ropinirole, and rasagiline. What is the best course of action?

○ Initiate a 'drug holiday' withholding all but co-careldopa for 1 week ×

○ Reduce ropinirole ×

○ Reduce rasagiline ×

○ Refer for cognitive behavioural therapy ×

○ Start quetiapine ×

Submit answer

Reference ranges

Score: **16%**

1 ✗
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A 75 year old man with Parkinson's disease is brought to see you in clinic by his son. He has become increasingly concerned that his neighbours have been watching him, and have put wiring throughout his walls to monitor his movements. He has also been describing visual hallucinations of animals climbing up his walls. His son is concerned that he has become increasingly anxious. He is currently on co-careldopa, ropinirole, and rasagiline. What is the best course of action?

Initiate a 'drug holiday' withholding all but co-careldopa for 1 week	20%
Reduce ropinirole	46%
Reduce rasagiline	10%
Refer for cognitive behavioural therapy	3%
Start quetiapine	20%

This gentleman is displaying psychotic symptoms. Psychosis in Parkinson's disease (PD) can be due to the disease process itself (non-motor feature of PD), or medication side effect. It is sensible to perform a medication review and alter any potentially precipitating drugs.

Ropinirole is a dopamine agonist and is associated with psychosis, and in particular visual hallucinations. It is also important to warn all patients initiated on dopamine agonists of the potential for impulsive behaviour disorders including gambling and excessive spending.

Abrupt discontinuation of medication is never indicated in Parkinson's disease since this can lead to rapid deterioration and worsening of symptoms.

Discuss (4) Improve

Next question >

Parkinson's disease: management ★

Parkinsons disease should only be diagnosed, and management initiated, by a specialist with expertise in movement disorders. However, it is important for all doctors to be aware of the medications used in Parkinson's given the prevalence of this condition. NICE published guidelines in 2017 regarding the management of Parkinson's disease.

For first-line treatment:

- if the motor symptoms are affecting the patient's quality of life: levodopa
- if the motor symptoms are not affecting the patient's quality of life: dopamine agonist (non-ergot derived), levodopa or monoamine oxidase B (MAO-B) inhibitor

Whilst all drugs used to treat Parkinson's can cause a wide variety of side-effects NICE produced tables to help with decision making:

	Levodopa	**Dopamine agonists**	**MAO-B inhibitors**
Motor symptoms	More improvement in motor symptoms	Less improvement in motor symptoms	Less improvement in motor symptoms
Activities of daily living	More improvement in activities of daily living	Less improvement in activities of daily living	Less improvement in activities of daily living
Motor complications	More motor complications	Fewer motor complications	Fewer motor complications
Adverse events	Fewer specified adverse events*	More specified adverse events*	Fewer specified adverse events*

* excessive sleepiness, hallucinations and impulse control disorders

If a patient continues to have symptoms despite optimal levodopa treatment or has developed dyskinesia then NICE recommend the addition of a dopamine agonist, MAO-B inhibitor or catechol-O-methyl transferase (COMT) inhibitor as an adjunct. Again, NICE summarise the main points in terms of decision making:

	Dopamine agonists	**MAO-B inhibitors**	**COMT inhibitors**	**Amantadine**
Motor symptoms	Improvement in motor symptoms	Improvement in motor symptoms	Improvement in motor symptoms	No evidence of improvement in motor symptoms
Activities of daily living	Improvement in activities of daily living	Improvement in activities of daily living	Improvement in activities of daily living	No evidence of improvement in activities of daily living

	Dopamine agonists	MAO-B inhibitors	COMT inhibitors	Amantadine
Off time	More off-time reduction	Off-time reduction	Off-time reduction	No studies reporting this outcome
Adverse events	Intermediate risk of adverse events	Fewer adverse events	More adverse events	No studies reporting this outcome
Hallucinations	More risk of hallucinations	Lower risk of hallucinations	Lower risk of hallucinations	No studies reporting this outcome

Specific points regarding Parkinson's medication

NICE reminds us of the risk of acute akinesia or neuroleptic malignant syndrome if medication is not taken/absorbed (for example due to gastroenteritis) and advise against giving patients a 'drug holiday' for the same reason.

Impulse control disorders have become a significant issue in recent years. These can occur with any dopaminergic therapy but are more common with:
- dopamine agonist therapy
- a history of previous impulsive behaviours
- a history of alcohol consumption and/or smoking

If excessive daytime sleepiness develops then patients should not drive. Medication should be adjusted to control symptoms. Modafinil can be considered if alternative strategies fail.

If orthostatic hypotension develops then a medication review looking at potential causes should be done. If symptoms persist then midodrine (acts on peripheral alpha-adrenergic receptors to increase arterial resistance) can be considered.

Consider glycopyrronium bromide to manage drooling of saliva in people with Parkinson's disease.

Further information regarding specific anti-Parkinson's medication

Levodopa

- nearly always combined with a decarboxylase inhibitor (e.g. carbidopa or benserazide)
  - this prevents the peripheral metabolism of levodopa to dopamine outside of the brain and hence can reduce side effects
- common adverse effects:
  - dry mouth
  - anorexia
  - palpitations
  - postural hypotension
  - psychosis
- some adverse effects are due to the difficulty in achieving a steady dose of levodopa
  - end-of-dose wearing off: symptoms often worsen towards the end of dosage interval. This results in a decline of motor activity
  - 'on-off' phenomenon: large variations in motor performance, with normal function during the 'on' period, and weakness and restricted mobility during the 'off' period
  - dyskinesias at peak dose: dystonia, chorea and athetosis (involuntary writhing movements)
  - these effects may worsen over time with - clinicians therefore may limit doses until necessary
- it is important not to acutely stop levodopa, for example, if a patient is admitted to hospital
  - if a patient with Parkinson's disease cannot take levodopa orally, they can be given a dopamine agonist patch as rescue medication to prevent acute dystonia

Dopamine receptor agonists

- e.g. bromocriptine, ropinirole, cabergoline, apomorphine
- ergot-derived dopamine receptor agonists (bromocriptine, cabergoline) have been associated with pulmonary, retroperitoneal and cardiac fibrosis. The Committee on Safety of Medicines advice that an echocardiogram, ESR, creatinine and chest x-ray should be obtained prior to treatment and patients should be closely monitored
- patients should be warned about the potential for dopamine receptor agonists to cause impulse control disorders and excessive daytime somnolence
- more likely than levodopa to cause hallucinations in older patients. Nasal congestion and postural hypotension are also seen in some patients

MAO-B (Monoamine Oxidase-B) inhibitors

- e.g. selegiline
- inhibits the breakdown of dopamine secreted by the dopaminergic neurons

Amantadine

- mechanism is not fully understood, probably increases dopamine release and inhibits its uptake at dopaminergic synapses
- side-effects include ataxia, slurred speech, confusion, dizziness and livedo reticularis

COMT (Catechol-O-Methyl Transferase) inhibitors

- e.g. entacapone, tolcapone
- COMT is an enzyme involved in the breakdown of dopamine, and hence may be used as an adjunct to levodopa therapy
- used in conjunction with levodopa in patients with established PD

Antimuscarinics

- block cholinergic receptors
- now used more to treat drug-induced parkinsonism rather than idiopathic Parkinson's disease
- help tremor and rigidity
- e.g. procyclidine, benzotropine, trihexyphenidyl (benzhexol)

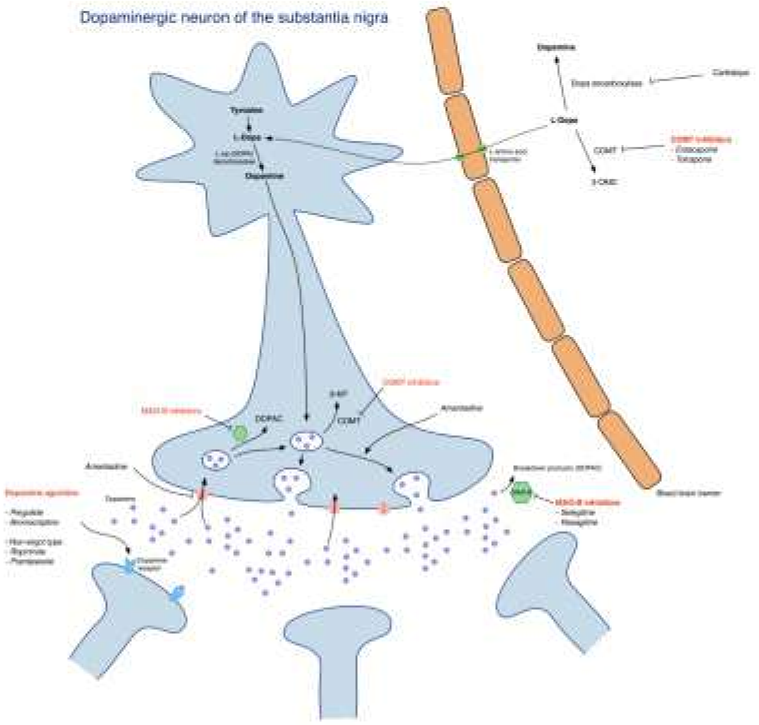

Dopaminergic neuron of the substantia nigra
Tyrosine
L-Dopa
Dopamine
L-Dopa
COMT
3-OMD
DOPAC
3-MT
COMT
Amantadine
Amantadine

Image sourced from Wikipedia

Diagram showing the mechanism of action of Parkinson's drugs

123

Next question

Textbooks

High-yield textbook

Extended textbook

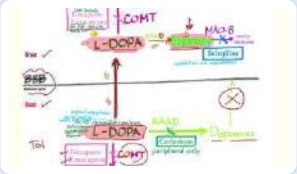
Links

NICE 👍 23 👎 12

2017 Parkinson's disease guidelines

Suggest link Report broken link

Media


COMT
L-DOPA
MAO-B
L-DOPA
AADC

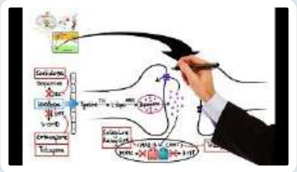
The use of Levodopa/ Carbidopa in the treatment of Parkinson's disease

Medicosis Perfectionalis - YouTube 👍 13 👎 0

PARKINSON'S
DISEASE
PHARMACOLOGY

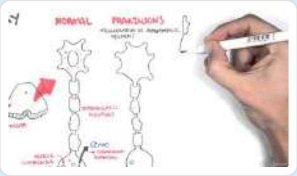
Parkinson's disease pharmacology

Soton Brain Hub - YouTube 👍 4 👎 0



Pharmacology - DRUGS FOR PARKINSON'S DISEASE

Speed Pharmacology - YouTube 👍 8 👎 1


NORMAL
PARKINSON'S

Pharmacology - Parkinson's Disease

Armando Hasudungan - YouTube 👍 16 👎 2

PARKINSON'S DISEASE
Osmosis.org

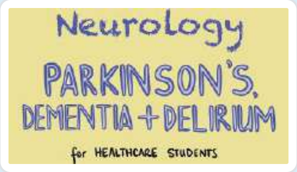
Parkinson's disease

Osmosis - YouTube 👍 4 👎 1

PARKINSON'S DISEASE
SHUFFLING GAIT
α-SYNUCLEIN
PILL ROLLING TREMOR
COG-WHEEL RIGIDITY
DaT SCAN

Understanding Parkinson's Disease

Rhesus Medicine - YouTube 👍 2 👎 1


Neurology
PARKINSON'S, DEMENTIA + DELIRIUM
for HEALTHCARE STUDENTS

Parkinson's disease

MedFlix - YouTube 👍 1 👎 2

Report broken media

Score: **17.6%**

1 ✗
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Back to top

You are asked for advice on a 57-year-old man who presented to primary care 12 weeks after an inferior myocardial infarction. He has had symptoms of persistent low mood that varies during the day, tearfulness and hopelessness. He has lost 6kg of weight in the past 3 months and his BMI is currently 19. He has fleeting thoughts of suicide but would not act on this as he does not want to hurt his family. You are told that 20 years ago, following an overdose of paracetamol, he was prescribed Citalopram and diagnosed with depression.

The GP tells you that other than the recent MI, he also has a diagnosis of Atrial Fibrillation, for which he is on warfarin and he has suffered from epigastric pain for the past 2 years. He had an OGD 1 year ago where treatment of a duodenal ulcer with adrenaline was required. Since then his symptoms of epigastric pain have decreased but are still present. There has been no repeat OGD.

What would be the safest course of action?

○ Prescribe Phenelzine ×
○ Prescribe Sertraline ×
○ Prescribe Mirtazapine ×
○ Prescribe Imipramine ×
○ Refer for 2 month wait CBT ×

Submit answer

Reference ranges

Score: **16%**

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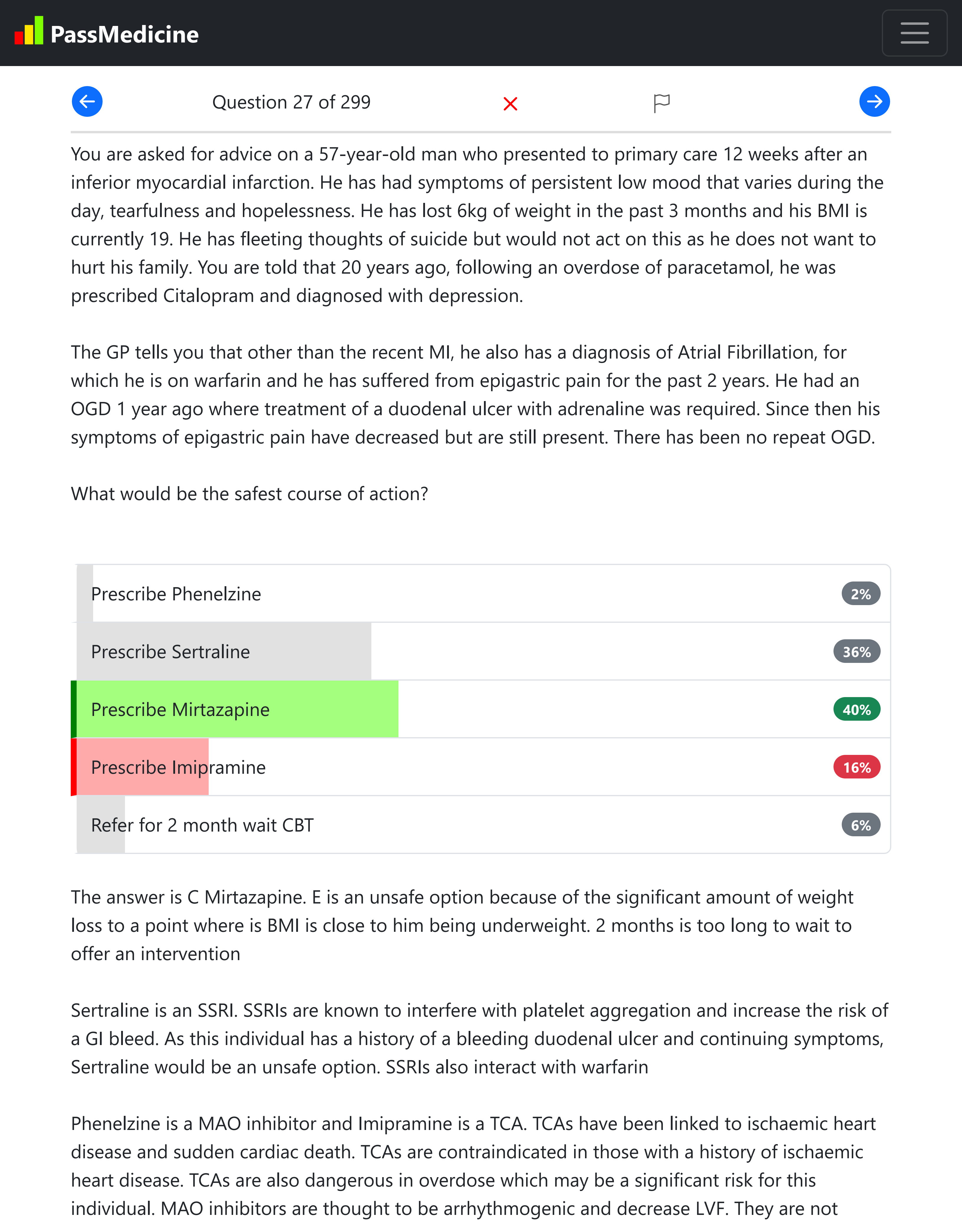

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What would be the safest course of action?

Prescribe Phenelzine	2%
Prescribe Sertraline	36%
Prescribe Mirtazapine	40%
Prescribe Imipramine	16%
Refer for 2 month wait CBT	6%

The answer is C Mirtazapine. E is an unsafe option because of the significant amount of weight loss to a point where is BMI is close to him being underweight. 2 months is too long to wait to offer an intervention

Sertraline is an SSRI. SSRIs are known to interfere with platelet aggregation and increase the risk of a GI bleed. As this individual has a history of a bleeding duodenal ulcer and continuing symptoms, Sertraline would be an unsafe option. SSRIs also interact with warfarin

Phenelzine is a MAO inhibitor and Imipramine is a TCA. TCAs have been linked to ischaemic heart disease and sudden cardiac death. TCAs are contraindicated in those with a history of ischaemic heart disease. TCAs are also dangerous in overdose which may be a significant risk for this individual. MAO inhibitors are thought to be arrhythmogenic and decrease LVF. They are not recommended in those with cardiovascular disease.

Mirtazapine is a good choice as it is not known to cause cardiac conduction disturbances and there is good evidence of its safety post-MI. It also causes increased appetite and weight gain which would be advantageous for this individual at this time. Mirtazapine does not interact with warfarin and it does not affect platelet aggregation. Generally speaking, Mirtazapine is a safe drug and is often prescribed for the elderly and people with multiple medical problems. Mirtazapine is also relatively safe in overdose to other antidepressants.

👍 👎 💬 Discuss (6) Improve

Next question >

Depression: screening and assessment ★

Screening

The following two questions can be used to screen for depression
- 'During the last month, have you often been bothered by feeling down, depressed or hopeless?'
- 'During the last month, have you often been bothered by having little interest or pleasure in doing things?'

A 'yes' answer to either of the above should prompt a more in depth assessment.

Assessment

There are many tools to assess the degree of depression including the Hospital Anxiety and Depression (HAD) scale and the Patient Health Questionnaire (PHQ-9).

Hospital Anxiety and Depression (HAD) scale
- consists of 14 questions, 7 for anxiety and 7 for depression
- each item is scored from 0-3
- produces a score out of 21 for both anxiety and depression
- severity: 0-7 normal, 8-10 borderline, 11+ case
- patients should be encouraged to answer the questions quickly

Patient Health Questionnaire (PHQ-9)

- asks patients 'over the last 2 weeks, how often have you been bothered by any of the following problems?'
- 9 items which can then be scored 0-3
- includes items asking about thoughts of self-harm

Traditionally, depression severity has been grouped under 4 categories (subthreshold, mild, moderate and severe). The updated NICE guideline uses a simpler 2 category definition of depression: less severe or more severe depression.

Less severe depression encompasses subthreshold and mild depression, and more severe depression encompasses moderate and severe depression. Thresholds on validated scales were used in this guideline as an indicator of severity

- a score < 16 on the PHQ-9: less severe depression
- a score of ≥ 16 on the PHQ-9: severe depression

The DSM-5 also provides criteria for diagnosing major depressive disorder (MDD), commonly referred to as depression.

Major Depressive Disorder (MDD) DSM-5 Criteria:

Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

- Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad, empty, hopeless) or observation made by others (e.g., appears tearful). (Note: In children and adolescents, can be irritable mood.)
- Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation).
- Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. (Note: In children, consider failure to make expected weight gain.)
- Insomnia or hypersomnia nearly every day.
- Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).
- Fatigue or loss of energy nearly every day.
- Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).
- Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).
- Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 23 10

PHQ 9 form

Suggest link Report broken link

Media

LOW MOOD
ANHEDONIA
SEROTONIN
MAJOR DEPRESSIVE DISORDER
ELECTROCONVULSION

Depression Explained (Major Depressive Disorder)

Rhesus Medicine - YouTube 0 1

Report broken media

Score: **17.6%**

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A 28-year-old female psychiatric nurse presents to the emergency department following a 2-minute tonic-clonic generalised seizure, which self-terminated. Whilst taking the history the following morning, she tells you that over the last 48 hours she has become increasingly unwell. She has been feeling increasingly anxious and has been having insomnia and vivid nightmares which wake her from sleep. She says that everything around her no longer looks real but more like a photocopy. Bowel and bladder movements have been normal. She feels generally weak and asks the lights to be dimmed in the examination room. When the nurse bell goes off in the next cubicle, she has to cover her ears.

On examination, she appears anxious, she is perspiring, respiratory rate 16/min, blood pressure 142/86 mmHg, heart rate 115/min regular, sats 98% on air. You notice a fine tremor, especially in the hands and eyelids. GCS = 15. Oriented in time place and person. When you examine her, she tells you that it feels as if her legs are floating off the bed even though they are stationary. There is no flushing of the face

From looking at the computer records, you can see that she has a history of panic disorder treated with PRN lorazepam and sertraline 1 year ago. and has been treated for depression in the past. You also note from your records that she presented to A+E 5 days ago due to stress as she was sacked from her job.

Hb	136 g/l
Platelets	232 * 10^9/l
WBC	6.9 * 10^9/l

Na^+	142 mmol/l
K^+	3.8 mmol/l
Urea	6.2 mmol/l
Creatinine	81 µmol/l

What is the most likely diagnosis?

○ Benzodiazepine withdrawal ×

○ Atypical Panic attack secondary to panic disorder ×

○ LSD intoxication ×

○ Benzodiazepine excess ×

○ Opiate withdrawal ×

Submit answer

Reference ranges ⌄

Score: **16%**

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WBC	6.9 * 10^9/l

Na^+	142 mmol/l
K^+	3.8 mmol/l
Urea	6.2 mmol/l
Creatinine	81 µmol/l

What is the most likely diagnosis?

Benzodiazepine withdrawal 43%

Atypical Panic attack secondary to panic disorder 26%

LSD intoxication 21%

Benzodiazepine excess 7%

Opiate withdrawal 3%

Benzodiazepine withdrawal can cause physical symptoms such as tachycardia, sweating, fine tremor which is more prominent in the tongue, eyelids and hands. It can also cause nightmares and insomnia, anxiety and phobic symptoms as well as seizures, hypersensitivity to light, sounds and touch. They can also experience derealisation (an unpleasant subjective experience that the outside world does not look real to them) and kinaesthetic hallucinations. Other symptoms include malaise and tinnitus and it sometimes can present as a delirium.

Some factors in the history also point towards this as being a likely diagnosis. She has a history of panic disorder and previous prescription for benzodiazepines when she may have developed dependence. As a psychiatric nurse, she may have access to benzodiazepines off prescription. The fact that she has recently lost her job may mean that she has run out of her supply and is now suffering from withdrawal syndrome.

An overdose of benzodiazepines would not present with tachycardia, sweating and anxiety. LSD intoxication could present with hallucinations, anxiety, tachycardia and sweating, however the duration of symptoms is too long for LSD unless multiple doses were taken and LSD intoxication. LSD is not linked to causing seizures. This presentation has many features of a panic attack but would not explain the seizure or kinaesthetic hallucination.

Opiate withdrawal could present in a similar way, however, you would expect to see a marked degree of GI disturbance in this syndrome and is not linked with kinaesthetic hallucinations or derealisation. In this question, the history of previous benzodiazepine usage makes this answer less likely

Discuss (5) Improve

Next question ›

Benzodiazepines ★

Benzodiazepines enhance the effect of the inhibitory neurotransmitter gamma-aminobutyric acid (GABA) by increasing the **frequency** of chloride channels. They therefore are used for a variety of purposes:

- sedation
- hypnotic
- anxiolytic
- anticonvulsant
- muscle relaxant

Patients commonly develop a tolerance and dependence to benzodiazepines and care should therefore be exercised on prescribing these drugs. The Committee on Safety of Medicines advises that benzodiazepines are only prescribed for a short period of time (2-4 weeks).

The BNF gives advice on how to withdraw a benzodiazepine. The dose should be withdrawn in steps of about 1/8 (range 1/10 to 1/4) of the daily dose every fortnight. A suggested protocol for patients experiencing difficulty is given:

- switch patients to the equivalent dose of diazepam
- reduce dose of diazepam every 2-3 weeks in steps of 2 or 2.5 mg
- time needed for withdrawal can vary from 4 weeks to a year or more

If patients withdraw too quickly from benzodiazepines they may experience benzodiazepine withdrawal syndrome, a condition very similar to alcohol withdrawal syndrome. This may occur up to 3 weeks after stopping a long-acting drug. Features include:

- insomnia
- irritability
- anxiety
- tremor
- loss of appetite
- tinnitus
- perspiration
- perceptual disturbances
- seizures

$GABA_A$ drugs

- benzodiazipines increase the **frequency** of chloride channels
- barbiturates increase the **duration** of chloride channel opening

Frequently **Ben**d - **Dur**ing **Barb**eque

...or...

Barbi**durat**es increase **durat**ion & **Fre**ndodiazepines increase **fre**quency

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

Benzodiazepines
Barbiturates
Non-benzodiazepine Hypnotics

Benzodiazepines, bariburates and hypnotics (made easy)

Speed Pharmacology - YouTube 👍 1 👎 0

Report broken media

Score: **17.6%**

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A 64 year old man presented to the Emergency Department after becoming unwell at home. His wife reported that the patient experienced a sudden onset and severe headache while watching television. Shortly afterwards she found him to have become confused and drowsy and an ambulance was called. On arrival in the Emergency Department, the patient suffered a witnessed tonic-clonic seizure, self-terminating after two minutes.

The patient had known hypertension and hypercholesterolaemia and had suffered a non-ST elevation myocardial infarction two years before, treated with a drug-eluting stent to the left anterior descending artery. Regular medications included ramipril 10 mg OD, bisoprolol 10 mg OD, bendroflumethiazide 2.5 mg OD, simvastatin 40 mg OD and aspirin 75 mg OD. The patient's wife reported that compliance with anti-hypertensive medications had been inconsistent and that controlling the patient's blood pressure had been an on-going problem over the previous two years. The patient was a retired builder and ex-smoker.

On examination, the patient was drowsy (GCS M4V2E3) but was protecting his own airway. Pupils were equal and reactive. The patient was spontaneously moving all his limbs and had downgoing plantar reflexes. Cardiovascular, respiratory and abdominal examination was unremarkable. Initial observations and investigations are listed below.

Blood pressure 220 / 115 mmHg
Heart rate 89 beat / minute
O2 sats (15 L O2) 100 %
Respiratory rate 19 / minute
Temperature 37.1°C.

CT brain: no extra-axial bleeding or collection; no intracerebral haemorrhage; no evidence acute ischaemic stroke; no subarachnoid blood; normal ventricular system; mild small vessel disease in keeping with patient's age.

Lumbar puncture:

CSF red cells	3/ mm^3
CSF white cells	3 / mm^3
CSF gram stain	unremarkable
CSF glucose	60 % serum level
protein	0.5 g / L
CSF	negative for bilirubin and xanthochromia

Given persistent reduced GCS and need to obtain blood pressure control, patient was intubated and transferred to the neuro intensive care unit. Following discussion with neurological team further imaging was arranged

MR brain with angiography: structurally normal brain as per previous study; bilateral symmetric vasogenic oedema involving the subcortical white matter in the parietal-occipital, posterior temporal and posterior frontal lobes; MRA unremarkable without any area of stenosis or vasospasm.

What is the correct diagnosis?

- ○ Reversible cerebral vasoconstriction syndrome ×
- ○ Posterior reversible leucoencephalopathy syndrome ×
- ○ Acute disseminated encephalomyelitis ×
- ○ Cerebral venous thrombosis ×
- ○ Pituitary apoplexy ×

Submit answer

Reference ranges

Score: **16%**

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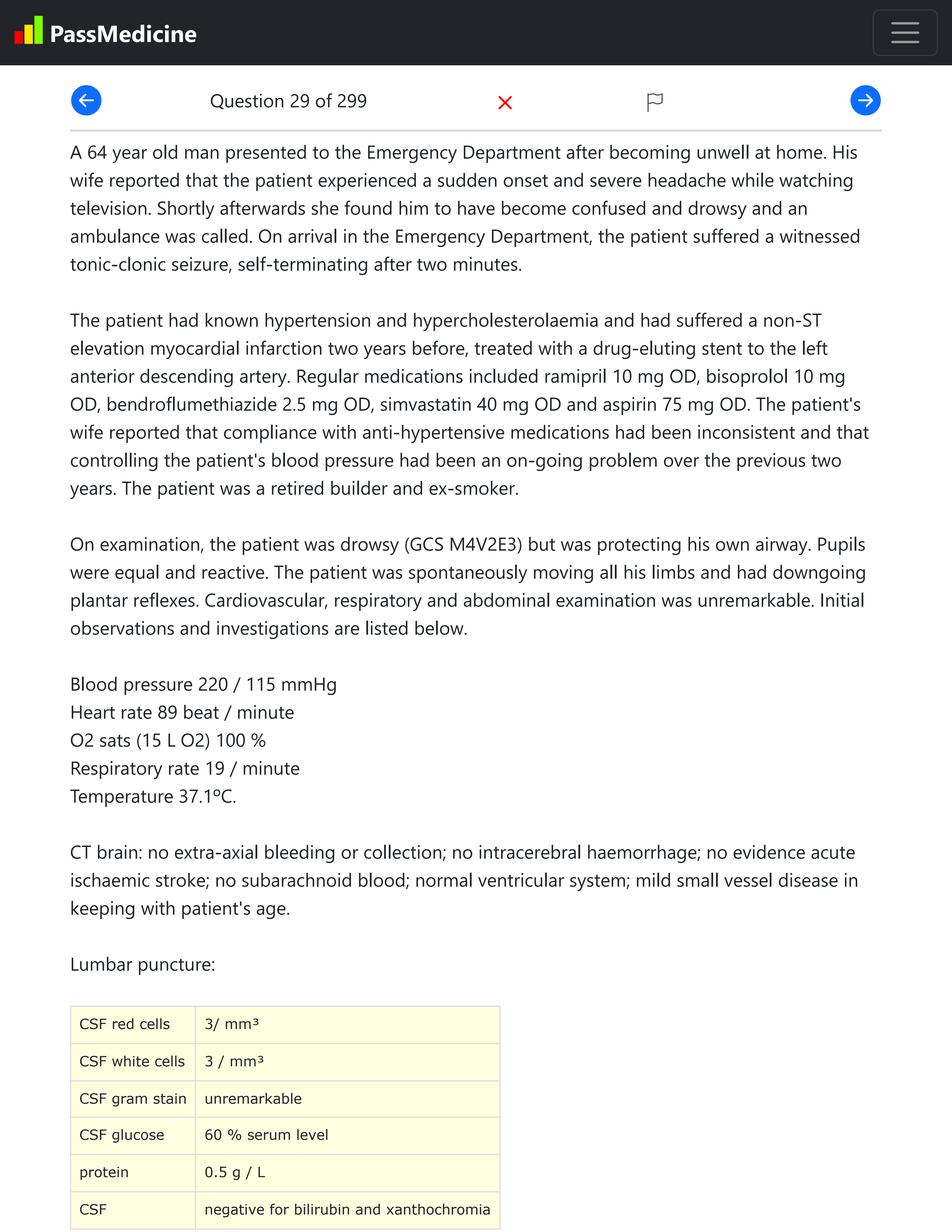

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What is the correct diagnosis?

Reversible cerebral vasoconstriction syndrome	32%
Posterior reversible leucoencephalopathy syndrome	49%
Acute disseminated encephalomyelitis	11%
Cerebral venous thrombosis	6%
Pituitary apoplexy	3%

Posterior reversible leucoencephalopathy syndrome may present with thunderclap headache, usually followed rapidly by confusion, seizures and visual symptoms. CT brain and lumbar puncture results are usually normal or near normal. The most common causes of PRES are hypertensive encephalopathy (as in this case) and eclampsia. Hypertension is commonly observed. Diagnosis is made by evidence of vasogenic brain oedema on MRI brain. PRES is often associated with reversible cerebrovascular vasoconstriction syndrome with vasospasms on angiography (although not in this case). Acute disseminated encephalomyelitis does not present with thunderclap headache.

Cerebral sinus thrombosis and pituitary apoplexy can both present with thunderclap headache and normal CT brain and LP although the other results and clinical picture in this case are not consistent with these diagnoses. Acute disseminated encephalomyelitis does not present with thunderclap headache.

Ducros A, Bousser M. Thunderclap headache. BMJ 2012;345:e8557.

Discuss (14) Improve

Next question ›

Thunderclap headache

Thunderclap headache describes a sudden (reaches maximum severity within seconds to minutes of onset) and severe headache.

Causes

- subarachnoid hemorrhage
- cerebral venous sinus thrombosis
- internal carotid artery dissection
- pituitary apoplexy
- reversible cerebral vasoconstriction syndrome
- primary sexual headache
- posterior reversible leucoencephalopathy syndrome
- acute hypertensive crisis

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Score: **17.6%**

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Back to top

A 61-year-old man is referred neurology after he noticed a tremor. He has a tremor affecting both his arms and his wife has noticed he is slow with activities and has struggled with his balance more recently. Further questioning revealed the patient has had some issues with urinary incontinence and erectile dysfunction.

His past medical history includes hypertension, myocardial infarction and hypercholesterolaemia. He takes ramipril, bisoprolol, aspirin and simvastatin.

On examination, he is found to increased tone, but power was normal. However, his coordination was impaired. The rest of the physical examination was normal. His GP did some blood tests prior to the referral, the results of which are shown below:

Thyroid stimulating hormone (TSH)	0.4 mU/L	(0.5-5.5)
Free thyroxine (T4)	12 pmol/L	(9.0 - 18)
Hb	125 g/L	(135-180)
WBC	5.7 * 10^9/L	(4.0 - 11.0)
Na$^+$	139 mmol/L	(135 - 145)
K$^+$	4.3 mmol/L	(3.5 - 5.0)
Urea	6.5 mmol/L	(2.0 - 7.0)
Creatinine	148 µmol/L	(55 - 120)

What is the most likely cause of this man's presentation?

○ Idiopathic parkinson's disease

○ Multiple system atrophy

○ Progressive supranuclear palsy

○ Drug induced parkinsonism

○ Hyperthyroidism

Submit answer

Reference ranges

Score: **16%**

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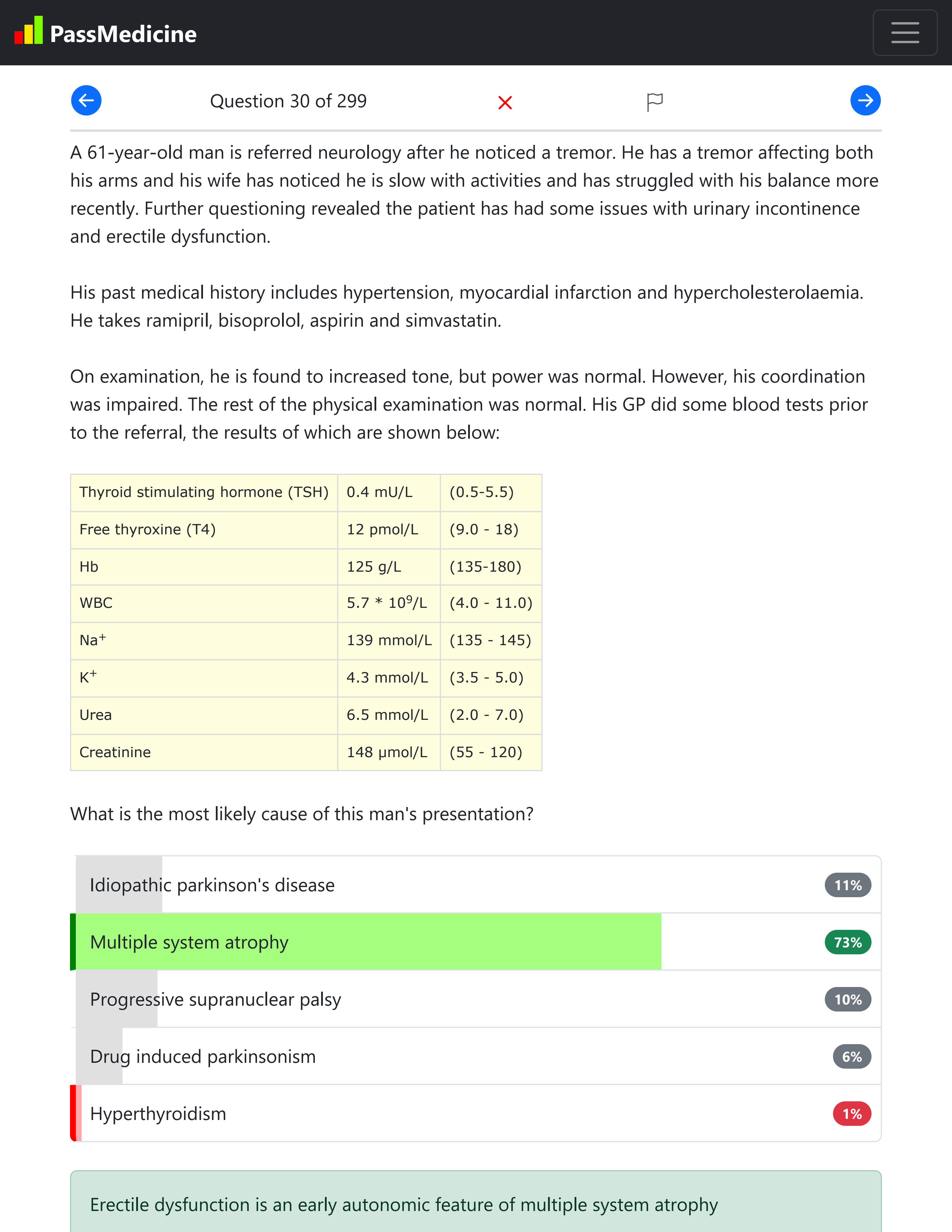

Question 30 of 299

A 61-year-old man is referred neurology after he noticed a tremor. He has a tremor affecting both his arms and his wife has noticed he is slow with activities and has struggled with his balance more recently. Further questioning revealed the patient has had some issues with urinary incontinence and erectile dysfunction.

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Na^+	139 mmol/L	(135 - 145)
K^+	4.3 mmol/L	(3.5 - 5.0)
Urea	6.5 mmol/L	(2.0 - 7.0)
Creatinine	148 μmol/L	(55 - 120)

What is the most likely cause of this man's presentation?

Idiopathic parkinson's disease	11%
Multiple system atrophy	73%
Progressive supranuclear palsy	10%
Drug induced parkinsonism	6%
Hyperthyroidism	1%

Erectile dysfunction is an early autonomic feature of multiple system atrophy

Important for me Less important

This patient has presented with Parkinsonism (tremor, bradykinesia and rigidity). There are lots of causes of Parkinsonism.

In this case, the clues to working out the diagnosis are the balance issues, coordination problems, urinary incontinence and in particular the erectile dysfunction. This points to a diagnosis of multiple system atrophy.

In Parkinson's disease, the onset of tremor is usually unilateral at the beginning. It is typically described as being present at rest and disappears on movement. Other features include micrographia.

Progressive supranuclear palsy is a Parkinson's plus syndrome. Differentiating features are difficulty with moving the eyes vertically, slurring of speech and difficulty swallowing.

Drug-induced Parkinsonism is usually seen with antipsychotics and is reversible upon cessation of the causative drug. None of the medications listed cause drug-induced Parkinsonism.

Tremor can be a feature of hyperthyroidism. However, there are no other features in the history to support this diagnosis such as weight loss despite increased appetite, heat intolerance, sweating, diarrhoea etc.

Discuss (2) Improve

Next question

Multiple system atrophy

There are 2 predominant types of multiple system atrophy
- 1) MSA-P - Predominant Parkinsonian features
- 2) MSA-C - Predominant Cerebellar features

Shy-Drager syndrome is a type of multiple system atrophy.

Features
- parkinsonism
- autonomic disturbance
  - erectile dysfunction: often an early feature
  - postural hypotension

    - atonic bladder
- cerebellar signs

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **17.6%**

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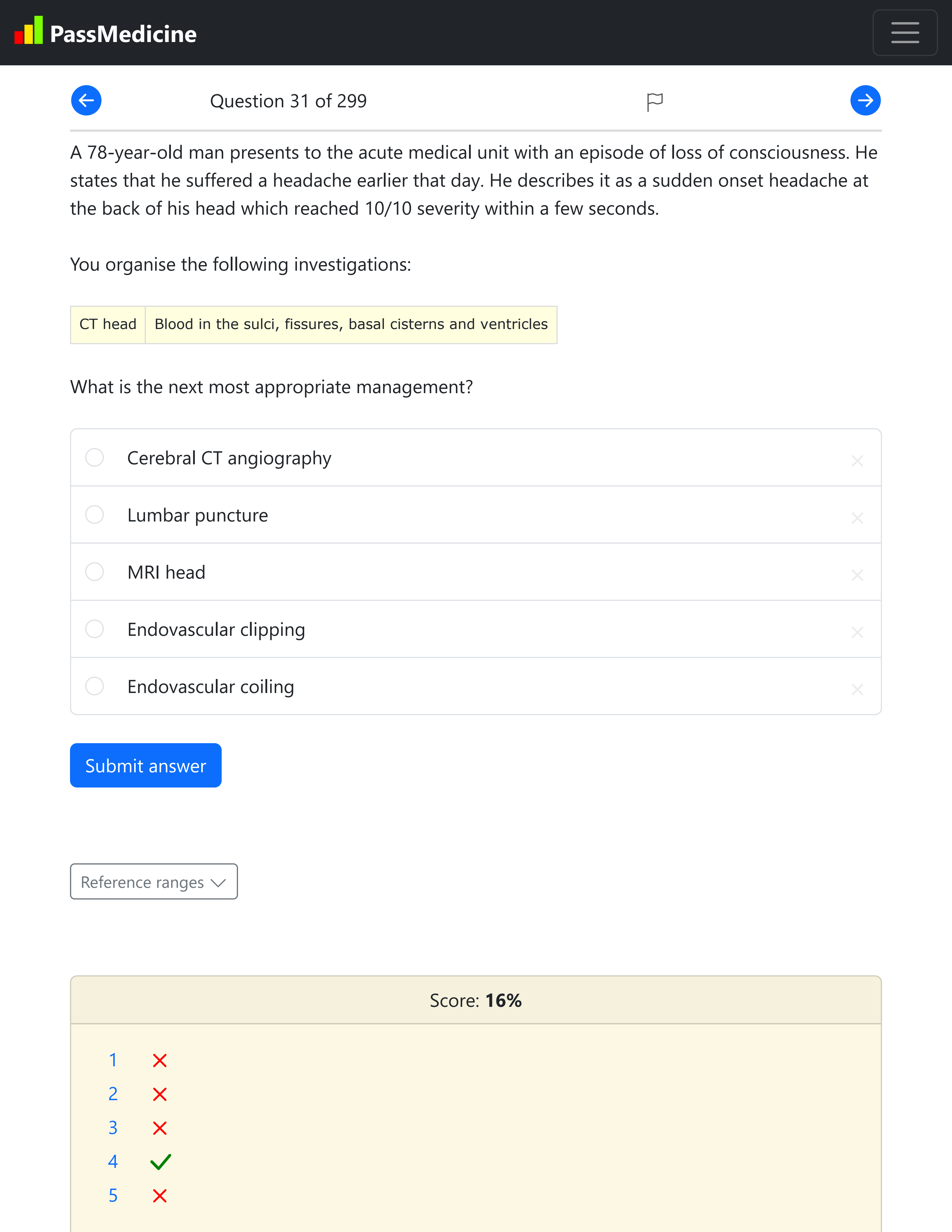

Question 31 of 299

A 78-year-old man presents to the acute medical unit with an episode of loss of consciousness. He states that he suffered a headache earlier that day. He describes it as a sudden onset headache at the back of his head which reached 10/10 severity within a few seconds.

You organise the following investigations:

CT head	Blood in the sulci, fissures, basal cisterns and ventricles

What is the next most appropriate management?

○ Cerebral CT angiography ×

○ Lumbar puncture ×

○ MRI head ×

○ Endovascular clipping ×

○ Endovascular coiling ×

Score: **16%**

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A 78-year-old man presents to the acute medical unit with an episode of loss of consciousness. He states that he suffered a headache earlier that day. He describes it as a sudden onset headache at the back of his head which reached 10/10 severity within a few seconds.

You organise the following investigations:

CT head	Blood in the sulci, fissures, basal cisterns and ventricles

What is the next most appropriate management?

Cerebral CT angiography	59%
Lumbar puncture	3%
MRI head	2%
Endovascular clipping	9%
Endovascular coiling	26%

In non-traumatic subarachnoid haemorrhages, a CT angiogram should be performed to identify intracranial aneurysms

Important for me Less important

The CT scan has confirmed a subarachnoid haemorrhage (SAH). As it is spontaneous, and not traumatic, it is likely to be an aneurysmal SAH. However to confirm this the patient needs urgent angiography - either CT or MRI. Once an aneurysm is confirmed radiologically the patient can undergo endovascular clipping or coiling.

A lumbar puncture is not indicated in this case as the CT has confirmed the diagnosis of SAH. A lumbar puncture should be performed 12 hours after the onset of headache in patients who have a negative CT scan.

MRI head would confirm a haemorrhage, however angiography is required to definitively confirm an aneurysm.

👍 👎 💬 Discuss (5) Improve

Next question >

Subarachnoid haemorrhage ★

A subarachnoid haemorrhage (SAH) is an intracranial haemorrhage that is defined as the presence of blood within the subarachnoid space, i.e. deep to the subarachnoid layer of the meninges.

The most common cause of SAH is head injury and this is called *traumatic SAH* . In the absence of trauma, SAH is termed *spontaneous SAH* . The rest of this note focuses on spontaneous SAH.

Causes of spontaneous SAH include:

- intracranial aneurysm (saccular 'berry' aneurysms)
    - accounts for around 85% of cases
    - conditions associated with berry aneurysms include hypertension,adult polycystic kidney disease, Ehlers-Danlos syndrome and coarctation of the aorta
- arteriovenous malformation
- pituitary apoplexy
- mycotic (infective) aneurysms

Classical presenting features include:

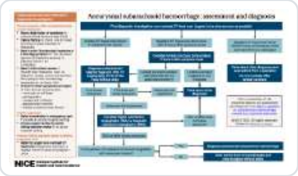
- headache
    - usually of sudden-onset ('thunderclap' or 'hit with a baseball bat')
    - severe ('worst of my life')
    - occipital
    - typically peaking in intensity within 1 to 5 minutes
    - there may be a history of a less-severe 'sentinel' headache in the weeks prior to presentation
- nausea and vomiting
- meningism (photophobia, neck stiffness)
- coma
- seizures
- ECG changes including ST elevation may be seen
    - this may be secondary to either autonomic neural stimulation from the hypothalamus or elevated levels of circulating catecholamines

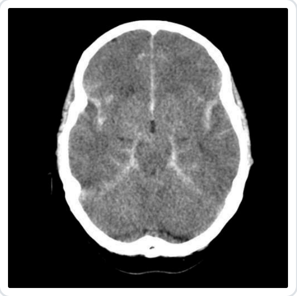
Investigation

- non-contrast CT head is the first-line investigation of choice
    - acute blood (hyperdense/bright on CT) is typically distributed in the basal cisterns, sulci and in severe cases the ventricular system.
- if CT head is done within 6 hours of symptom onset and is normal
    - new guidelines suggest **not** doing a lumbar puncture
    - consider an alternative diagnosis
- if CT head is done more than 6 hours after symptom onset and is normal
    - do a lumber puncture (LP)
    - timing wise the LP should be performed at least 12 hours following the onset of symptoms to allow the development of xanthochromia (the result of red blood cell breakdown).
    - xanthochromia helps to distinguish true SAH from a 'traumatic tap' (blood introduced by the LP procedure).
    - as well as xanthochromia, CSF findings consistent with subarachnoid haemorrhage include a normal or raised opening pressure
- if the CT shows evidence of a SAH
    - referral to neurosurgery to be made as soon as SAH is confirmed

After spontaneous SAH is confirmed, the aim of investigation is to identify a causative pathology that needs urgent treatment:

- CT intracranial angiogram (to identify a vascular lesion e.g. aneurysm or AVM)
- +/- digital subtraction angiogram (catheter angiogram)


Aneurysmal subarachnoid haemorrhage: assessment and diagnosis
NICE



Management of a confirmed aneurysmal subarachnoid haemorrhage

- supportive
    - bed rest
    - analgesia
    - venous thromboembolism prophylaxis
    - discontinuation of antithrombotics (reversal of anticoagulation if present)
- vasospasm is prevented using a course of oral nimodipine
- intracranial aneurysms are at risk of rebleeding and therefore require prompt intervention, preferably within 24 hours

  - most intracranial aneurysms are now treated with a coil by interventional neuroradiologists, but a minority require a craniotomy and clipping by a neurosurgeon

Complications of aneurysmal SAH:

- re-bleeding
  - happens in around 10% of cases and most common in the first 12 hours
  - if rebleeding is suspected (e.g. sudden worsening of neurological symptoms) then a repeat CT should be arranged
  - associated with a high mortality (up to 70%)
- hydrocephalus
  - hydrocephalus is temporarily treated with an external ventricular drain (CSF diverted into a bag at the bedside) or, if required, a long-term ventriculoperitoneal shunt
- vasospasm (also termed delayed cerebral ischaemia), typically 7-14 days after onset
  - ensure euvolaemia (normal blood volume)
  - consider treatment with a vasopressor if symptoms persist
- hyponatraemia (most typically due to syndrome inappropriate anti-diuretic hormone (SIADH))
- seizures

Important predictive factors in SAH:

- conscious level on admission
- age
- amount of blood visible on CT head

+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia 👍 15 👎 15

Subarachnoid haemorrhage

Life in the Fast Lane 👍 19 👎 10

Example ECG changes in subarachnoid haemorrhage

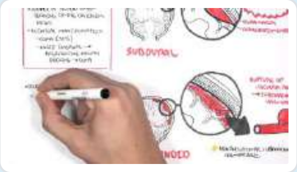
NICE 👍 9 👎 4

2022 Subarachnoid haemorrhage caused by a ruptured aneurysm: diagnosis and management

Suggest link

Report broken link

Media



Intracranial Haemorrhage Types

Armando Hasudungan - YouTube 👍 2 👎 0

Arteriovenous malformation

Osmosis - YouTube 👍 0 👎 0

NEUROLOGY
NEUROSURGERY
SUBARACHNOID HAEMORRHAGE

Subarachnoid Haemorrhage / pathophysiology, complications and management

Armando Hasudungan - YouTube 👍 0 👎 0

Report broken media

Score: **17.6%**

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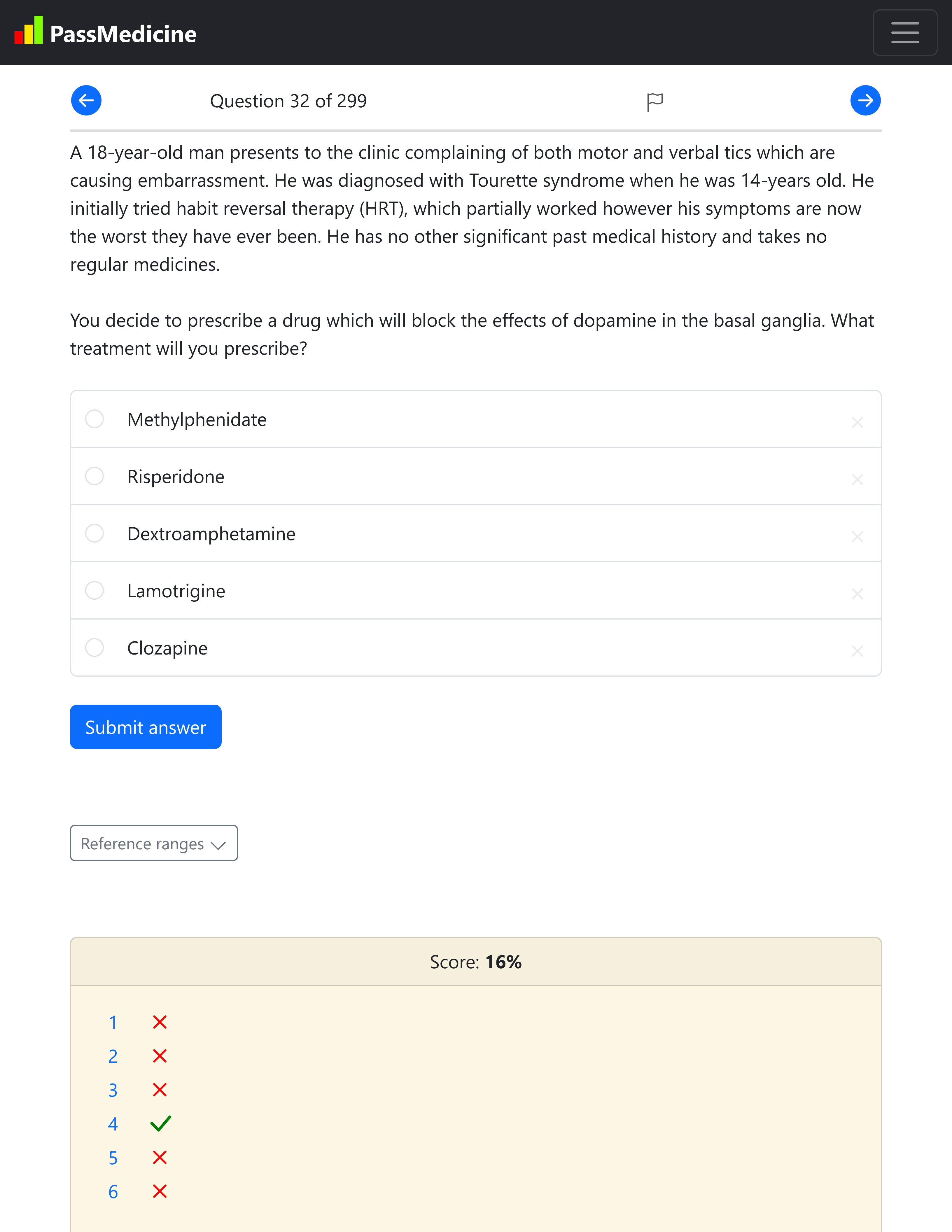

Question 32 of 299

A 18-year-old man presents to the clinic complaining of both motor and verbal tics which are causing embarrassment. He was diagnosed with Tourette syndrome when he was 14-years old. He initially tried habit reversal therapy (HRT), which partially worked however his symptoms are now the worst they have ever been. He has no other significant past medical history and takes no regular medicines.

You decide to prescribe a drug which will block the effects of dopamine in the basal ganglia. What treatment will you prescribe?

- ○ Methylphenidate
- ○ Risperidone
- ○ Dextroamphetamine
- ○ Lamotrigine
- ○ Clozapine

Score: **16%**

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You decide to prescribe a drug which will block the effects of dopamine in the basal ganglia. What treatment will you prescribe?

Methylphenidate	17%
Risperidone	57%
Dextroamphetamine	9%
Lamotrigine	3%
Clozapine	13%

Antipsychotics have the greatest efficacy at treating tics

Important for me Less important

Tics are classed as a hyperkinetic movement disorder consisting of repetitive, stereotyped motor or vocal movements that may be simple or complex. There is an urge to perform the movement with relief after performing the movement. They are caused by enhanced dopamine levels. Therefore dopamine antagonists such as the antipsychotics are first line pharmacological agents. Both risperidone and clozapine are antipsychotics, however due to the potentially life threatening side effects of clozapine (e.g. myocarditis and neutropaenia) it is reserved as a third line agent.

Methylphenidate, dextroamphetamine, and, lamotrigine are known drug induced causes of tics.

Discuss (4) Improve

Next question

Tics ★

Tics may be defined as intermittent, stereotypical, repetitive, involuntary movements. Around 15% of primary school age children have tics such as blinking and shrugging

Treatment options include
- clonidine
- atypical antipsychotics

+ Q 123

Next question ›

B *I*

Textbooks

High-yield textbook

Extended textbook

Score: **17.6%**

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Question 33 of 299

A 24-year-old man presents with lower limb weakness. He states that he had an episode of food poisoning two weeks ago which consisted of severe abdominal pain and profuse non-bloody diarrhoea. Following this he noticed progressive weakness in his legs. His past medical history is largely unremarkable, however, he does admit to intravenous drug use with heroin. He last injected heroin three weeks ago.

On examination, you note weakness of ankle plantar flexion (MRC grade 3), ankle dorsiflexion (MRC grade 3), knee flexion (MRC grade 4) and knee extension (MRC grade 4). The ankle jerk and knee jerk are only present on reinforcement. There is a loss of sensation to the mid-calves. A neurological examination was otherwise unremarkable.

What is the most likely diagnosis?

- ○ Acute inflammatory demyelinating polyradiculoneuropathy (AIDP)
- ○ Acute motor axonal neuropathy (AMAN)
- ○ Botulism
- ○ Miller Fisher syndrome
- ○ Tetanus

Submit answer

Reference ranges

Score: **16%**

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On examination, you note weakness of ankle plantar flexion (MRC grade 3), ankle dorsiflexion (MRC grade 3), knee flexion (MRC grade 4) and knee extension (MRC grade 4). The ankle jerk and knee jerk are only present on reinforcement. There is a loss of sensation to the mid-calves. A neurological examination was otherwise unremarkable.

What is the most likely diagnosis?

Acute inflammatory demyelinating polyradiculoneuropathy (AIDP)	69%
Acute motor axonal neuropathy (AMAN)	9%
Botulism	14%
Miller Fisher syndrome	7%
Tetanus	1%

Worsening lower limb weakness following gastroenteritis - think Guillain-Barre

Important for me Less important

Acute inflammatory demyelinating polyradiculoneuropathy (AIDP) is correct. The most likely diagnosis is Guillain-Barre syndrome. The clinical features are most in keeping with AIDP which is the most common subtype. The preceding gastroenteritis is classic of this condition with approximately 30% of cases being provoked by *Campylobacter jejuni* infection.

Acute motor axonal neuropathy (AMAN) is incorrect. This is a less common subtype of Guillain-Barre syndrome which presents with motor involvement only. There is often early and severe respiratory involvement. The presence of sensory involvement, in this case, makes this a less likely diagnosis.

Botulism is incorrect. Botulism is characterised by a descending, flaccid paralysis, in contrast to Guillain-Barre syndrome which presents with an ascending paralysis. It is important to note that the patient is an intravenous drug user which is a risk factor for botulism. However, this method of transmission would not explain the gastroenteritis features. These can of course be related to food-borne botulism, however regardless the clinical features of an ascending paralysis are more in keeping with Guillain-Barre syndrome.

Miller Fisher syndrome is incorrect. This subtype of Guillain-Barre syndrome is characterised by areflexia, ophthalmoplegia, and ataxia. Limb weakness in this subtype is uncommon making this a less likely diagnosis.

Tetanus is incorrect. Tetanus is a life-threatening disease characterised by a descending spastic paralysis. Although the causative bacteria *Clostridium tetani* has been found in contaminated heroin, the clinical features of an ascending paralysis are more suggestive of Guillain-Barre syndrome.

👍 👎 Discuss (3) Improve

Next question >

Guillain-Barre syndrome: features ★

Guillain-Barre syndrome describes an immune-mediated demyelination of the peripheral nervous system often triggered by an infection (classically *Campylobacter jejuni*).

Initial symptoms
- around 65% of patients experience back/leg pain in the initial stages of the illness

The characteristic features of Guillain-Barre syndrome is progressive, symmetrical weakness of all the limbs.
- the weakness is classically **ascending** i.e. the legs are affected first
- reflexes are reduced or absent
- sensory symptoms tend to be mild (e.g. distal paraesthesia) with very few sensory signs

Other features
- there may be a history of gastroenteritis
- respiratory muscle weakness
- cranial nerve involvement

    - diplopia
    - bilateral facial nerve palsy
    - oropharyngeal weakness is common
- autonomic involvement
    - urinary retention
    - diarrhoea

Less common findings

- papilloedema: thought to be secondary to reduced CSF resorption

Investigations

- lumbar puncture
    - rise in protein with a normal white blood cell count (albuminocytologic dissociation) - found in 66%
- nerve conduction studies may be performed
    - decreased motor nerve conduction velocity (due to demyelination)
    - prolonged distal motor latency
    - increased F wave latency

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Medcomic 10 8

Guillain-Barre syndrome

Patient.info 👍 6 👎 5

Guillain-Barre syndrome review

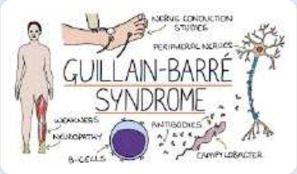
Suggest link Report broken link

Media

SYMPTOMS - BASED on NERVES AFFECTED
MUSCLE WEAKNESS & ABSENCE of REFLEXES

Guillain-Barre syndrome

Osmosis - YouTube 👍 1 👎 0


NERVE CONDUCTION STUDIES
PERIPHERAL NERVES
GUILLAIN-BARRÉ SYNDROME
WEAKNESS
NEUROPATHY
ANTIBODIES
B-CELLS
CAMPYLOBACTER

Understanding Guillain-Barre Syndrome

Zero To Finals - YouTube 👍 1 👎 0

Report broken media

Score: **17.6%**

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Question 34 of 299

A 23-year-old man is brought into the Emergency Department with a severe headache.

He was awoken from sleep early this morning with the most severe headache he has ever experienced. He describes an immediate onset posterior headache that has not improved at all.

He has felt his vision slightly blurred and vomited 3 times since his arrival in the department.

You perform an urgent CT brain which reveals a subarachnoid haemorrhage.

What investigation is now indicated?

○ CT angiogram

○ Digital subtraction angiography

○ Lumbar puncture

○ MRI brain

○ Ophthalmoscopy

Submit answer

Reference ranges

Score: **16%**

1 ✗
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He was awoken from sleep early this morning with the most severe headache he has ever experienced. He describes an immediate onset posterior headache that has not improved at all.

He has felt his vision slightly blurred and vomited 3 times since his arrival in the department.

You perform an urgent CT brain which reveals a subarachnoid haemorrhage.

What investigation is now indicated?

CT angiogram	80%
Digital subtraction angiography	11%
Lumbar puncture	5%
MRI brain	2%
Ophthalmoscopy	2%

In non-traumatic subarachnoid haemorrhages, a CT angiogram should be performed to identify intracranial aneurysms

Important for me   Less important

The next step when a subarachnoid haemorrhage is confirmed is to determine whether the haemorrhage is aneurysmal or not and so the correct answer is a **CT angiogram**.

Subarachnoid haemorrhage has been confirmed on plain CT and so a **lumbar puncture** to look for xanthochromia is not necessary and therefore incorrect. This would be the correct answer if CT had not revealed a cause for the headache and you were still suspicious of a subarachnoid haemorrhage.

MRI brain is incorrect. It will not necessarily show an aneurysm so may not be helpful and an angiogram would still need to be performed.

Digital subtraction angiography is incorrect. It is sometimes performed by neurosurgeons or interventional radiology to look for an aneurysm but CT angiography is quicker and safer to confirm this.

Ophthalmoscopy is also incorrect and has little value here as the diagnosis and raised intracranial pressure are already known.

👍 👎 Discuss Improve

Next question >

Subarachnoid haemorrhage

A subarachnoid haemorrhage (SAH) is an intracranial haemorrhage that is defined as the presence of blood within the subarachnoid space, i.e. deep to the subarachnoid layer of the meninges.

The most common cause of SAH is head injury and this is called *traumatic SAH* . In the absence of trauma, SAH is termed *spontaneous SAH* . The rest of this note focuses on spontaneous SAH.

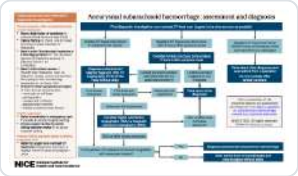
Causes of spontaneous SAH include:
- intracranial aneurysm (saccular 'berry' aneurysms)
    - accounts for around 85% of cases
    - conditions associated with berry aneurysms include hypertension,adult polycystic kidney disease, Ehlers-Danlos syndrome and coarctation of the aorta
- arteriovenous malformation
- pituitary apoplexy
- mycotic (infective) aneurysms

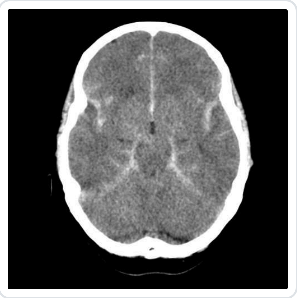
Classical presenting features include:
- headache
    - usually of sudden-onset ('thunderclap' or 'hit with a baseball bat')
    - severe ('worst of my life')
    - occipital
    - typically peaking in intensity within 1 to 5 minutes
    - there may be a history of a less-severe 'sentinel' headache in the weeks prior to presentation
- nausea and vomiting
- meningism (photophobia, neck stiffness)

- coma
- seizures
- ECG changes including ST elevation may be seen
  - this may be secondary to either autonomic neural stimulation from the hypothalamus or elevated levels of circulating catecholamines

Investigation
- non-contrast CT head is the first-line investigation of choice
  - acute blood (hyperdense/bright on CT) is typically distributed in the basal cisterns, sulci and in severe cases the ventricular system.
- if CT head is done within 6 hours of symptom onset and is normal
  - new guidelines suggest **not** doing a lumbar puncture
  - consider an alternative diagnosis
- if CT head is done more than 6 hours after symptom onset and is normal
  - do a lumber puncture (LP)
  - timing wise the LP should be performed at least 12 hours following the onset of symptoms to allow the development of xanthochromia (the result of red blood cell breakdown).
  - xanthochromia helps to distinguish true SAH from a 'traumatic tap' (blood introduced by the LP procedure).
  - as well as xanthochromia, CSF findings consistent with subarachnoid haemorrhage include a normal or raised opening pressure
- if the CT shows evidence of a SAH
  - referral to neurosurgery to be made as soon as SAH is confirmed

After spontaneous SAH is confirmed, the aim of investigation is to identify a causative pathology that needs urgent treatment:
- CT intracranial angiogram (to identify a vascular lesion e.g. aneurysm or AVM)
- +/- digital subtraction angiogram (catheter angiogram)





Management of a confirmed aneurysmal subarachnoid haemorrhage
- supportive
  - bed rest
  - analgesia

  - venous thromboembolism prophylaxis
  - discontinuation of antithrombotics (reversal of anticoagulation if present)
- vasospasm is prevented using a course of oral nimodipine
- intracranial aneurysms are at risk of rebleeding and therefore require prompt intervention, preferably within 24 hours
  - most intracranial aneurysms are now treated with a coil by interventional neuroradiologists, but a minority require a craniotomy and clipping by a neurosurgeon

Complications of aneurysmal SAH:

- re-bleeding
  - happens in around 10% of cases and most common in the first 12 hours
  - if rebleeding is suspected (e.g. sudden worsening of neurological symptoms) then a repeat CT should be arranged
  - associated with a high mortality (up to 70%)
- hydrocephalus
  - hydrocephalus is temporarily treated with an external ventricular drain (CSF diverted into a bag at the bedside) or, if required, a long-term ventriculoperitoneal shunt
- vasospasm (also termed delayed cerebral ischaemia), typically 7-14 days after onset
  - ensure euvolaemia (normal blood volume)
  - consider treatment with a vasopressor if symptoms persist
- hyponatraemia (most typically due to syndrome inappropriate anti-diuretic hormone (SIADH))
- seizures

Important predictive factors in SAH:

- conscious level on admission
- age
- amount of blood visible on CT head

+ Q 123

Next question >

Textbooks

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Extended textbook

Links

Radiopaedia 👍 15 👎 15

Subarachnoid haemorrhage

Life in the Fast Lane 👍 19 👎 10

Example ECG changes in subarachnoid haemorrhage

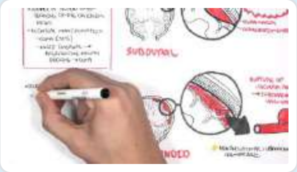
NICE 👍 9 👎 4

2022 Subarachnoid haemorrhage caused by a ruptured aneurysm: diagnosis and management

Suggest link

Report broken link

Media



Intracranial Haemorrhage Types

Armando Hasudungan - YouTube 👍 2 👎 0

Arteriovenous malformation

Osmosis - YouTube 👍 0 👎 0

NEUROLOGY
NEUROSURGERY
SUBARACHNOID HAEMORRHAGE

Subarachnoid Haemorrhage / pathophysiology, complications and management

Armando Hasudungan - YouTube 👍 0 👎 0

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Score: **17.6%**

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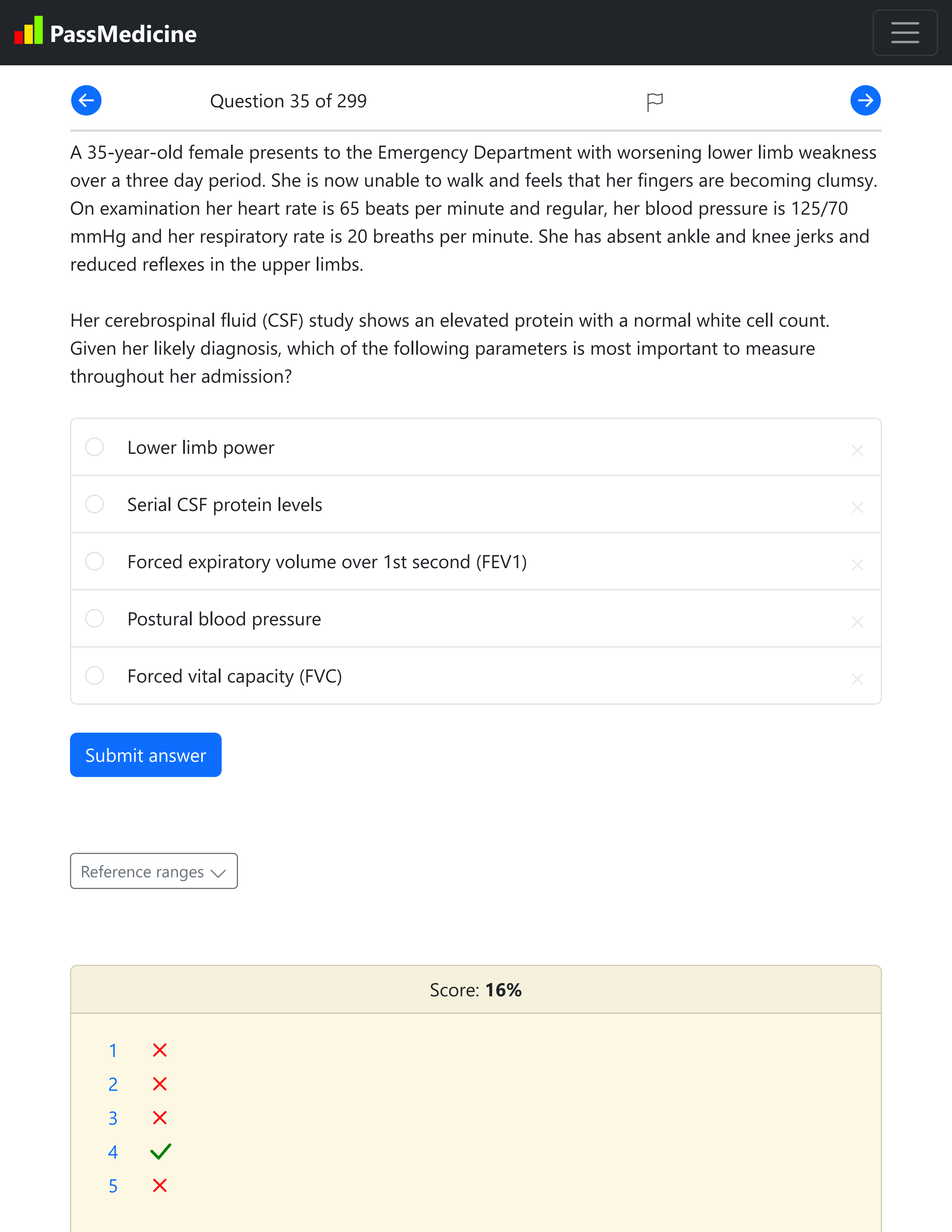

Question 35 of 299

A 35-year-old female presents to the Emergency Department with worsening lower limb weakness over a three day period. She is now unable to walk and feels that her fingers are becoming clumsy. On examination her heart rate is 65 beats per minute and regular, her blood pressure is 125/70 mmHg and her respiratory rate is 20 breaths per minute. She has absent ankle and knee jerks and reduced reflexes in the upper limbs.

Her cerebrospinal fluid (CSF) study shows an elevated protein with a normal white cell count. Given her likely diagnosis, which of the following parameters is most important to measure throughout her admission?

○ Lower limb power

○ Serial CSF protein levels

○ Forced expiratory volume over 1st second (FEV1)

○ Postural blood pressure

○ Forced vital capacity (FVC)

Score: **16%**

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Her cerebrospinal fluid (CSF) study shows an elevated protein with a normal white cell count. Given her likely diagnosis, which of the following parameters is most important to measure throughout her admission?

Lower limb power	1%
Serial CSF protein levels	1%
Forced expiratory volume over 1st second (FEV1)	10%
Postural blood pressure	1%
Forced vital capacity (FVC)	88%

FVC is used to monitor respiratory function in Guillain-Barre syndrome

Important for me Less important

The most common cause of death in patients with the severe Guillain-Barre Syndrome (GBS) is respiratory failure. This is due primarily to weakness in chest wall muscles, but may also be due to bulbar dysfunction and aspiration. Patients may require intensive care admission and ventilatory support.

For this reason, it is of great importance that forced vital capacity (FVC) is measured on a routine basis and results documented so that additional support can be sought as soon as possible. FEV1 may be preserved in early deterioration as it is not a measure of vital capacity or a marker of chest wall expansion. Therefore, a reduction in FVC is a more sensitive marker of deterioration.

👍 👎 Discuss (2) Improve

Next question ›

Guillain-Barre syndrome: management ★

Guillain-Barre syndrome describes an immune-mediated demyelination of the peripheral nervous system often triggered by an infection (classically *Campylobacter jejuni*).

Management

- IV immunoglobulins (IVIG) or plasma exchange can be used
    - IVIG is as effective as plasma exchange better tolerated and easier to administer
    - therefore IVIG is generally used first-line for Guillain-Barre syndrome where patients require immunomodulatory therapy
    - no benefit in combining both treatments
- steroids and immunosuppressants have not been shown to be beneficial
- FVC regularly to monitor respiratory function

Prognosis

- severe motor problems persist in about 15%
- around 5% die

+ Q 123

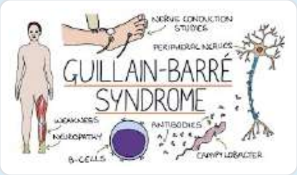
Next question ›

Textbooks

High-yield textbook

Extended textbook

Media


NERVE CONDUCTION STUDIES
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Understanding Guillain-Barre Syndrome

Zero To Finals - YouTube 👍 3 👎 1

SYMPTOMS - BASED on NERVES AFFECTED

Guillain-Barre syndrome

Osmosis - YouTube 👍 1 👎 1

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A 17-year-old man is referred by the emergency department with weakness of his lower limbs and hands, worsening over the last 2 days. He is having increasing difficulty walking and is becoming very clumsy and dropping things. He burnt his hand yesterday in the kitchen without realising until afterwards that he had done it. He also complains of feeling dizzy on standing but has no chest pain or palpitations. He is normally fit and well, although did suffer a bought of diarrhoea and vomiting 4 weeks ago. He is on no regular medication and denies any alcohol or smoking history.

On examination he is afebrile, his blood pressure is 128/79 mmHg on lying but on standing it drops to 90/60 mmHg. His respiratory rate is 20/min and his saturations are 97% on air. His cardiovascular, respiratory and abdominal examinations are normal. Neurological examination reveals symmetrical distal weakness of his lower limbs with relative proximal sparing and altered sensation to fine touch, vibration and proprioception. He has similar findings in his upper limbs. He is suspected of having Guillain-Barre syndrome and following blood tests, CT head and lumbar puncture is started on IV immunoglobulins. Despite this, he begins to become increasingly short of breath and he is referred to ITU for respiratory support.

What respiratory parameter is used to determine if invasive ventilator support is needed?

○ pO2 on ABG < 8kPa ×

○ FEV1:FVC ratio < 0.7 ×

○ Saturations on 15 litres O2 less than 93% ×

○ FVC < 15ml/kg ×

○ Peak flow of less than 300ml ×

Submit answer

Reference ranges

Score: **16%**

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36 -

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What respiratory parameter is used to determine if invasive ventilator support is needed?

pO2 on ABG < 8kPa	6%
FEV1:FVC ratio < 0.7	5%
Saturations on 15 litres O2 less than 93%	3%
FVC < 15ml/kg	80%
Peak flow of less than 300ml	6%

FVC is used to monitor respiratory function in Guillain-Barre syndrome

Important for me Less important

This man has Guillain-Barre syndrome with autonomic involvement and compromise to his respiratory function. The parameter used to assess whether a patient needs ventilator support is an FVC <15-20ml/kg. low pO2, low saturations and low peak flow are all indicatory of poor respiratory function but FVC is the parameter used in these cases. FEV1:FVC ratio is not used.

Discuss (7) | Improve

Next question ›

Guillain-Barre syndrome: management ★

Guillain-Barre syndrome describes an immune-mediated demyelination of the peripheral nervous system often triggered by an infection (classically *Campylobacter jejuni*).

Management
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Prognosis
- severe motor problems persist in about 15%
- around 5% die

AI + Q 123

Next question ›

B *I*

Textbooks

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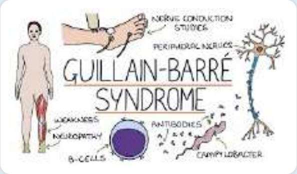
Media


GUILLAIN-BARRÉ
SYNDROME
NERVE CONDUCTION STUDIES
PERIPHERAL NERVES
WEAKNESS
NEUROPATHY
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B-CELLS
CAMPYLOBACTER

Understanding Guillain-Barre Syndrome

Zero To Finals - YouTube 👍 3 👎 1

Guillain-Barre syndrome

Osmosis - YouTube 👍 1 👎 1

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Score: **17.6%**

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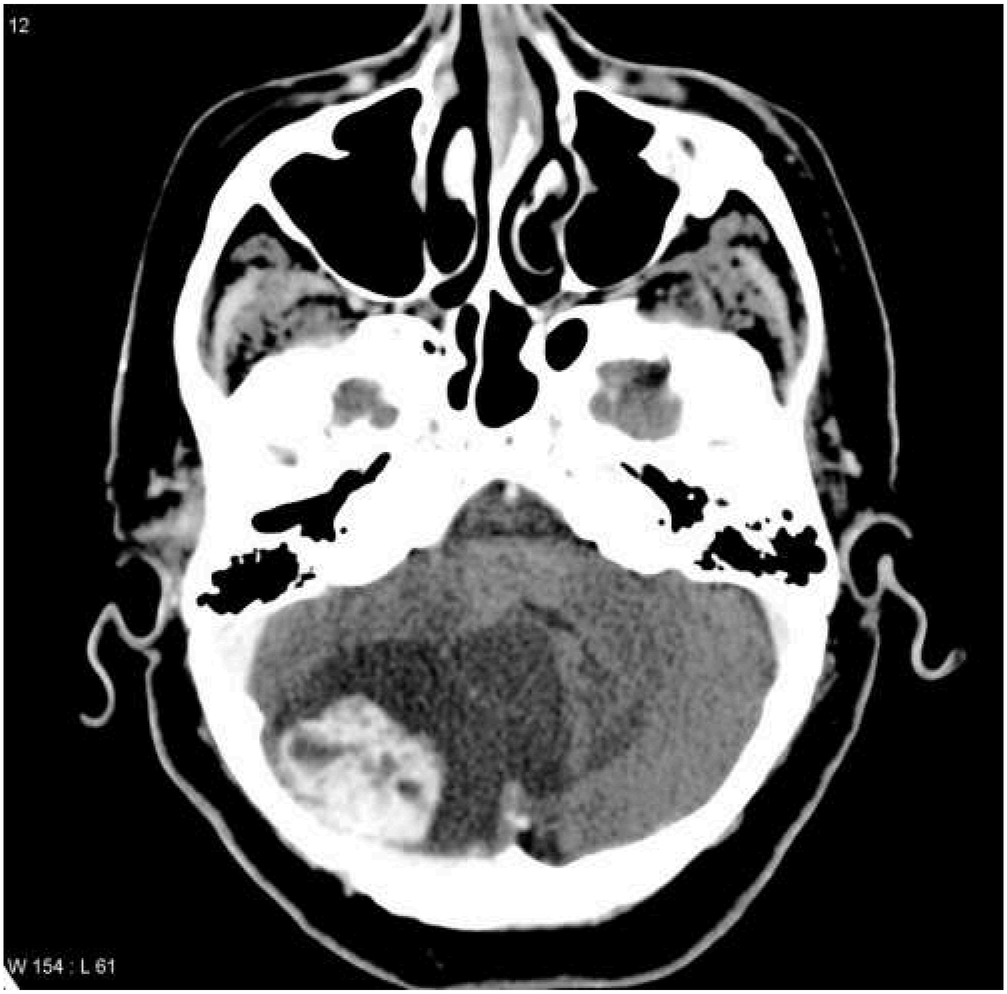
A 26-year-old man is admitted for investigation after having increasing trouble walking. On admission he is found to have a severely ataxic gait and is unable to walk in a straight-line. For many weeks he has been complaining of visual problems, dizziness, headaches and clumsiness.

On examination he is noted to have horizontal nystagmus, an intention tremor and past pointing. On fundoscopy a large, erythematous retinal lesion is present surrounded by tortuous, dilated vessels.

His father had similar problems in his 20's.

A CT scan is arranged:



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What is the most likely underlying diagnosis?

○ Friedreich's ataxia

○ Ataxic telangiectasia

○ Neurofibromatosis

○ Tuberous sclerosis

○ Von Hippel-Lindau syndrome

Submit answer

Reference ranges

Score: **16%**

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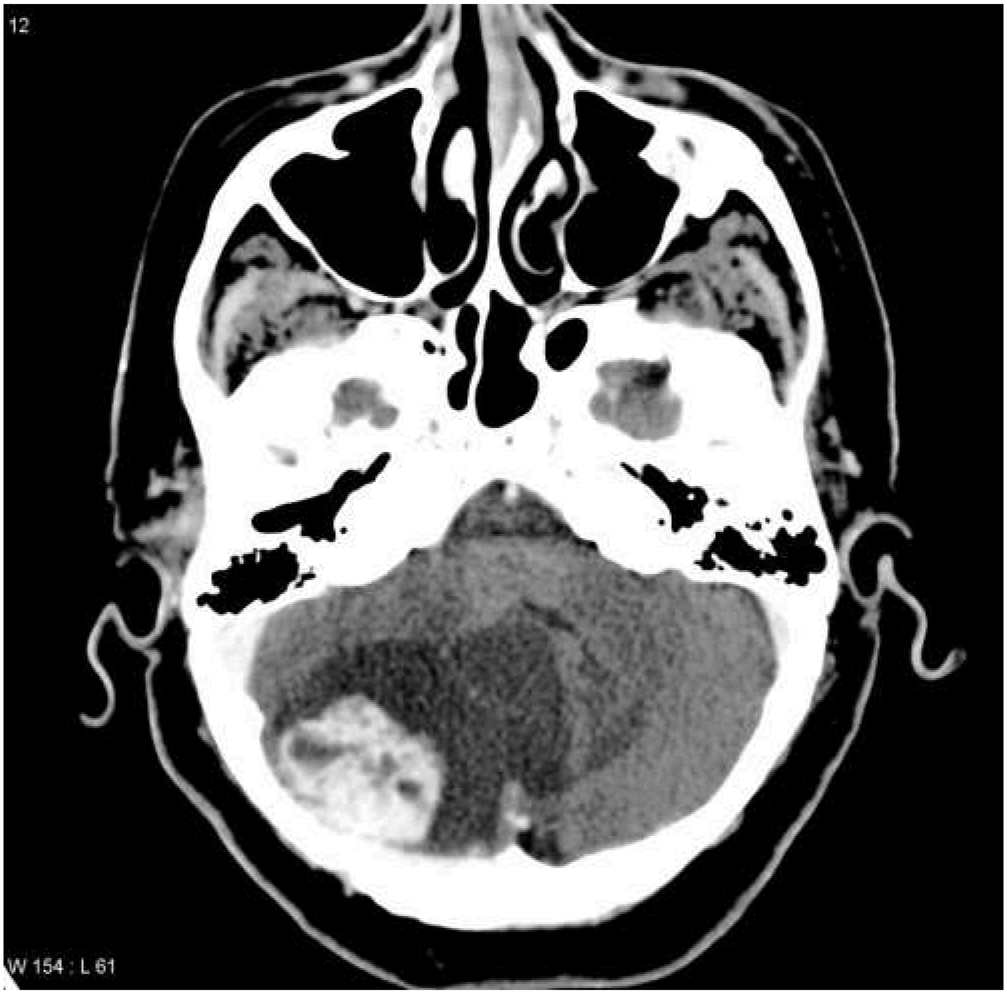

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What is the most likely underlying diagnosis?

Friedreich's ataxia 9%

Ataxic telangiectasia	16%
Neurofibromatosis	6%
Tuberous sclerosis	11%
Von Hippel-Lindau syndrome	58%

Retinal + cerebellar haemangiomas = Von Hippel-Lindau syndrome

Important for me Less important

The correct answer is **Von Hippel-Lindau syndrome**. Von Hippel-Lindau (VHL) syndrome is a rare, autosomal dominant disorder characterized by the development of various benign and malignant tumours, including hemangioblastomas in the central nervous system (CNS), retinal angiomas, renal cell carcinomas, and pheochromocytomas. The patient's presentation with ataxia, nystagmus, intention tremor, past pointing, and a retinal lesion surrounded by dilated vessels is highly suggestive of CNS hemangioblastoma and retinal angioma associated with VHL. A cerebellar lesion is also clearly seen in the neuroimaging. The family history of similar problems in his father also supports this diagnosis.

Friedreich's ataxia is an autosomal recessive inherited disorder that primarily affects the spinal cord and peripheral nerves. It typically presents with progressive limb and gait ataxia, dysarthria, loss of deep tendon reflexes, sensory neuropathy, and skeletal deformities such as scoliosis. Although Friedreich's ataxia can cause some similar neurological symptoms to VHL syndrome (e.g., ataxia), it does not explain the retinal lesion or strong family history seen in this case.

Ataxic telangiectasia, also known as Louis-Bar syndrome or Ataxia-telangiectasia (A-T), is an autosomal recessive disorder characterized by cerebellar degeneration leading to progressive ataxia along with oculocutaneous telangiectasias. Patients often present with recurrent sinopulmonary infections due to immunodeficiency. While some features like ataxia overlap with VHL syndrome in this case, A-T would not account for the retinal lesion or CT scan findings.

Neurofibromatosis refers to a group of genetic disorders causing the development of benign tumours, most commonly neurofibromas, in the nervous system. There are two main types: Neurofibromatosis type 1 (NF1) and Neurofibromatosis type 2 (NF2). NF1 is characterized by multiple cafe-au-lait spots, axillary and inguinal freckling, Lisch nodules in the iris, and neurofibromas. NF2 is associated with bilateral vestibular schwannomas (acoustic neuromas), meningiomas, and ependymomas. Neither type of neurofibromatosis would explain the retinal lesion or specific neurological symptoms seen in this case.

Tuberous sclerosis is an autosomal dominant disorder characterized by the formation of benign hamartomas in multiple organs including brain, skin, kidneys, heart, lungs, and eyes. Common features include seizures, cognitive impairment, facial angiofibromas, ash leaf spots on the skin and renal angiomyolipomas. Although some aspects of this syndrome might overlap with VHL syndrome (e.g., CNS involvement), it does not account for the specific retinal lesion described or other clinical findings in this case.

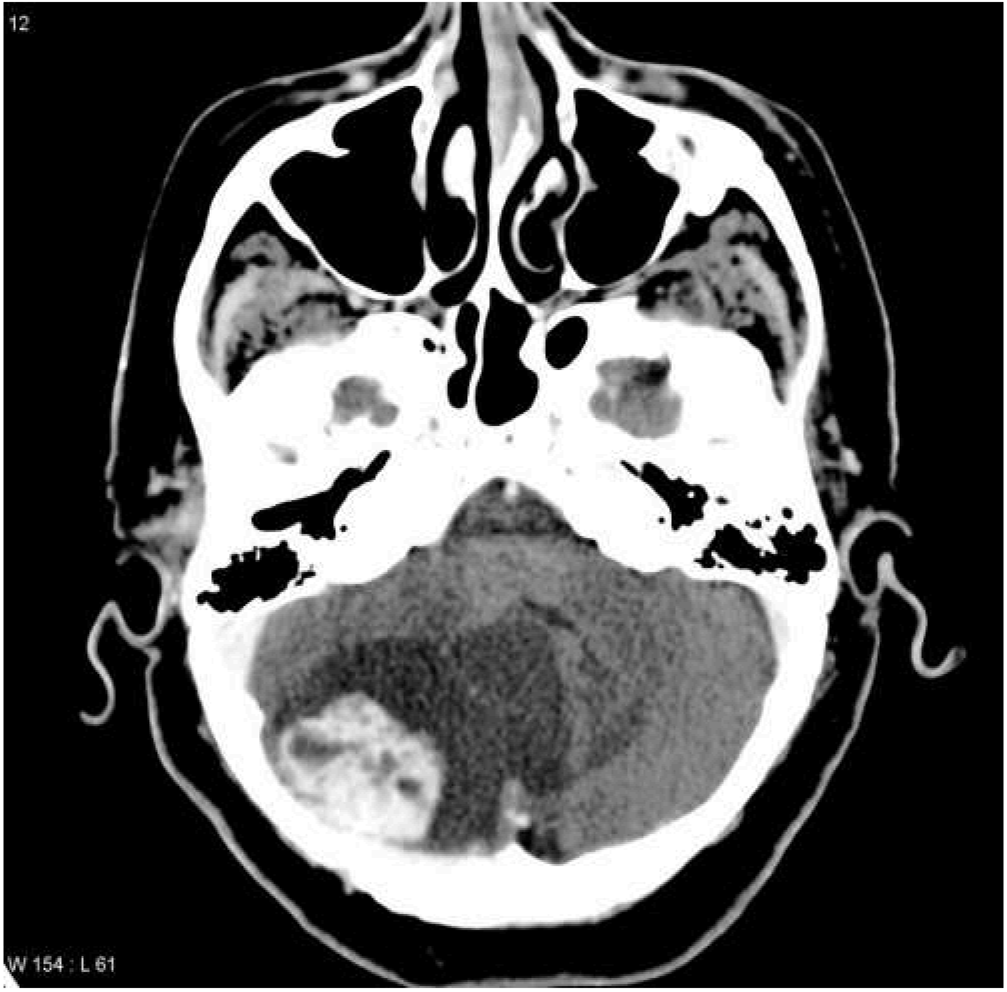
👍 | 👎 | Discuss (1) | Improve

Next question ›

Von Hippel-Lindau syndrome ★

Von Hippel-Lindau (VHL) syndrome is an autosomal dominant condition predisposing to neoplasia. It is due to an abnormality in the VHL gene located on short arm of chromosome 3

Features
- cerebellar haemangiomas: these can cause subarachnoid haemorrhages
- retinal haemangiomas: vitreous haemorrhage
- renal cysts (premalignant)
- phaeochromocytoma
- extra-renal cysts: epididymal, pancreatic, hepatic
- endolymphatic sac tumours
- clear-cell renal cell carcinoma



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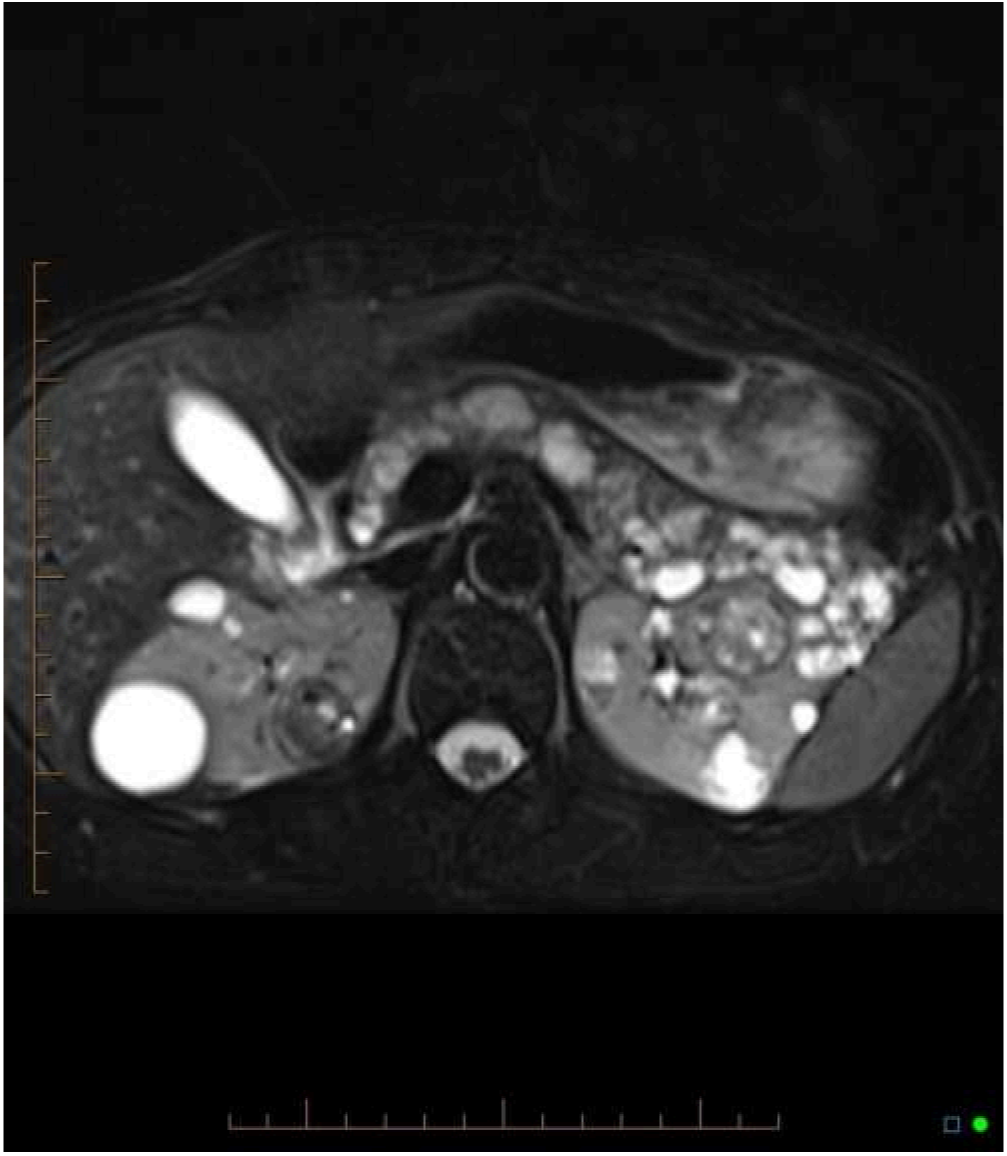
CT scan showing a cerebellar haemangioma in a patient with Von Hippel-Lindau syndrome.



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MRI showing renal cysts in patient with known Von Hippel-Lindau syndrome.

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **17.6%**

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A 32-year-old man is admitted to the neurology ward with a new leg weakness. He describes progressive weakness of his legs and now also his hands, which initially started 7 months ago and has progressed very slowly. He has minimal sensory disturbance in all four limbs but on examination of the legs he has grade 3/5 power throughout with absent reflexes and tone is not raised. He has no past medical history and no recent travel. A set of bloods on admission show the following:

Hb	141 g/l (115-165 g/l)	Na^+	141 mmol/l (135-145 mmol/l)
Platelets	301×10^9/l ($150-450 \times 10^9$/l)	K^+	4.1 mmol/l (3.5-5.2 mmol/l)
WBC	6.7×10^9/l ($2-11 \times 10^9$/l)	Urea	4.1 mmol/l (2.5-6.5 mmol/l)
Neuts	5.1×10^9/l ($1.5-7 \times 10^9$/l)	Creatinine	81 µmol/l (60-100 µmol/l)
Lymphs	1.2×10^9/l ($0.5-1.5 \times 10^9$/l)	CRP	0.9 mg/l (<5 mg/l)

A non-contrast CT head and a diagnostic lumbar puncture are also performed which show the following:

CT Head	No mass lesions, haemorrhage or acute major territory infarct seen. Ventricles of normal diameter. No skull vault fractures.

CSF Protein	611 mg/l (140-450 mg/l)
CSF WBC	1×10^6/l ($<5 \times 10^6$/l)
CSF Glucose	2.9 mmol/l (2.3-4.5 mmol/l)
CSF Electrophoresis	Normal pattern of IgG banding

Given the most likely diagnosis, which of the following treatments is most appropriate for this patient?

- ○ Conservative management
- ○ Therapeutic lumbar puncture
- ○ Intravenous antimicrobials
- ○ Intravenous immunoglobulin

○ High-dose steroids ×

Submit answer

Reference ranges ⌵

Score: **16%**

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CSF Glucose	2.9 mmol/l (2.3-4.5 mmol/l)
CSF Electrophoresis	Normal pattern of IgG banding

Given the most likely diagnosis, which of the following treatments is most appropriate for this patient?

Conservative management	8%
Therapeutic lumbar puncture	1%
Intravenous antimicrobials	1%
Intravenous immunoglobulin	67%

High-dose steroids 22%

CIDP is like GBS but with a more chronic onset and treated with steroids
Important for me Less important

From the history and clinical findings here, the most likely diagnosis would be chronic inflammatory demyelinating polyneuropathy or CIDP. The two main differentials would be Guillain-Barre syndrome and multiple sclerosis. Guillain-Barre syndrome would be a far more acute presentation with a rapid deterioration and multiple sclerosis would not produce such a global picture and in addition there would also be oligoclonal bands present on electrophoresis of the CSF. The chronic picture and high CSF protein are both in keeping with a diagnosis of CIDP above the others.

CIDP is an autoimmune inflammatory neuropathy which has an ongoing disease process and thus it responds well to immunosuppressive therapy. The most appropriate first line therapy therefore is high-dose steroids as this will achieve the goal of reducing inflammation and immunosuppressing a patient with a single drug. Therapeutic lumbar punctures are not indicated as there is no evidence for this being due to benign intracranial hypertension and antibiotics are also not indicated given the lack of infection markers.

Discuss (7) Improve

Next question ›

Chronic inflammatory demyelinating polyneuropathy

Overview

- cause of peripheral neuropathy
- antibody mediated inflammation results in segmental demyelination of peripheral nerves
- males more commonly affected than females
- features similar to Guillain-Barre syndrome (GBS), with motor features predominating
- more insidious onset, over weeks to months - often thought of as the chronic version of GBS
- high protein content in the CSF
- treatment with steroids and immunosuppressants may have a role (unlike in GBS)

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Score: **17.6%**

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A 27-year-old man is brought to the hospital having become unwell at a party. On arrival, he is drowsy and only responds to voice. His airway appears patent and his chest is clear. Pupils are 4mm and reactive to light. Observations are as follows.

- Respiratory rate 12 breaths /min
- Oxygen saturations 95% on air
- Heart rate 84 beats /min
- Blood pressure 117/65mmHg
- Temperature 36.7ºC

Due to his reduced conscious level, you organise a CT head. While there, a friend phones the hospital and tells you he was seen taking some tablets at the party roughly 90 minutes ago and they have since found empty packets of diazepam in the house.

He returns from CT head which is reported as normal. You are asked to see him urgently as he is now unresponsive and making snoring sounds. These improve with a jaw thrust but recur if this is released. The examination is otherwise the same as on arrival.

What is the best course of action?

○ Activated charcoal

○ Flumazenil

○ Haemodialysis

○ Intubation and ventilation

○ Naloxone

Submit answer

Reference ranges

Score: **16%**

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He returns from CT head which is reported as normal. You are asked to see him urgently as he is now unresponsive and making snoring sounds. These improve with a jaw thrust but recur if this is released. The examination is otherwise the same as on arrival.

What is the best course of action?

Activated charcoal	2%
Flumazenil	29%
Haemodialysis	1%
Intubation and ventilation	64%
Naloxone	4%

Benzodiazepine overdose is best managed supportively and with airway protection and ventilation if needed. Flumazenil should be avoided unless for reversal of anaesthesia

Important for me Less important

The correct answer is **intubation and ventilation**. Drug ingestion leading to CNS depression is common and may be due to a variety of substances. This picture with normal observations and normal pupils should raise the suspicion of benzodiazepine use, especially with the empty packets found at the house. The mainstay of treatment in such a case is supportive, with securing the airway the main priority in the case described.

Activated charcoal is incorrect. This can bind some substances and minimise their effects if given sufficiently early after ingestion. However, its role in benzodiazepine overdose is uncertain. Toxbase advises that it can be considered within the first hour after ingestion provided the airway is protected. In this case, the patient is outwith this timeframe and the airway is unsafe.

Flumazenil is incorrect. This antidote is effective in reversing the effects of benzodiazepine toxicity. However, reversal of an unknown quantity can lead to rebound seizures which are then refractory to benzodiazepine therapy as the receptor is blocked by the drug. Flumazenil should generally only be used in an iatrogenic overdose where the exact quantity is known. In all other cases, supportive treatment such as airway management is the mainstay.

Haemodialysis is incorrect. This is used for the removal of some toxic agents such as lithium. However, it does not remove benzodiazepines and would not be effective here.

Naloxone is incorrect. This is a highly effective antidote for opiate toxicity and acts quickly enough that it could avoid the need for intubation in such cases. However, the clinical picture is not suggestive of opiate toxicity. This generally presents with other features such as myoclonic jerks and pinpoint pupils. Opiates are also more commonly used by injection rather than as tablets.

👍 👎 Discuss (2) Improve

Next question >

Status epilepticus ★

Status epilepticus is defined as:

- a single seizure lasting >5 minutes, or
- >= 2 seizures within a 5-minute period without the person returning to normal between them

This is a medical emergency. The priority is the termination of seizure activity, which if prolonged will lead to irreversible brain damage.

Management

- ABC
  - airway adjunct
  - oxygen

    - check blood glucose
- First-line drugs are benzodiazepines
    - in the prehospital setting PR diazepam or buccal midazolam may be given
    - in hospital IV lorazepam is generally used. This may be repeated once after 5-10minutes
- If ongoing (or 'established') status it is appropriate to start a second-line agent such as levetiracetam, phenytoin or sodium valproate
    - NICE state 'Take into account that levetiracetam may be quicker to administer and have fewer adverse effects than the alternative options.'
- If no response ('refractory status') within 45 minutes from onset, then the best way to achieve rapid control of seizure activity is induction of general anaesthesia or phenobarbital

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 14 👎 9

2022 Epilepsy guidelines

Suggest link Report broken link

Score: **17.6%**

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Question 40 of 299

A 37-year-old man is being reviewed in the eye clinic for painless loss of vision bilaterally. The symptoms started 1 year ago when he initially developed central scotomas, followed by loss of colour vision and ultimately loss of all vision. He has no significant past medical history.

On examination, visual acuity is 3/60 in both eyes. Fundoscopy reveals optic atrophy in both eyes, with narrow vessels and absent macular reflexes. An MRI scan of the brain shows significantly lower optic nerve volumes than normal. His mother also lost her vision at 34 years of age. His brother and sister, who are both a few years older, developed the same symptoms several years ago.

What is the most likely diagnosis?

- ○ Autosomal dominant optic atrophy (ADOA)
- ○ DIDMOAD syndrome
- ○ Leber's optic atrophy
- ○ MELAS syndrome
- ○ Optic neuritis

Submit answer

Reference ranges

Score: **16%**

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What is the most likely diagnosis?

Autosomal dominant optic atrophy (ADOA)	27%
DIDMOAD syndrome	4%
Leber's optic atrophy	61%
MELAS syndrome	7%
Optic neuritis	2%

30-years-old, central scotoma → loss of colour vision → rapid onset of significant visual impairment ? Leber's optic atrophy

Important for me Less important

The history of central scotoma, colour blindness, and rapid onset of visual impairment point in an adult male point toward a diagnosis of **Leber's optic atrophy** in this scenario. A history of a similar illness in the mother and all siblings confirms the mitochondrial pattern of inheritance in which all children of an affected mother inherit the disease. Diagnosis of mitochondrial disease is confirmed by muscle biopsy showing ragged red fibres due to an increased number of mitochondria.

Although optic atrophy is a prominent feature of **autosomal dominant optic atrophy (ADOA)** , vision loss in ADOA occurs in late childhood or early adolescence. The onset of vision loss in ADOA is gradual and not rapid as in this scenario. The pattern of inheritance described is mitochondrial and not autosomal dominant, therefore this option is incorrect.

MELAS syndrome is characterised by mitochondrial encephalomyopathy lactic acidosis and stroke-like episodes. There is no history of seizures, weakness, metabolic disturbances, or altered consciousness and so MELAS is an unlikely diagnosis here.

The family history in this scenario demonstrates mitochondrial inheritance and effectively rules out acquired/autoimmune causes of vision loss such as **optic neuritis**. Optic neuritis is usually associated with multiple sclerosis, diabetes mellitus, or recent infection.

Optic atrophy and vision loss in **DIDMOAD syndrome** occur in childhood or adolescence. Optic atrophy in DIDMOAD syndrome is accompanied by diabetes insipidus, diabetes mellitus, and sensorineural deafness. The unremarkable past medical history of this patient rules out this diagnosis.

👍 👎 Discuss (1) Improve

Next question >

Mitochondrial diseases

Whilst most DNA is found in the cell nucleus, a small amount of double-stranded DNA is present in the mitochondria. It encodes protein components of the respiratory chain and some special types of RNA

Mitochondrial inheritance has the following characteristics:
- inheritance is only via the maternal line as the sperm contributes no cytoplasm to the zygote
- none of the children of an affected male will inherit the disease
- all of the children of an affected female will inherit the disease
- generally, encode rare neurological diseases
- poor genotype:phenotype correlation - within a tissue or cell there can be different mitochondrial populations - this is known as heteroplasmy

Histology
- muscle biopsy classically shows 'red, ragged fibres' due to increased number of mitochondria

Examples include:
- Leber's optic atrophy
  - symptoms typically develop at around the age of 30 years
  - central scotoma → loss of colour vision → rapid onset of significant visual impairment
- MELAS syndrome: mitochondrial encephalomyopathy lactic acidosis and stroke-like episodes

- MERRF syndrome: myoclonus epilepsy with ragged-red fibres
- Kearns-Sayre syndrome: onset in patients < 20 years old, external ophthalmoplegia, retinitis pigmentosa. Ptosis may be seen
- sensorineural hearing loss

+ Q 123

Next question >

B *I*

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 5 👎 6

2013 How to spot mitochondrial disease in adults

Rare Disease Video Library - NORD 👍 3 👎 4

Primary Mitochondrial Myopathy

Suggest link

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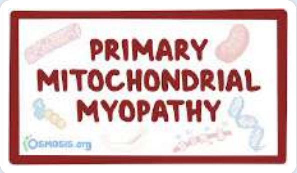
Media



Primary mitochondrial myopathy

Osmosis - YouTube 👍 6 👎 1

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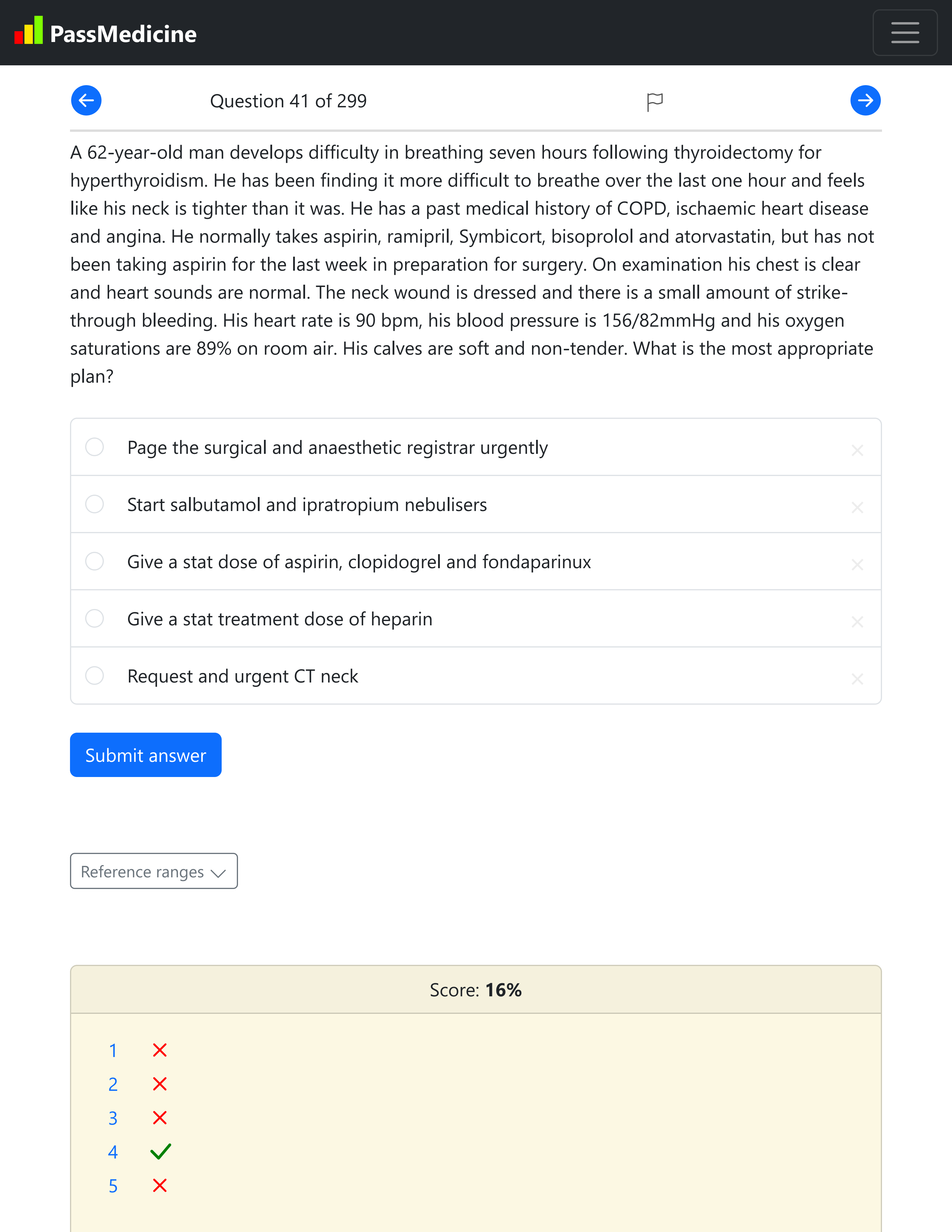

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Question 41 of 299

A 62-year-old man develops difficulty in breathing seven hours following thyroidectomy for hyperthyroidism. He has been finding it more difficult to breathe over the last one hour and feels like his neck is tighter than it was. He has a past medical history of COPD, ischaemic heart disease and angina. He normally takes aspirin, ramipril, Symbicort, bisoprolol and atorvastatin, but has not been taking aspirin for the last week in preparation for surgery. On examination his chest is clear and heart sounds are normal. The neck wound is dressed and there is a small amount of strike-through bleeding. His heart rate is 90 bpm, his blood pressure is 156/82mmHg and his oxygen saturations are 89% on room air. His calves are soft and non-tender. What is the most appropriate plan?

○ Page the surgical and anaesthetic registrar urgently ×

○ Start salbutamol and ipratropium nebulisers ×

○ Give a stat dose of aspirin, clopidogrel and fondaparinux ×

○ Give a stat treatment dose of heparin ×

○ Request and urgent CT neck ×

Submit answer

Reference ranges

Score: **16%**

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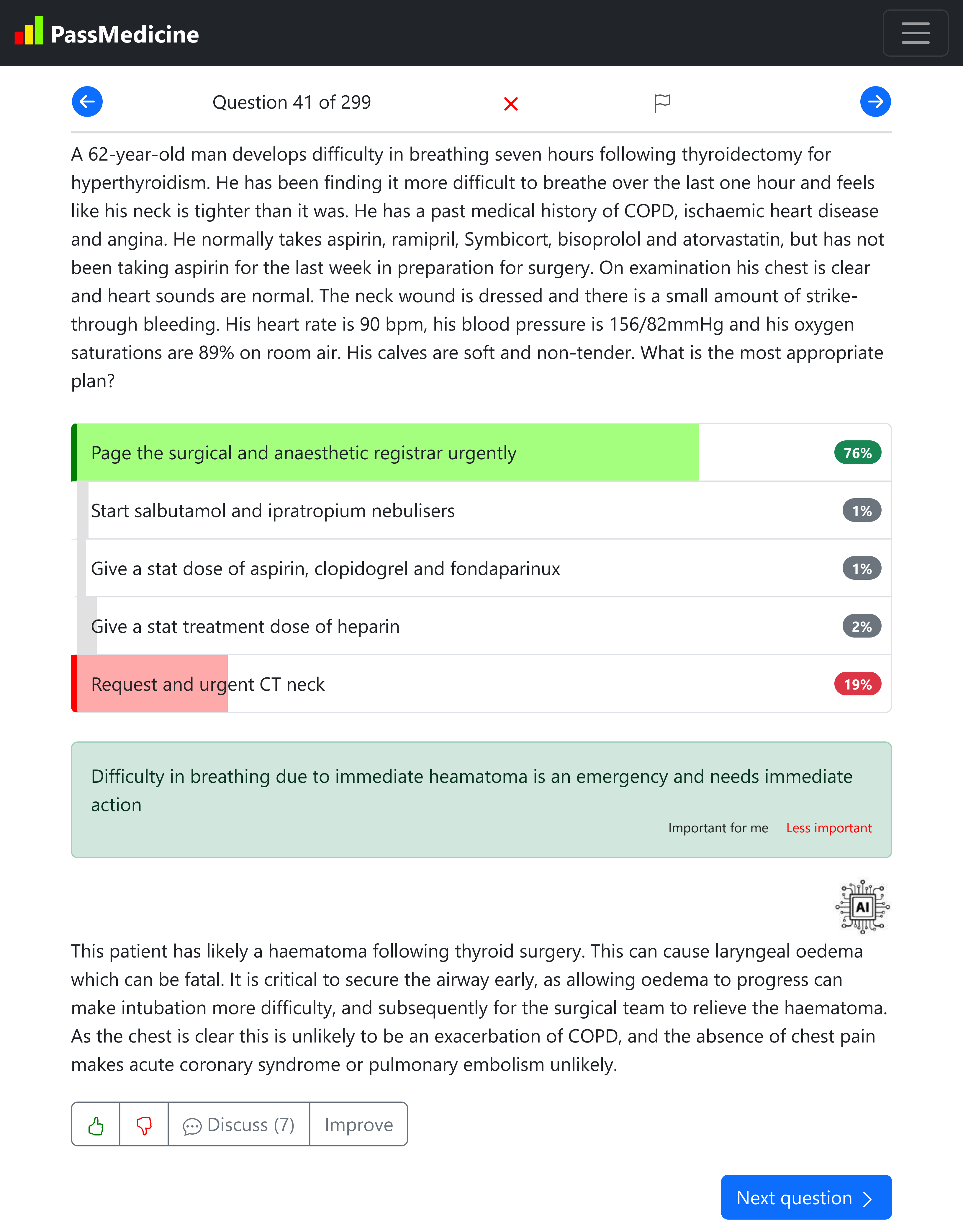

A 62-year-old man develops difficulty in breathing seven hours following thyroidectomy for hyperthyroidism. He has been finding it more difficult to breathe over the last one hour and feels like his neck is tighter than it was. He has a past medical history of COPD, ischaemic heart disease and angina. He normally takes aspirin, ramipril, Symbicort, bisoprolol and atorvastatin, but has not been taking aspirin for the last week in preparation for surgery. On examination his chest is clear and heart sounds are normal. The neck wound is dressed and there is a small amount of strike-through bleeding. His heart rate is 90 bpm, his blood pressure is 156/82mmHg and his oxygen saturations are 89% on room air. His calves are soft and non-tender. What is the most appropriate plan?

Page the surgical and anaesthetic registrar urgently	76%
Start salbutamol and ipratropium nebulisers	1%
Give a stat dose of aspirin, clopidogrel and fondaparinux	1%
Give a stat treatment dose of heparin	2%
Request and urgent CT neck	19%

Difficulty in breathing due to immediate heamatoma is an emergency and needs immediate action

Important for me Less important

This patient has likely a haematoma following thyroid surgery. This can cause laryngeal oedema which can be fatal. It is critical to secure the airway early, as allowing oedema to progress can make intubation more difficulty, and subsequently for the surgical team to relieve the haematoma. As the chest is clear this is unlikely to be an exacerbation of COPD, and the absence of chest pain makes acute coronary syndrome or pulmonary embolism unlikely.

Subtotal thyroidectomy ★

Surgery complications

- hypothyroidism: most common
- immediate haematoma (airway compression)
- hypoparathyroidism → hypocalcaemia (paraesthesia, spasm)
- recurrent laryngeal nerve palsy (hoarseness, bovine cough)
- superior laryngeal nerve injury (loss of pitch)
- thyroid crisis (fast AF, pulmonary oedema)

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **17.6%**

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A 33-year-old female is referred to the neurology outpatient department by her general practitioner. She complains of five months of excruciating right-sided throbbing headaches, localized to the temporal region. The headaches start and stop abruptly and last for approximately 15 minutes at a time. They occur 9-12 times a day and are associated with nasal congestion and rhinorrhoea. There is no significant past medical history and she is not on any regular medications. She has a family history of migraine and smokes ten cigarettes per day. She does not drink alcohol.

Neurological examination at the time of clinic review is unremarkable.

Hb	140 g/L	Male: (135-180) Female: (115 - 160)
Platelets	176 * 10^9/L	(150 - 400)
WBC	8 * 10^9/L	(4.0 - 11.0)
Na$^+$	139 mmol/L	(135 - 145)
K$^+$	4 mmol/L	(3.5 - 5.0)
Urea	5 mmol/L	(2.0 - 7.0)
Creatinine	64 µmol/L	(55 - 120)
CRP	3 mg/L	(< 5)

An MRI brain is organized, which is reported as normal.

Given the likely diagnosis, which medication treatment will result in resolution of her symptoms?

○ Carbamazepine

○ High flow oxygen

○ Indomethacin

○ Prednisolone

○ Sumatriptan

Submit answer

Reference ranges ⌵

Score: **16%**

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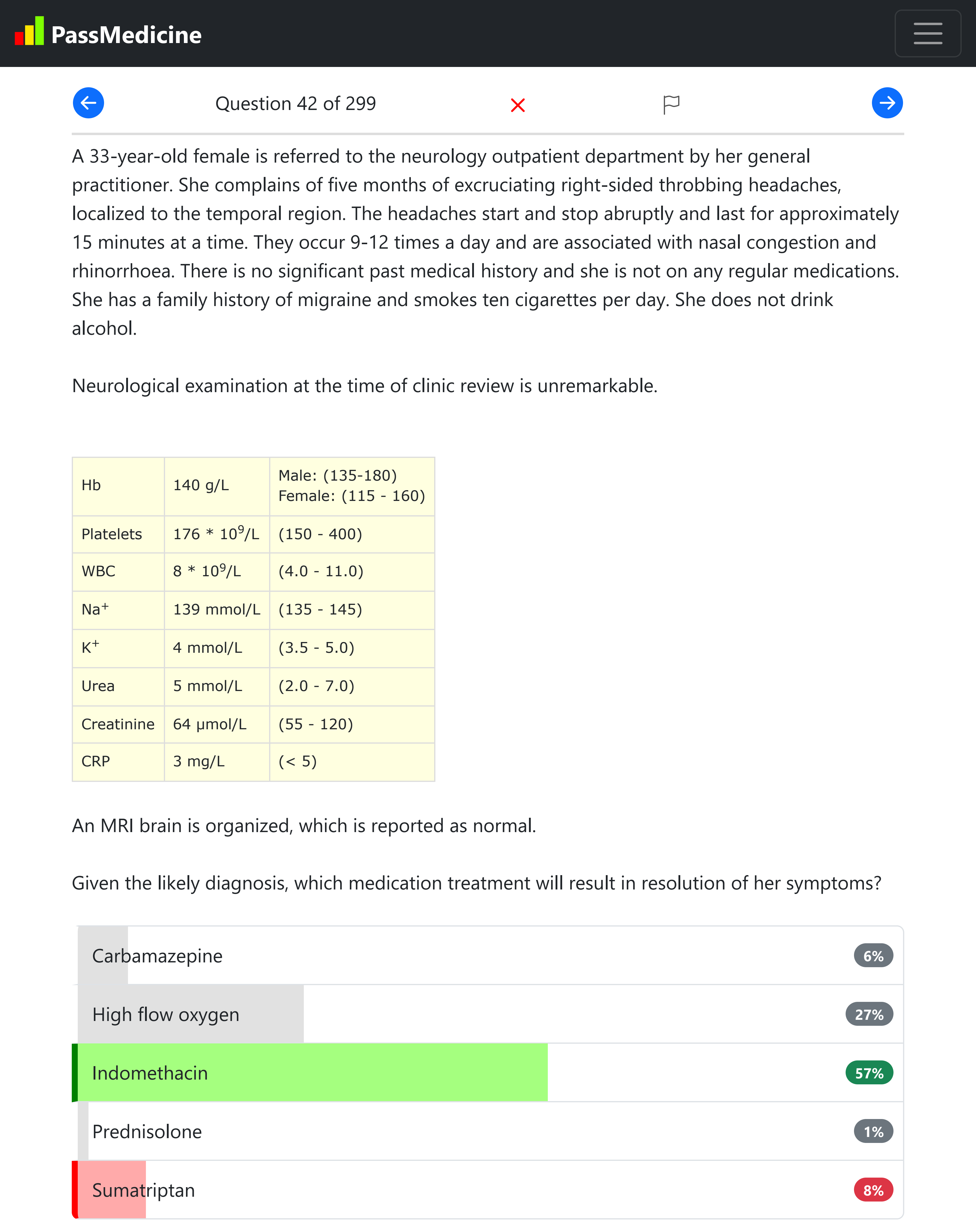

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Urea	5 mmol/L	(2.0 - 7.0)
Creatinine	64 µmol/L	(55 - 120)
CRP	3 mg/L	(< 5)

An MRI brain is organized, which is reported as normal.

Given the likely diagnosis, which medication treatment will result in resolution of her symptoms?

Carbamazepine	6%
High flow oxygen	27%
Indomethacin	57%
Prednisolone	1%
Sumatriptan	8%

Paroxysmal hemicrania is completely responsive to treatment with indomethacin

Important for me Less important

Indomethacin is the correct answer. Paroxysmal hemicrania is characterized by relatively short-lasting, frequent unilateral headaches associated with ipsilateral autonomic features (such as rhinorrhoea and nasal congestion in this case). It shares many features with cluster headaches but can be differentiated based on its dramatic response to indomethacin, higher prevalence in females, shorter attack duration and higher attack frequency. Brain imaging is useful to exclude a secondary cause of presentation.

Carbamazepine is not the right answer. This is used as a treatment for trigeminal neuralgia. This headache is not characterized by ipsilateral autonomic features and the pain affects the lower part of the face in the distribution of the trigeminal nerve.

High flow oxygen is not the right answer. This treatment is used to abort attacks of cluster headache. Cluster headaches share many of the same features as paroxysmal hemicrania but the attacks tend to be of longer duration (45-90 minutes) and occur less frequently (1-8 times per day on average). Patients often describe circadian rhythmicity to their symptoms, with the headaches occurring at similar times each day.

Prednisolone is not the right answer. High dose prednisolone can be used to treat the temporal headache of giant cell arteritis. However, the young age of this patient essentially excludes this diagnosis.

Sumatriptan is the wrong answer. This is used as a treatment for both cluster headaches and migraines, neither of which is the correct answer here. The duration of migraine is typically longer and more classically associated with visual aura, paraesthesia, photophobia and phonophobia.

👍 👎 Discuss (2) Improve

Next question >

Paroxysmal hemicrania ★

Paroxysmal hemicrania (PH) is defined by attacks of severe, unilateral headache, usually in the orbital, supraorbital or temporal region. These attacks are often associated with autonomic features, usually last less than 30 minutes and can occur multiple times a day.

PH sits within the group of disorders called trigeminal autonomic cephalgias which also contains cluster headache, a condition which shares many features with PH.

Importantly, PH is completely responsive to treatment with indomethacin.

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **17.6%**

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A 55-year-old man is referred to neurology clinic by his GP with reports of a six month history of tremor affecting both hands. It worsens with emotional stress. He has no past medical history and does not take any medications. He does not smoke or drink alcohol. He works as a cooper.

On examination, there is a tremor of both hands with an frequency of 5 Hz and a medium amplitude. It is slightly more prominent on the right hand than the left but this is subtle. It is present both posturally and at rest. There are no other discriminating features.

Given the clinical presentation, which test is most appropriate to guide diagnosis?

- ○ Acetylcholine receptor antibodies
- ○ Lumbar puncture
- ○ MRI head
- ○ Nerve conduction studies
- ○ 123I-FP-CIT SPECT

Submit answer

Reference ranges

Score: **16%**

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On examination, there is a tremor of both hands with an frequency of 5 Hz and a medium amplitude. It is slightly more prominent on the right hand than the left but this is subtle. It is present both posturally and at rest. There are no other discriminating features.

Given the clinical presentation, which test is most appropriate to guide diagnosis?

Acetylcholine receptor antibodies	3%
Lumbar puncture	1%
MRI head	21%
Nerve conduction studies	21%
123I-FP-CIT SPECT	54%

^{123}I-FP-CIT SPECT can be used to help differentiate essential tremor from Parkinson's

Important for me Less important

123I-FP-CIT SPECT is the correct answer. In this scenario, there is uncertainty as to whether this tremor represents an essential tremor or a Parkinsonian tremor. The so called 'DaTSCAN' can be used to differentiate between essential tremor and a Parkinsonian tremor if there is diagnostic uncertainty. A DaTSCAN measures the density of nigrostriatial dopamine transporter sites. A radioactive tracer is injected into the blood and binds to the dopamine transporter in the brain. The imaging technique assess for the level of binding observed. In Parkinson's disease, there is a decreased level of binding between the tracer and the dopamine transporter in the brain. In essential tremor, the binding level will be normal.

Lumbar puncture is incorrect. Typically, this investigation is not necessary in differentiating between essential tremor and PD. However, if there was a suspicion of an intra-cerebral infection-causing tremor, it may be useful.

MRI head is incorrect. This may show vascular changes if vascular parkinsonism is suspected.

However, this type of Parkinsonism tends to be lower body predominant and tremor is a less common feature.

Nerve conduction studies is incorrect. This is a test that can help diagnose neuropathies but is little value in determining the cause of tremors.

Acetylcholine receptor antibodies are incorrect. This is an antibody test used to support a diagnosis of myasthenia gravis. This disorder is characterized by fatigable weakness rather than tremor.

👍 👎 Discuss (3) Improve

Next question >

Parkinson's disease: features

Parkinson's disease is a progressive neurodegenerative condition caused by degeneration of dopaminergic neurons in the substantia nigra. The reduction in dopaminergic output results in a classical triad of features: bradykinesia, tremor and rigidity. The symptoms of Parkinson's disease are characteristically asymmetrical.

Epidemiology
- around twice as common in men
- mean age of diagnosis is 65 years

Bradykinesia
- poverty of movement also seen, sometimes referred to as hypokinesia
- short, shuffling steps with reduced arm swinging
- difficulty in initiating movement

Tremor
- most marked at rest, 3-5 Hz
- worse when stressed or tired, improves with voluntary movement
- typically 'pill-rolling', i.e. in the thumb and index finger

Rigidity
- lead pipe
- cogwheel: due to superimposed tremor

Other characteristic features

- mask-like facies
- flexed posture
- micrographia
- drooling of saliva
- psychiatric features: depression is the most common feature (affects about 40%); dementia, psychosis and sleep disturbances may also occur
- impaired olfaction
- REM sleep behaviour disorder
- fatigue
- autonomic dysfunction:
  - postural hypotension

Drug-induced parkinsonism has slightly different features to Parkinson's disease:

- motor symptoms are generally rapid onset and bilateral
- rigidity and rest tremor are uncommon

Diagnosis is usually clinical. However, if there is difficulty differentiating between essential tremor and Parkinson's disease NICE recommend considering ^{123}I-FP-CIT single photon emission computed tomography (SPECT).

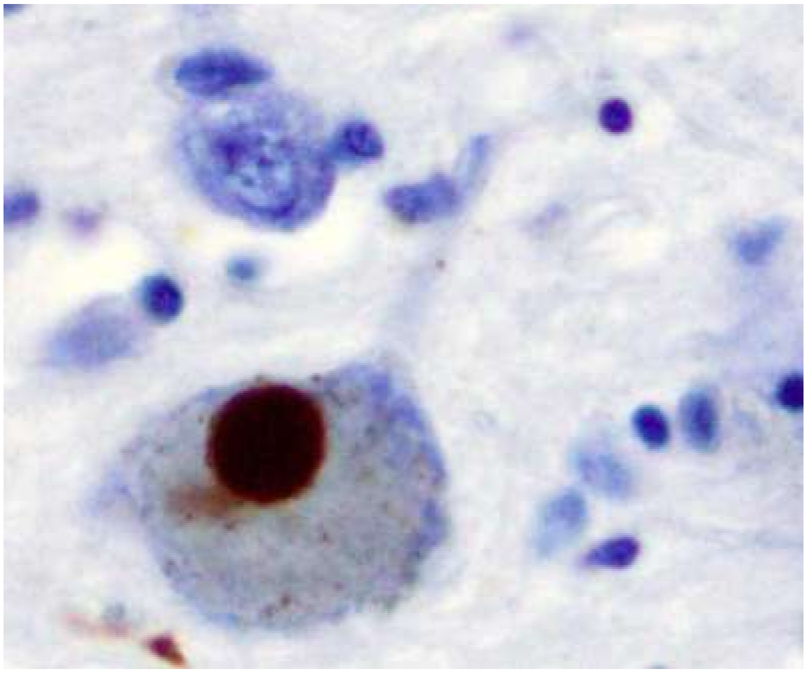


Image sourced from Wikipedia

A Lewy body (stained brown) in a brain cell of the substantia nigra in Parkinson's disease. The brown colour is positive immunohistochemistry staining for alpha-synuclein.

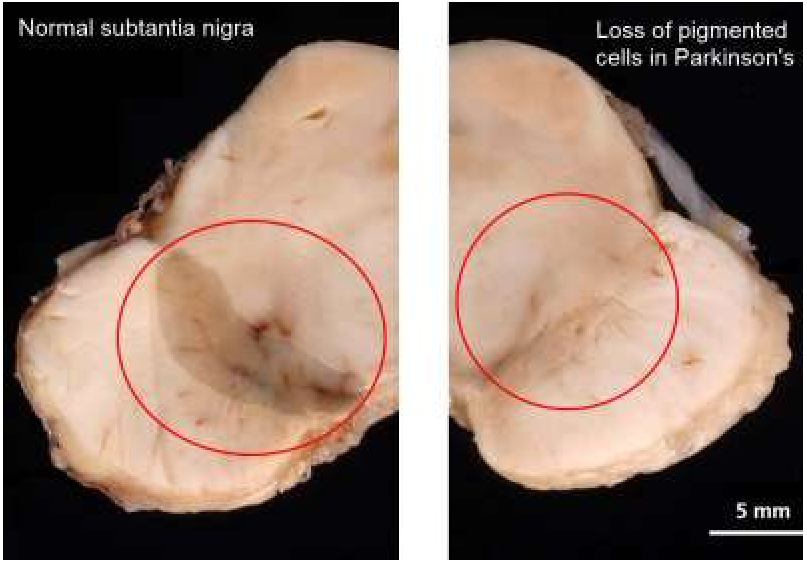

Normal subtantia nigra
Loss of pigmented cells in Parkinson's
5 mm

Image sourced from Wikipedia

Discoloration of the substantia nigra due to loss of pigmented nerve cells.

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 8 👎 9

2017 Parkinson's disease guidelines

Suggest link

Report broken link

Media

Parkinsonian gait

mhdbelalalsabek - YouTube 👍 6 👎 0

PARKINSON'S
DISEASE
PATHOPHYSIOLOGY

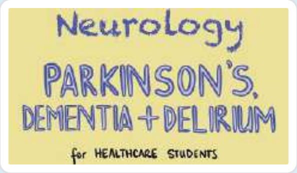
Parkinson's Disease: How is the brain affected?

Soton Brain Hub - YouTube 👍 1 👎 0

Parkinson's
Disease
Symptoms

Parkinson's Disease Symptoms

Soton Brain Hub - YouTube 👍 0 👎 0


Neurology
PARKINSON'S,
DEMENTIA + DELIRIUM
for HEALTHCARE STUDENTS

Parkinson's disease

MedFlix - YouTube 👍 0 👎 0

PARKINSON'S
DISEASE
Osmosis.org

Parkinson's disease

Osmosis - YouTube 👍 2 👎 1

SHUFFLING GAIT
PARKINSON'S
DISEASE
α-SYNUCLEIN
PILL ROLLING TREMOR
COG-WHEEL RIGIDITY
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Understanding Parkinson's Disease

Rhesus Medicine - YouTube 👍 1 👎 1

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Score: **17.6%**

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Question 44 of 299

A 23-year-old presents to the emergency department. He has arrived with a headache however nursing staff are concerned by the appearance on the ECG taken at triage. He is usually well. He is clear that he does not have any chest pain or shortness of breath. He does report that his head is really hurting and this started suddenly in the morning when he was preparing his breakfast.

On examination, he is a young slim gentleman with normal heart sounds. His lung fields are clear and there is no visible JVP. He is difficult to examine due to agitation and he refuses to open his eyes to the light. His blood pressure is 140/80 mmHg.

ECG	ST elevation in leads V4-V6

What is the likeliest cause of the ECG changes?

- ○ Benign early re-polarisation
- ○ Left ventricular hypertrophy
- ○ Subarachnoid haemorrhage
- ○ Silent myocardial infarction
- ○ Acute pericarditis

Submit answer

Reference ranges

Score: **16%**

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ECG	ST elevation in leads V4-V6

What is the likeliest cause of the ECG changes?

Benign early re-polarisation	7%
Left ventricular hypertrophy	1%
Subarachnoid haemorrhage	89%
Silent myocardial infarction	1%
Acute pericarditis	1%

ST elevation can occur in subarachnoid haemorrhage

Important for me Less important

This gentleman has ST elevation but no cardiac signs. This could be benign early re-polarisation also called 'high take off' but the severe headache suggests this could be a subarachnoid haemorrhage. His slim physique may suggest a Marfan's syndrome which is a recognised cause of cerebral aneurysm. A silent myocardial infarction is rare particularly in young males and there are no other symptoms seen like shortness of breath. Acute pericarditis has ST elevation in all leads and sharp chest pains. Left ventricular hypertrophy presents with large volume complexes but not ST elevation.

Discuss (3) | Improve

Next question >

Subarachnoid haemorrhage

A subarachnoid haemorrhage (SAH) is an intracranial haemorrhage that is defined as the presence of blood within the subarachnoid space, i.e. deep to the subarachnoid layer of the meninges.

The most common cause of SAH is head injury and this is called *traumatic SAH* . In the absence of trauma, SAH is termed *spontaneous SAH* . The rest of this note focuses on spontaneous SAH.

Causes of spontaneous SAH include:

- intracranial aneurysm (saccular 'berry' aneurysms)
    - accounts for around 85% of cases
    - conditions associated with berry aneurysms include hypertension,adult polycystic kidney disease, Ehlers-Danlos syndrome and coarctation of the aorta
- arteriovenous malformation
- pituitary apoplexy
- mycotic (infective) aneurysms

Classical presenting features include:

- headache
    - usually of sudden-onset ('thunderclap' or 'hit with a baseball bat')
    - severe ('worst of my life')
    - occipital
    - typically peaking in intensity within 1 to 5 minutes
    - there may be a history of a less-severe 'sentinel' headache in the weeks prior to presentation
- nausea and vomiting
- meningism (photophobia, neck stiffness)
- coma
- seizures
- ECG changes including ST elevation may be seen
    - this may be secondary to either autonomic neural stimulation from the hypothalamus or elevated levels of circulating catecholamines

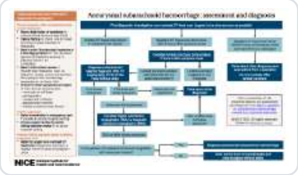
Investigation

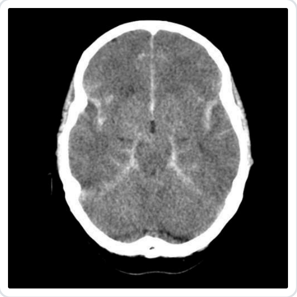
- non-contrast CT head is the first-line investigation of choice

    - acute blood (hyperdense/bright on CT) is typically distributed in the basal cisterns, sulci and in severe cases the ventricular system.
- if CT head is done within 6 hours of symptom onset and is normal
    - new guidelines suggest **not** doing a lumbar puncture
    - consider an alternative diagnosis
- if CT head is done more than 6 hours after symptom onset and is normal
    - do a lumber puncture (LP)
    - timing wise the LP should be performed at least 12 hours following the onset of symptoms to allow the development of xanthochromia (the result of red blood cell breakdown).
    - xanthochromia helps to distinguish true SAH from a 'traumatic tap' (blood introduced by the LP procedure).
    - as well as xanthochromia, CSF findings consistent with subarachnoid haemorrhage include a normal or raised opening pressure
- if the CT shows evidence of a SAH
    - referral to neurosurgery to be made as soon as SAH is confirmed

After spontaneous SAH is confirmed, the aim of investigation is to identify a causative pathology that needs urgent treatment:

- CT intracranial angiogram (to identify a vascular lesion e.g. aneurysm or AVM)
- +/- digital subtraction angiogram (catheter angiogram)





Management of a confirmed aneurysmal subarachnoid haemorrhage

- supportive
    - bed rest
    - analgesia
    - venous thromboembolism prophylaxis
    - discontinuation of antithrombotics (reversal of anticoagulation if present)
- vasospasm is prevented using a course of oral nimodipine
- intracranial aneurysms are at risk of rebleeding and therefore require prompt intervention, preferably within 24 hours
    - most intracranial aneurysms are now treated with a coil by interventional neuroradiologists, but a minority require a craniotomy and clipping by a neurosurgeon

Complications of aneurysmal SAH:

- re-bleeding
  - happens in around 10% of cases and most common in the first 12 hours
  - if rebleeding is suspected (e.g. sudden worsening of neurological symptoms) then a repeat CT should be arranged
  - associated with a high mortality (up to 70%)
- hydrocephalus
  - hydrocephalus is temporarily treated with an external ventricular drain (CSF diverted into a bag at the bedside) or, if required, a long-term ventriculoperitoneal shunt
- vasospasm (also termed delayed cerebral ischaemia), typically 7-14 days after onset
  - ensure euvolaemia (normal blood volume)
  - consider treatment with a vasopressor if symptoms persist
- hyponatraemia (most typically due to syndrome inappropriate anti-diuretic hormone (SIADH))
- seizures

Important predictive factors in SAH:

- conscious level on admission
- age
- amount of blood visible on CT head

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia 👍 15 👎 15

Subarachnoid haemorrhage

Life in the Fast Lane 👍 19 👎 10

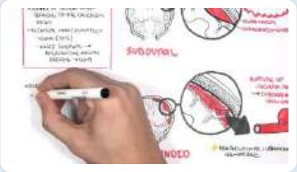
Example ECG changes in subarachnoid haemorrhage

NICE 👍 9 👎 4

2022 Subarachnoid haemorrhage caused by a ruptured aneurysm: diagnosis and management

Suggest link Report broken link

Media



Intracranial Haemorrhage Types

Armando Hasudungan - YouTube 👍 2 👎 0

Arteriovenous malformation

Osmosis - YouTube 👍 0 👎 0

NEUROLOGY
NEUROSURGERY
SUBARACHNOID HAEMORRHAGE

Subarachnoid Haemorrhage / pathophysiology, complications and management

Armando Hasudungan - YouTube 👍 0 👎 0

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A 25-year-old gentleman presents with a headache to the emergency department. He describes walking back from the gym and developing an occipital headache that came on suddenly and became a 9/10 severity within seconds. He started to vomit and a bystander called an ambulance.

He is normally fit and well. He takes no medication regularly and denies any recreational substance use. He has been referred to the genetics clinic due to his father having been diagnosed with a phaeochromocytoma and his maternal grandmother had kidney tumours.

On arrival to the emergency department, his glasgow coma scale has dropped from 14 to 6. He has reactive pupils and an intact corneal reflex. His neck is rigid and he screams out when you try to flex his neck. His blood pressure is 180/90mmHg, heart rate 60/min and respiratory rate is 11/min. He has two cafe-au-lait spots on his trunk but there are no palpable masses and no changes seen in the irises. He is 6 foot 5 inches and weighs 95 kilograms. Abdominal examination is unremarkable.

Urine dip	blood trace, leucocytes positive
ECG	ST elevation in V1-V4
CT head	large high attenuation signal seen in the left cerebellar hemisphere with surrounding oedema, no mass effect
Lumbar puncture	xanthochromia colour, raised red cells

What is the likely unifying diagnosis?

- ○ Tuberous sclerosis
- ○ Neurofibromatosis type 1
- ○ Von Hippel Lindau
- ○ Multiple endocrine neoplasia
- ○ Polycystic kidney disease

Submit answer

Reference ranges ⌵

Score: **16%**

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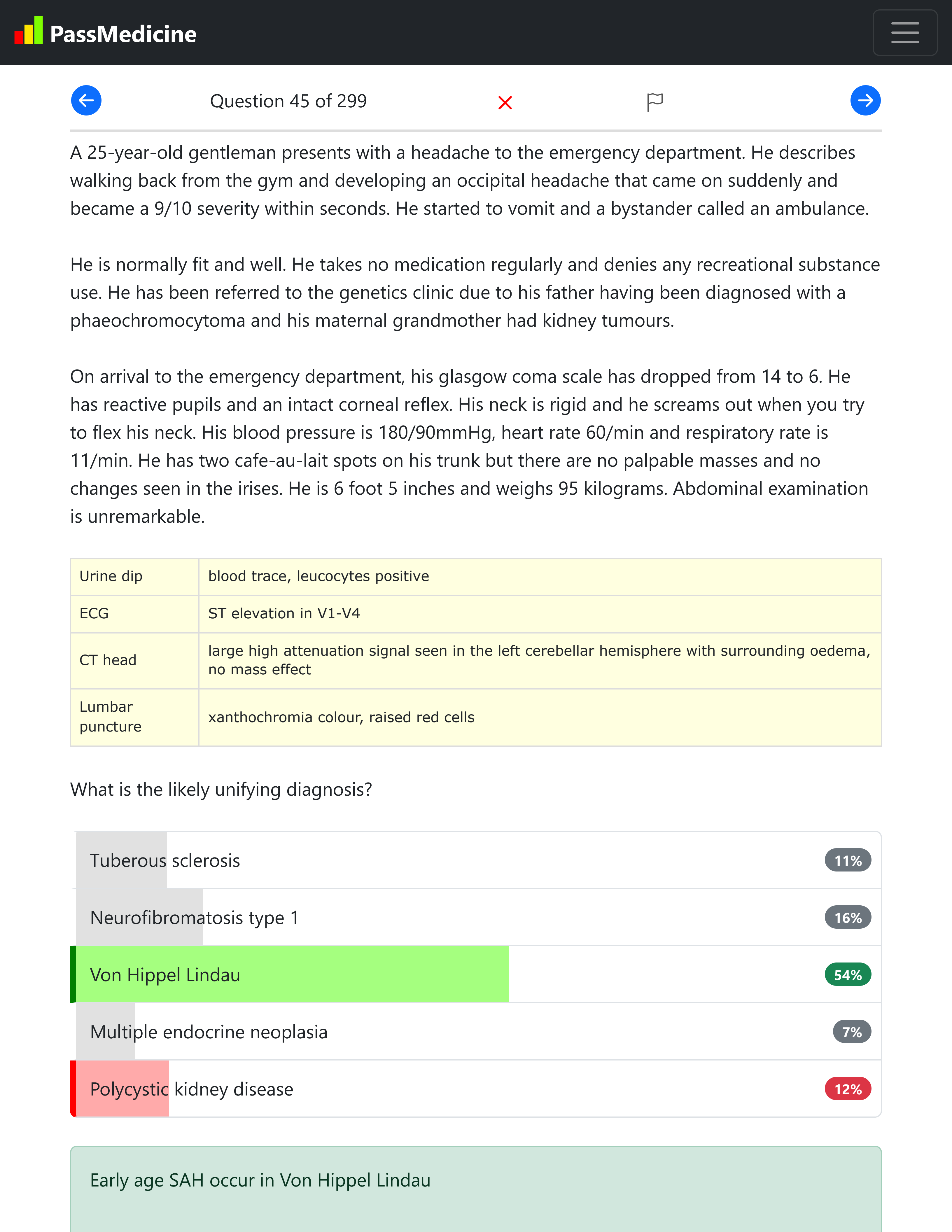

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CT head	large high attenuation signal seen in the left cerebellar hemisphere with surrounding oedema, no mass effect
Lumbar puncture	xanthochromia colour, raised red cells

What is the likely unifying diagnosis?

Tuberous sclerosis	11%
Neurofibromatosis type 1	16%
Von Hippel Lindau	54%
Multiple endocrine neoplasia	7%
Polycystic kidney disease	12%

Early age SAH occur in Von Hippel Lindau

Important for me Less important

This gentleman presents with a subarachnoid haemorrhage at a young age which is likely aneurysmal. He has a family history notable for renal and adrenal malignancy and there is a suspected genetic condition present in the family. Of the above, PKD and VHL are associated with subarachnoid haemorrhage and so these should stand out as possible causes. Polycystic kidney disease usually has palpable bilateral enlarged kidneys or gross haematuria. Therefore, the answer is Von Hippel Lindau syndrome where c3p25 mutation causes 80% to develop cerebellar haemangiomas or haemangioblastomas which can bleed acutely.

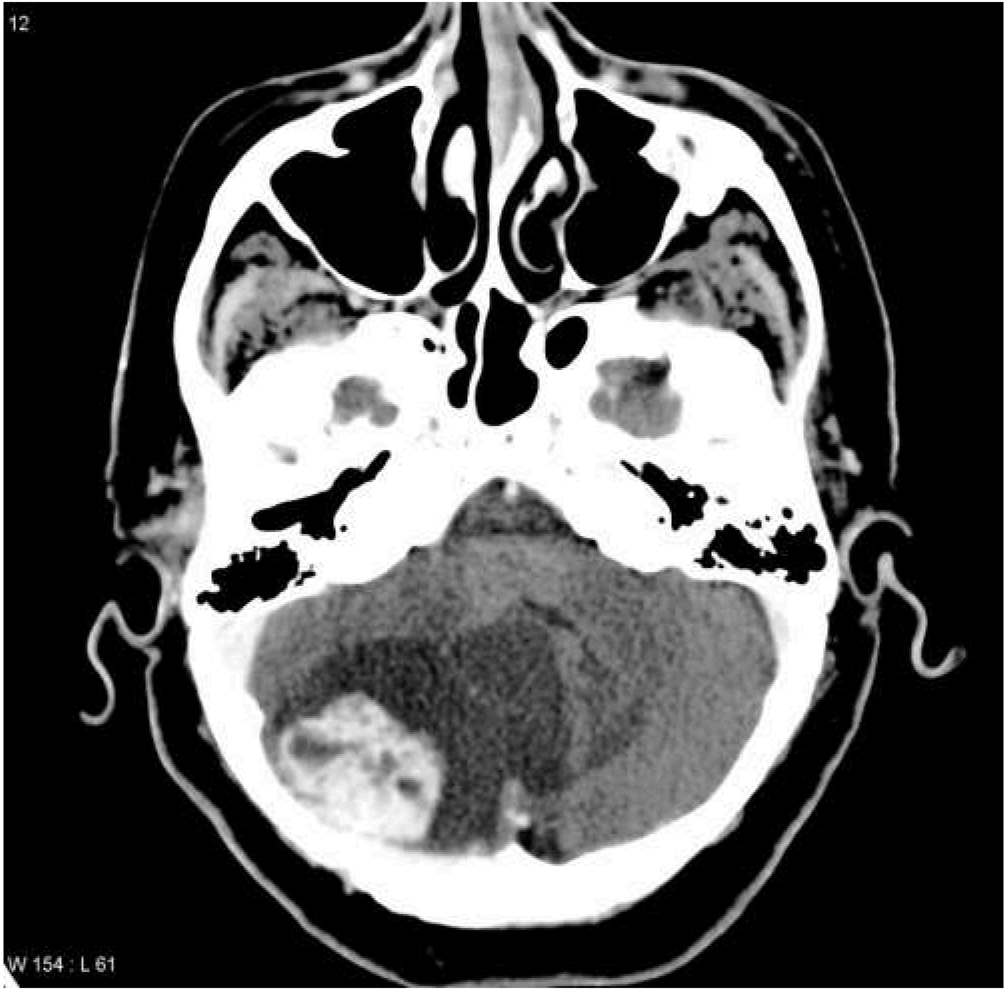
👍 👎 Discuss (9) Improve

Next question ›

Von Hippel-Lindau syndrome ★

Von Hippel-Lindau (VHL) syndrome is an autosomal dominant condition predisposing to neoplasia. It is due to an abnormality in the VHL gene located on short arm of chromosome 3

Features
- cerebellar haemangiomas: these can cause subarachnoid haemorrhages
- retinal haemangiomas: vitreous haemorrhage
- renal cysts (premalignant)
- phaeochromocytoma
- extra-renal cysts: epididymal, pancreatic, hepatic
- endolymphatic sac tumours
- clear-cell renal cell carcinoma



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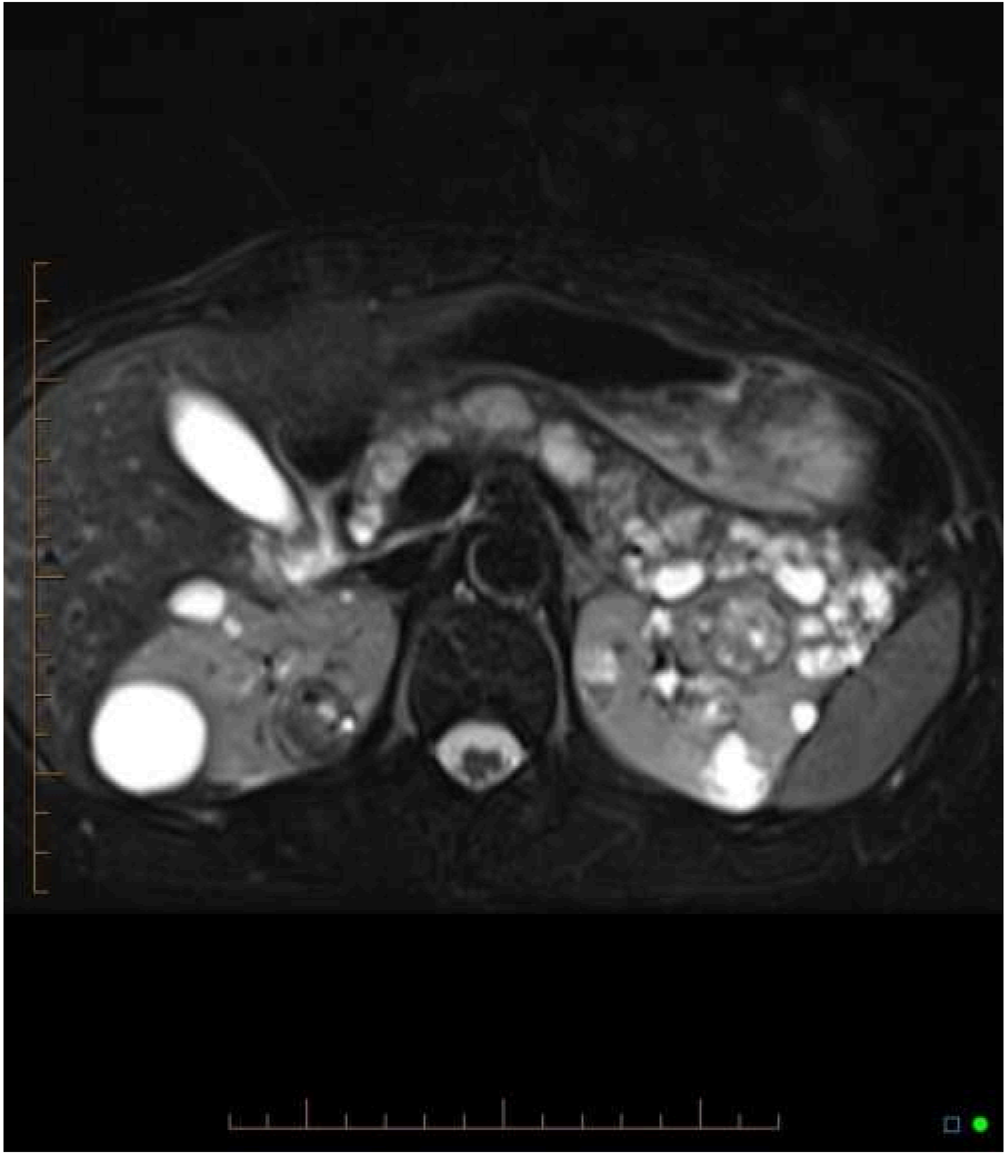
CT scan showing a cerebellar haemangioma in a patient with Von Hippel-Lindau syndrome.



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MRI showing renal cysts in patient with known Von Hippel-Lindau syndrome.

123

Next question >

Textbooks

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Score: **17.6%**

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An 87-year-old lady presents to the emergency department with unilateral weakness and according to her family difficulty in naming objects. This started about six hours ago.

Examination was consistent with weakness in left arm and leg (arm > leg) with decreased sensation. You also notice that she becomes increasingly tearful and has difficulty in naming objects around her, although she is able to describe their function. A systems review indicates a Heart Rate of 72/min and a blood pressure of 110/84 mmHg. There are no precordial murmurs and no carotid bruits. An ECG is performed showing that the patient is in sinus rhythm.

An urgent CT reveals an area of grey matter differentiation in the right hemisphere. Therefore a diagnosis of Right Partial Anterior Circulation Syndrome (R PACS) is made.

She has previously been on amlodipine 5 mg and mirtazapine 30 mg once per day.

She is started on aspirin. Which other intervention would you consider regarding her treatment.

- ○ Atorvastatin 80 mg OD to be started 48 hours post onset of symptoms ×
- ○ Amlodipine to be raised to 10 mg ×
- ○ Anti-embolism stockings to be prescribed ×
- ○ Bisoprolol 1.25 mg OD to be initiated ×
- ○ Atorvastatin 40 mg OD to be started now ×

Submit answer

Reference ranges

Score: **16%**

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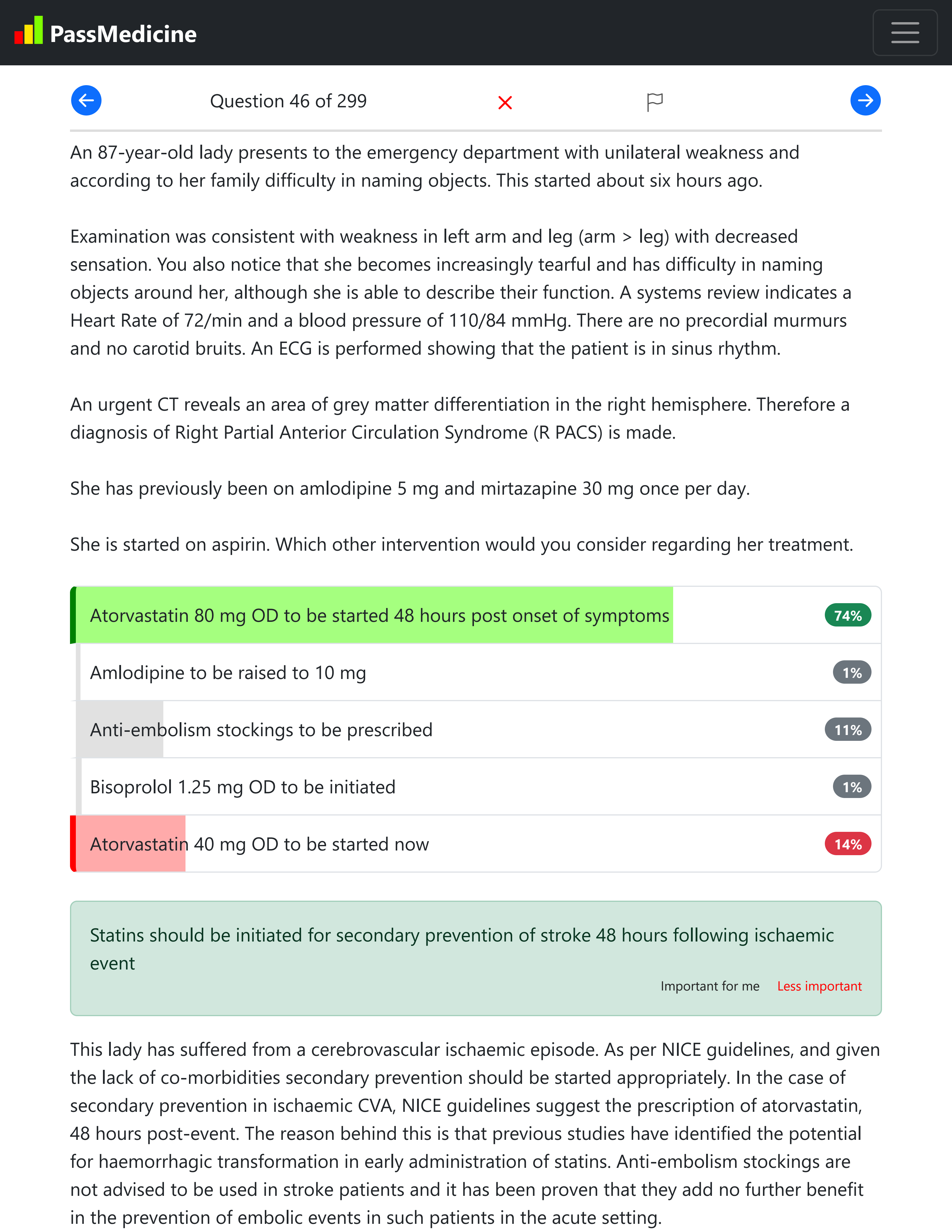

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She has previously been on amlodipine 5 mg and mirtazapine 30 mg once per day.

She is started on aspirin. Which other intervention would you consider regarding her treatment.

Atorvastatin 80 mg OD to be started 48 hours post onset of symptoms	74%
Amlodipine to be raised to 10 mg	1%
Anti-embolism stockings to be prescribed	11%
Bisoprolol 1.25 mg OD to be initiated	1%
Atorvastatin 40 mg OD to be started now	14%

Statins should be initiated for secondary prevention of stroke 48 hours following ischaemic event

Important for me Less important

This lady has suffered from a cerebrovascular ischaemic episode. As per NICE guidelines, and given the lack of co-morbidities secondary prevention should be started appropriately. In the case of secondary prevention in ischaemic CVA, NICE guidelines suggest the prescription of atorvastatin, 48 hours post-event. The reason behind this is that previous studies have identified the potential for haemorrhagic transformation in early administration of statins. Anti-embolism stockings are not advised to be used in stroke patients and it has been proven that they add no further benefit in the prevention of embolic events in such patients in the acute setting.

Stroke: management of acute ischaemic stroke

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
    - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
    - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:

- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.

- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
    - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
    - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

123

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Links

Royal College of Physicians 👍 11 👎 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 16

Stroke and TIA guidelines

Age and Ageing 👍 21 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

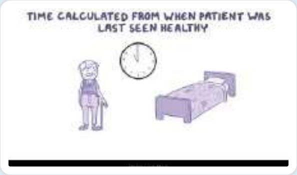
2023 National Clinical Guideline for Stroke

NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia
Acute Ischaemic Stroke

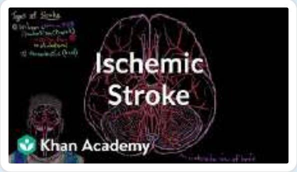
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

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Report broken media

Score: **17.6%**

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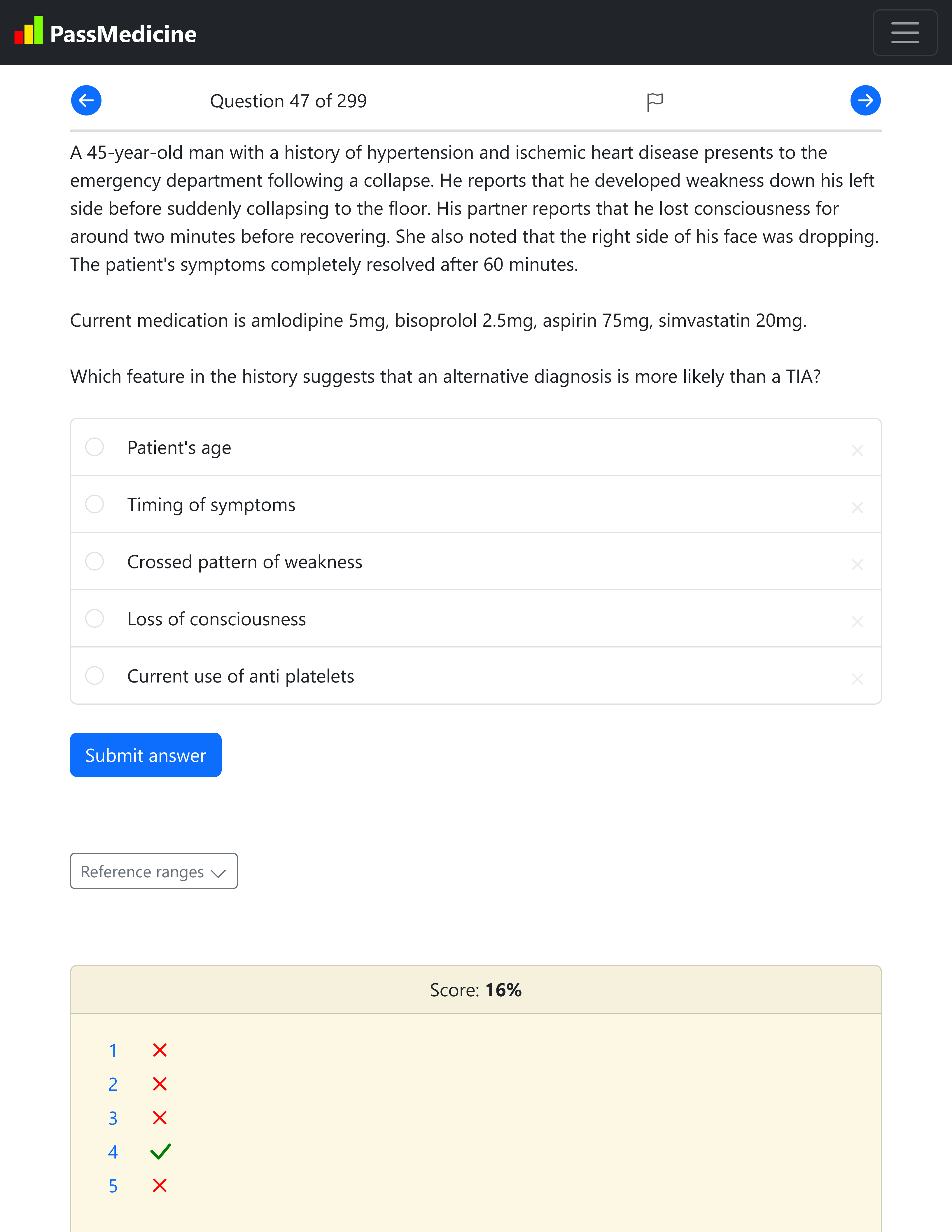

Question 47 of 299

A 45-year-old man with a history of hypertension and ischemic heart disease presents to the emergency department following a collapse. He reports that he developed weakness down his left side before suddenly collapsing to the floor. His partner reports that he lost consciousness for around two minutes before recovering. She also noted that the right side of his face was dropping. The patient's symptoms completely resolved after 60 minutes.

Current medication is amlodipine 5mg, bisoprolol 2.5mg, aspirin 75mg, simvastatin 20mg.

Which feature in the history suggests that an alternative diagnosis is more likely than a TIA?

- ○ Patient's age
- ○ Timing of symptoms
- ○ Crossed pattern of weakness
- ○ Loss of consciousness
- ○ Current use of anti platelets

Score: **16%**

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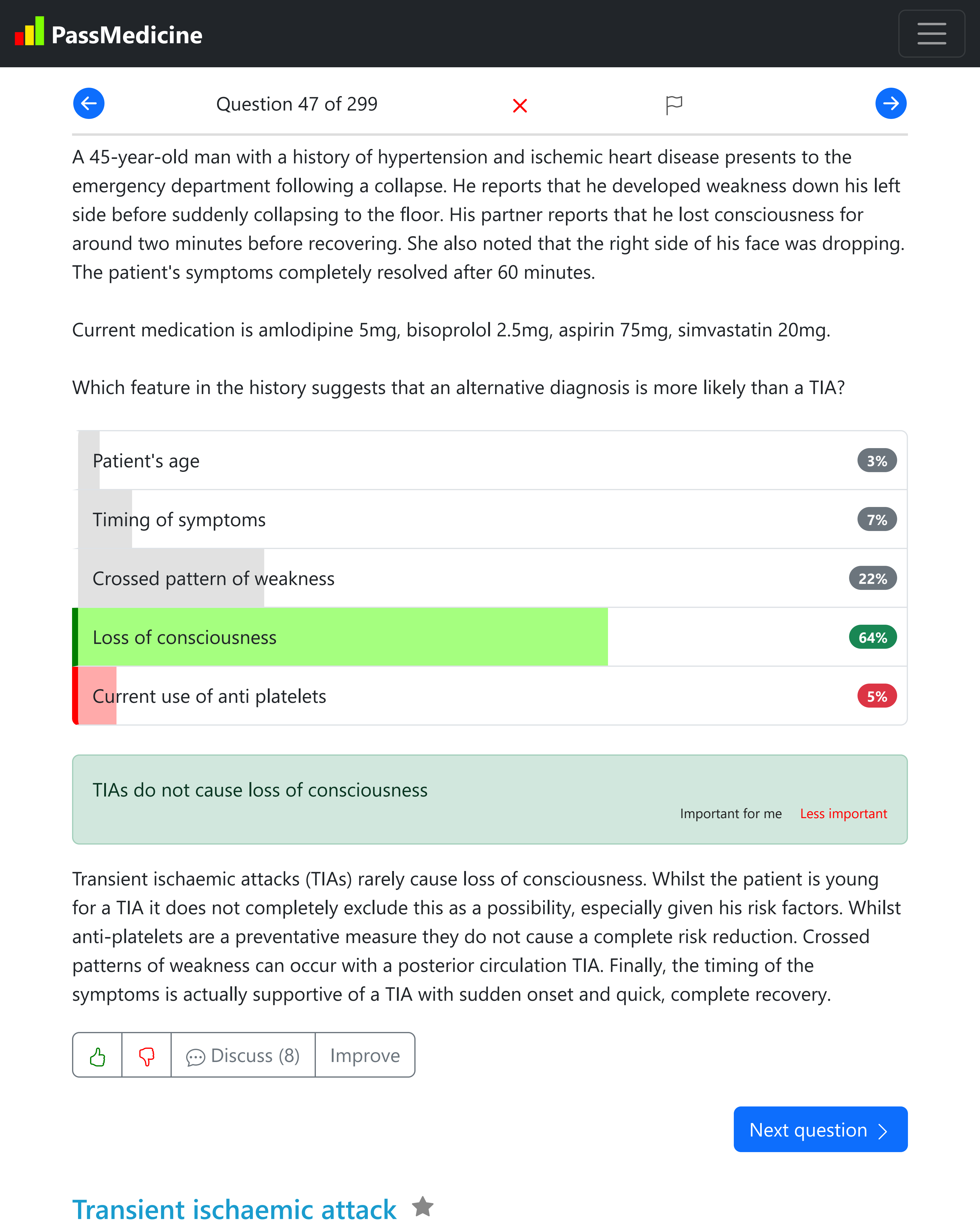

Question 47 of 299

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Current medication is amlodipine 5mg, bisoprolol 2.5mg, aspirin 75mg, simvastatin 20mg.

Which feature in the history suggests that an alternative diagnosis is more likely than a TIA?

Patient's age	3%
Timing of symptoms	7%
Crossed pattern of weakness	22%
Loss of consciousness	64%
Current use of anti platelets	5%

TIAs do not cause loss of consciousness

Transient ischaemic attacks (TIAs) rarely cause loss of consciousness. Whilst the patient is young for a TIA it does not completely exclude this as a possibility, especially given his risk factors. Whilst anti-platelets are a preventative measure they do not cause a complete risk reduction. Crossed patterns of weakness can occur with a posterior circulation TIA. Finally, the timing of the symptoms is actually supportive of a TIA with sudden onset and quick, complete recovery.

Transient ischaemic attack

A transient ischaemic attack (TIA), as the name suggests, is a brief period of neurological deficit due to a vascular cause, typically lasting less than an hour. The National Clinical Guideline for Stroke was published in 2023 and made a number of updated recommendations, including the use of dual antiplatelet therapy (DAPT). It should be noted that DAPT is not currently recommended for 'major' ischaemic stroke as the risk of haemorrhagic transformation is too high.

The original definition of a TIA was time-based: a sudden onset of a focal neurologic symptom and/or sign lasting less than 24 hours, brought on by a transient decrease in blood flow. However, this has now changed as it is recognised that even short periods of ischaemia can result in pathological changes to the brain. Therefore, a new '**tissue-based**' definition is now used: a transient episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischaemia, without acute infarction. REF

Patients often use the term 'mini-stroke' for TIAs.

Clinical features

The clinical features are similar to those of a stroke, i.e. sudden onset, focal neurological deficit but, rather than persisting, the features resolve, typically within 1 hour.

Possible features include
- unilateral weakness or sensory loss.
- aphasia or dysarthria
- ataxia, vertigo, or loss of balance
- visual problems
    - sudden transient loss of vision in one eye (amaurosis fugax)
    - diplopia
    - homonymous hemianopia

Assessment and referral

Patients with acute focal neurological symptoms that resolve completely within 24 hours of onset (i.e. suspected TIA) should:
- be given aspirin 300 mg immediately unless contraindicated
- assessed urgently within 24 hours by a stroke specialist clinician

Examples of TIA mimics that require exclusion
- hypoglycaemia
- intracranial haemorrhage

    - all patients on anticoagulants or with similar risk factors should be admitted for urgent imaging to exclude haemorrhage

If a patient presents more than 7 days ago they should be seen by a stroke specialist clinician as soon as possible within 7 days.

The ABCD2 prognostic score has previously been used to risk stratify patients who present with a suspected TIA. However, data from studies have suggested it performs poorly and it is therefore no longer recommended.

Patients with suspected TIA should be assessed by a stroke specialist clinician before a decision on brain imaging NICE

- NICE recommend that CT brains should not be done '*unless there is clinical suspicion of an alternative diagnosis that CT could detect*'
    - an example exception would be when there is a concern about haemorrhage as the patient is taking anticoagulants
- MRI (including diffusion-weighted and blood-sensitive sequences) is preferred to determine the territory of ischaemia, or to detect haemorrhage or alternative pathologies
    - it should be done on the same day as the specialist assessment if possible

Management of TIA

It should be remembered that patients who've had a TIA are at high risk of further vascular events, particularly in the first few days. This section covers management following a diagnosis, i.e. after being seen by a stroke specialist clinician.

Medication

Immediate antithrombotic therapy: REF

- patients with TIA or minor ischaemic stroke should be given antiplatelet therapy provided there is neither a contraindication nor a high risk of bleeding
- for patients within 24 hours of onset of TIA or minor ischaemic stroke and with a low risk of bleeding, the following DAPT regimes should be considered:
    - clopidogrel (initial dose 300 mg followed by 75 mg od) + aspirin (initial dose 300 mg followed by 75 mg od for 21 days) followed by monotherapy with clopidogrel 75 mg od
    - ticagrelor + clopidogrel is an alternative
- if not appropriate for DAPT:
    - clopidogrel 300 mg loading dose followed by 75 mg od should be given
- proton pump inhibitor therapy should be considered for DAPT

The different antithrombotic recommendations may seem confusing - it is helpful to think of 3 different phases for a typical patient:

Resolved TIA symptoms, awaiting specialist review within 24 hours	Reviewed by specialist, initial 21 days when at high risk of further events	Long-term secondary prevention after 21 days
Aspirin	Aspirin + Clopidogrel	Clopidogrel

If a patient has atrial fibrillation they should be anticoagulated as soon as intracranial haemorrhage has been excluded.

Lipid modification
- high-intensity statin (such as atorvastatin 20-80 mg daily) - the aim of statin therapy is to reduce non-HDL cholesterol by more than 40%

Further investigation

Carotid imaging
- atherosclerosis in the carotid artery may be a source of emboli in some patients
- patients who are considered candidates for carotid intervention should have carotid imaging performed within 24 hours of assessment
    - carotid duplex ultrasound or either CT angiography or MR angiography
- carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled NICE
    - should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

+ it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%
- it should be performed as soon as possible within 7 days

AI + Q 123

Next question ›

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Links

RCP 👍 13 👎 13

2016 Stroke guidelines

National Clinical Guideline for Stroke 👍 7 👎 3

2023 National Clinical Guideline for Stroke

NICE 👍 17 👎 6

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Clinical Knowledge Summaries 👍 8 👎 3

Transient ischaemic attack

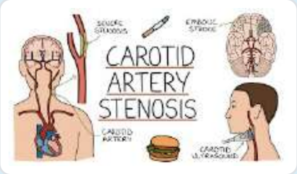
Radiopaedia 👍 7 👎 1

Transient ischaemic attack

Suggest link

Report broken link

Media



Understanding Carotid Artery Stenosis

Zero To Finals - YouTube 👍 2 👎 1

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A 32-year-old woman is seen in the neurology clinic with a 6-month history of intermittent headaches. The headaches typically affect her left side and are associated with paraesthesia in her left hand and occasional nausea and vomiting. She says that her symptoms resolve after a period of 4-6 hours each time. She has no past medical history of note and has no regular medications or allergies.

On examination, her observations are recorded within normal limits. There is equal power in all 4 limbs with no sensory or coordination disturbance. Her cranial nerve examination is unremarkable.

What is the most appropriate next step in the long-term management of this patient?

○ Amitriptyline

○ Aspirin

○ Propranolol

○ Sumatriptan

○ Topiramate

Submit answer

Reference ranges

Score: **16%**

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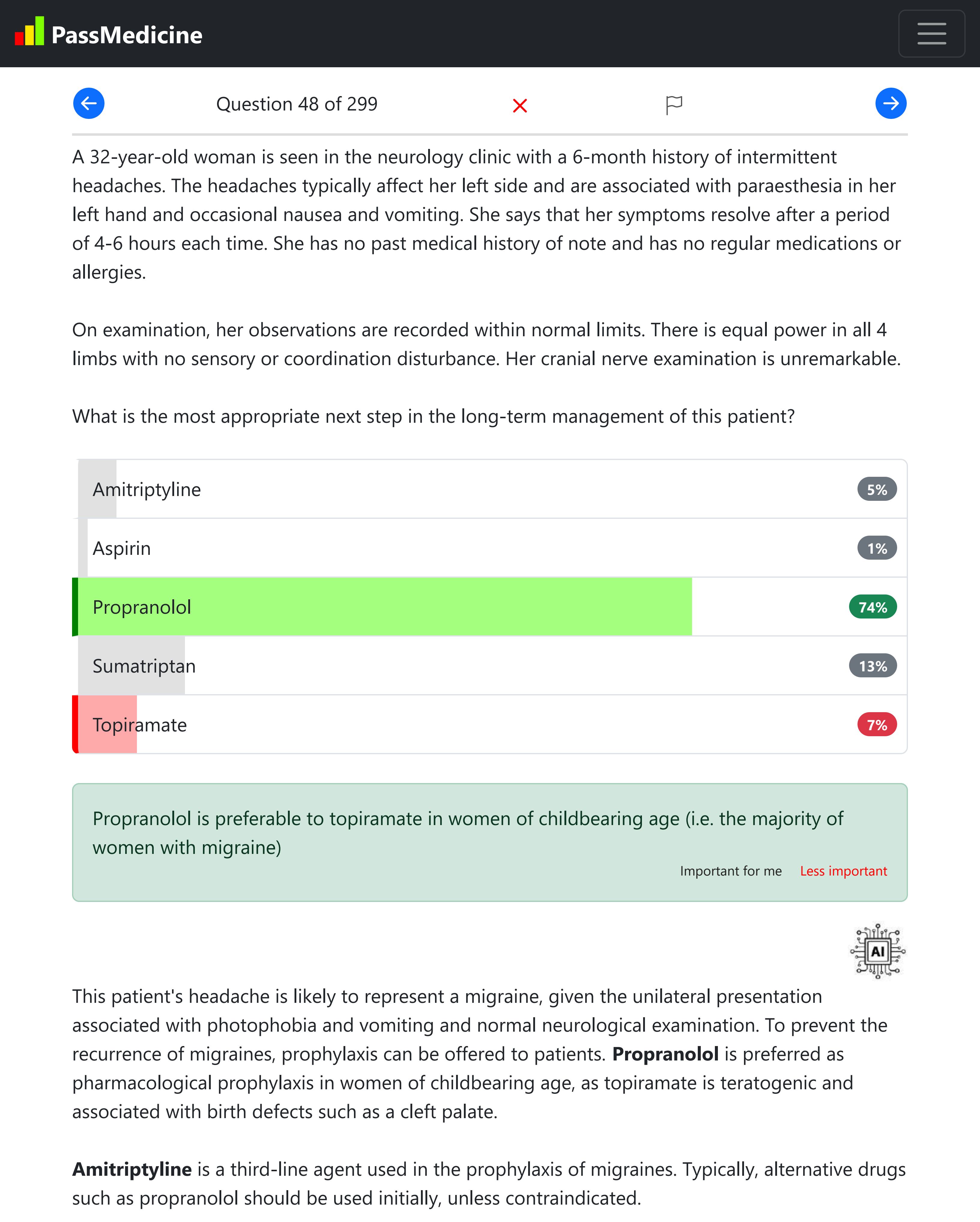
Back to top



Question 48 of 299

A 32-year-old woman is seen in the neurology clinic with a 6-month history of intermittent headaches. The headaches typically affect her left side and are associated with paraesthesia in her left hand and occasional nausea and vomiting. She says that her symptoms resolve after a period of 4-6 hours each time. She has no past medical history of note and has no regular medications or allergies.

On examination, her observations are recorded within normal limits. There is equal power in all 4 limbs with no sensory or coordination disturbance. Her cranial nerve examination is unremarkable.

What is the most appropriate next step in the long-term management of this patient?

Amitriptyline	5%
Aspirin	1%
Propranolol	74%
Sumatriptan	13%
Topiramate	7%

Propranolol is preferable to topiramate in women of childbearing age (i.e. the majority of women with migraine)

Important for me   Less important

This patient's headache is likely to represent a migraine, given the unilateral presentation associated with photophobia and vomiting and normal neurological examination. To prevent the recurrence of migraines, prophylaxis can be offered to patients. **Propranolol** is preferred as pharmacological prophylaxis in women of childbearing age, as topiramate is teratogenic and associated with birth defects such as a cleft palate.

Amitriptyline is a third-line agent used in the prophylaxis of migraines. Typically, alternative drugs such as propranolol should be used initially, unless contraindicated.

Aspirin is used in the acute treatment of migraines. However, it plays no role in the prophylaxis of migraine.

Sumatriptan is another medication used in the acute treatment of migraines. However, long-term sumatriptan is not used to prevent the recurrence of migraines.

Topiramate is used in the prophylaxis of migraines. However, as this patient is a woman of childbearing age, it is inappropriate to initiate topiramate for long-term use as it is teratogenic and associated with birth defects.

Discuss (2) Improve

Next question

Migraine: management

It should be noted that as a general rule 5-HT receptor agonists are used in the acute treatment of migraine whilst 5-HT receptor antagonists are used in prophylaxis. NICE produced guidelines in 2012 on the management of headache, including migraines.

Acute treatment

- first-line: offer combination therapy with
  - an oral triptan and an NSAID, or
  - an oral triptan and paracetamol
- for young people aged 12-17 years consider a nasal triptan in preference to an oral triptan
- if the above measures are not effective or not tolerated offer a non-oral preparation of metoclopramide or prochlorperazine and consider adding a non-oral NSAID or triptan
  - caution should be exercised when prescribing metoclopramide to young patients as acute dystonic reactions may develop

Prophylaxis

- prophylaxis should generally be given if '*Migraine attacks are having a significant impact on quality of life and daily function, for example they occur frequently (more than once a week on average) or are prolonged and severe despite optimal acute treatment*'
- options:
  - propranolol
  - topiramate: should be avoided in women of childbearing age as it may be teratogenic and it can reduce the effectiveness of hormonal contraceptives
  - amitriptyline

- if these measures fail NICE recommends 'a course of up to 10 sessions of acupuncture over 5-8 weeks'
- NICE recommend: '*Advise people with migraine that riboflavin (400 mg once a day) may be effective in reducing migraine frequency and intensity for some people*'
- for women with predictable menstrual migraine treatment NICE recommend either frovatriptan (2.5 mg twice a day) or zolmitriptan (2.5 mg twice or three times a day) as a type of 'mini-prophylaxis'
- treatment options that may be considered by specialists, but fall outside the NICE guidelines:
    - candesartan: recommended by the British Association for the Study of Headache guidelines
    - monoclonal antibodies directed against the calcitonin gene-related peptide (CGRP) receptor: examples include erenumab
- pizotifen is no longer recommended. Adverse effects such as weight gain & drowsiness are common

AI + Q 123

Next question >

B *I*

Textbooks

High-yield textbook

Extended textbook

Links

British Association for the Study of Headache 14 21

Headache guidelines

Clinical Knowledge Summaries 14 7

Migraine guidelines

NICE 👍 16 👎 4

2012 Headache guidelines

Suggest link Report broken link

Media

Management of Migraine
Podmedics

Migraine

Podmedics - YouTube 👍 1 👎 1

Report broken media

Score: **17.6%**

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Question 49 of 299

A 60-year-old woman was referred with dizziness, nausea, and vomiting. This had come on insidiously the previous day. She described the room spinning, which was there constantly but worse with any sudden movement. She had suffered flu-like symptoms in the previous week, which were now resolving. On examination, she had horizontal nystagmus with the fast beat towards the right. Speech was normal. There was no past-pointing and finger-nose testing was normal. Gait was normal. Power was 5/5 throughout all limbs. Her hearing was normal.

What is the most likely diagnosis?

- ○ Benign paroxysmal positional vertigo
- ○ Meniere's disease
- ○ Vestibular neuronitis
- ○ Transient ischaemic attack
- ○ Vertebrobasilar insufficiency

Submit answer

Reference ranges

Score: **16%**

1 ✗
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A 60-year-old woman was referred with dizziness, nausea, and vomiting. This had come on insidiously the previous day. She described the room spinning, which was there constantly but worse with any sudden movement. She had suffered flu-like symptoms in the previous week, which were now resolving. On examination, she had horizontal nystagmus with the fast beat towards the right. Speech was normal. There was no past-pointing and finger-nose testing was normal. Gait was normal. Power was 5/5 throughout all limbs. Her hearing was normal.

What is the most likely diagnosis?

Benign paroxysmal positional vertigo	20%
Meniere's disease	4%
Vestibular neuronitis	75%
Transient ischaemic attack	1%
Vertebrobasilar insufficiency	2%

Prodromal viral illness with associated vertigo and nystagmus is suggestive of vestibular neuronitis

Important for me   Less important

The presence of horizontal nystagmus in the absence of any other neurological signs suggests a vestibular origin of the acute symptoms in this case. A posterior circulation vascular event would be likely to be associated with other signs of cerebellar dysfunction or other cranial nerve palsies. The preceding probable viral illness and the persistence of the symptoms suggest a viral vestibular neuronitis as the cause of her vertigo rather than benign paroxysmal positional vertigo.
There are also none of the additional features that would suggest Meniere's disease such as unilateral deafness, severe nausea, and a pressure sensation in the ear.

Discuss (6)   Improve

Next question

Vestibular neuronitis ★

Vestibular neuronitis is a cause of vertigo that often develops following a viral infection.

Features
- recurrent vertigo attacks lasting hours or days
- nausea and vomiting may be present
- horizontal nystagmus is usually present
- no hearing loss or tinnitus

Differential diagnosis
- viral labyrinthitis
- posterior circulation stroke: the HiNTs exam can be used to distinguish vestibular neuronitis from posterior circulation stroke

Management
- buccal or intramuscular prochlorperazine is often used to provide rapid relief for severe cases
- a short oral course of prochlorperazine, or an antihistamine (cinnarizine, cyclizine, or promethazine) may be used to alleviate less severe cases
- vestibular rehabilitation exercises are the preferred treatment for patients who experience chronic symptoms

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

North Lincolnshire and Goole NHS 👍 9 👎 23

Example of vestibular rehabilitation exercises

Clinical Knowledge Summaries 👍 16 👎 9

Vestibular neuronitis

Suggest link

Report broken link

Media

Normal HIT (worrisome)

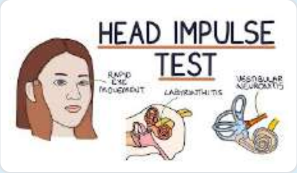
The HINTS exam in vertigo

Individual - YouTube 👍 19 👎 0

VESTIBULAR
NEURONITIS

Understanding Vestibular Neuronitis

Zero To Finals - YouTube 👍 0 👎 0


HEAD IMPULSE
TEST

The Head Impulse Test

Zero To Finals - YouTube 👍 0 👎 0

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A 53-year-old man presents to the emergency department with a 1-day history of bilateral leg weakness. He is now struggling to stand up from a chair. Whilst waiting to be seen in the department, the patient starts to experience finger paraesthesia.

The patient reports no medical history of note and is in good health aside from a recent bout of diarrhoea. He takes no regular medications, smokes 20 cigarettes per day and drinks up to 4 pints of beer 'most nights'. He works as a plumber.

On clinical examination, there is a marked weakness of the lower limbs, absent reflexes at the ankle, knee, and elbow with a grossly normal sensory assessment. He is dysarthric and coughs significantly on a bedside swallow assessment.

An MRI whole spine is ordered and reported as normal. Blood tests show a raised ALT, but no other abnormalities.

Given the likely diagnosis, assuming he receives standard treatment, what is the probability of long term weakness?

○ 30%

○ 40%

○ 50%

○ 1%

○ 15%

Submit answer

Reference ranges

Score: **16%**

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Back to top

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An MRI whole spine is ordered and reported as normal. Blood tests show a raised ALT, but no other abnormalities.

Given the likely diagnosis, assuming he receives standard treatment, what is the probability of long term weakness?

30%	22%
40%	7%
50%	8%
1%	8%
15%	56%

Up to 15 percent of individuals with Guillain-Barre syndrome will experience long-term weakness

Important for me   Less important

This patient presents with classical Guillain-Barre syndrome(GBS). Early recognition and treatment with intravenous immunoglobulin and/or plasma exchange both increase the speed of recovery and are important in reducing the incidence of long term sequelae.

The most common long term sequelae of GBS is fatigue, although, depending on the clinical course, pain and persistent neurological deficits may also be problematic, with long term neurological difficulties reported in 7-15% of patients.

Discuss (6) | Improve

Next question ›

Guillain-Barre syndrome: management ★

Guillain-Barre syndrome describes an immune-mediated demyelination of the peripheral nervous system often triggered by an infection (classically *Campylobacter jejuni*).

Management

- IV immunoglobulins (IVIG) or plasma exchange can be used
    - IVIG is as effective as plasma exchange better tolerated and easier to administer
    - therefore IVIG is generally used first-line for Guillain-Barre syndrome where patients require immunomodulatory therapy
    - no benefit in combining both treatments
- steroids and immunosuppressants have not been shown to be beneficial
- FVC regularly to monitor respiratory function

Prognosis

- severe motor problems persist in about 15%
- around 5% die

+ Q 123

Next question ›

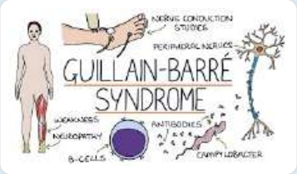
B *I*

Textbooks

High-yield textbook

Extended textbook

Media


NERVE CONDUCTION STUDIES
PERIPHERAL NERVES
GUILLAIN-BARRÉ SYNDROME
WEAKNESS
NEUROPATHY
ANTIBODIES
B-CELLS
CAMPYLOBACTER

Understanding Guillain-Barre Syndrome

Zero To Finals - YouTube 👍 3 👎 1

SYMPTOMS - BASED on NERVES AFFECTED
PARESTHESIA
CRANIAL NERVES INVOLVED
MUSCLE WEAKNESS & ABSENCE of REFLEXES
AUTONOMIC NERVES → VARIETY of SYMPTOMS

Guillain-Barre syndrome

Osmosis - YouTube 👍 1 👎 1

Report broken media

Score: **17.6%**

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A 27-year-old caucasian woman is 28 weeks pregnant. She has been epileptic since the age of 7 and takes lamotrigine, which she has continued throughout the pregnancy. She has not had a seizure for 2 years. What must you consider in pregnant patients taking lamotrigine?

- Serum lamotrigine levels fall in the second trimester 29%
- She must not breastfeed while taking lamotrigine 12%
- She should be advised to stop lamotrigine 4%
- Folic acid 400mcg should be taken throughout pregnancy 38%
- It is protein bound and therefore drug levels increase in pregnancy 17%

Due to the increased plasma volume and enhanced renal/hepatic drug clearance, lamotrigine levels decrease in the second trimester of pregnancy. It may be necessary to increase the dose of lamotrigine at this time (normally increased two or three fold). A baseline serum drug level is useful to establish compliance and inform future changes in drug doses.

Breastfeeding is not contraindicated in women taking lamotrigine. However, the infant should be monitored for signs of accumulation such as a rash or drowsiness.

Patients should not stop their anti-epileptic medications unless a joint decision has been made in conjunction with an obstetrician/obstetric physician doctor. If a patient has been seizure-free for >2 years and is on dual therapy then it may be beneficial to reduce/stop one of the medications.

Patients on anti-epileptic medications should take folic acid 5mg throughout pregnancy.

Lamotrigine has low levels of protein binding and therefore is not affected by the decreased serum protein levels in pregnancy.

Further reading:
RCOG: Epilepsy in Pregnancy (Green-top Guideline No.68)
https://www.rcog.org.uk/en/guidelines-research-services/guidelines/gtg68/

Discuss (4) Improve

Next question ›

Epilepsy: pregnancy and breast feeding ★

The risks of uncontrolled epilepsy during pregnancy generally outweigh the risks of medication to the fetus. All women thinking about becoming pregnant should be advised to take folic acid 5mg per day well before pregnancy to minimise the risk of neural tube defects. Around 1-2% of newborns born to non-epileptic mothers have congenital defects. This rises to 3-4% if the mother takes antiepileptic medication.

Other points
- aim for monotherapy
- there is no indication to monitor antiepileptic drug levels
- sodium valproate: associated with neural tube defects
- carbamazepine: often considered the least teratogenic of the older antiepileptics
- phenytoin: associated with cleft palate
- lamotrigine: studies to date suggest the rate of congenital malformations may be low. The dose of lamotrigine may need to be increased in pregnancy

Breast feeding is generally considered safe for mothers taking antiepileptics with the possible exception of the barbiturates

It is advised that pregnant women taking phenytoin are given vitamin K in the last month of pregnancy to prevent clotting disorders in the newborn

Sodium valproate

The November 2013 issue of the Drug Safety Update also carried a warning about new evidence showing a significant risk of neurodevelopmental delay in children following maternal use of sodium valproate.

The update concludes that sodium valproate should not be used during pregnancy and in women of childbearing age unless clearly necessary. Women of childbearing age should not start treatment without specialist neurological or psychiatric advice.

+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

SIGN 👍 8 👎 7

2018 Diagnosis and management of epilepsy in adults

NICE 👍 9 👎 4

2022 Epilepsy guidelines

Royal College of Obstetricians and Gynaecologists 👍 8 👎 2

2016 Epilepsy in Pregnancy

Suggest link

Report broken link

Score: **17.6%**

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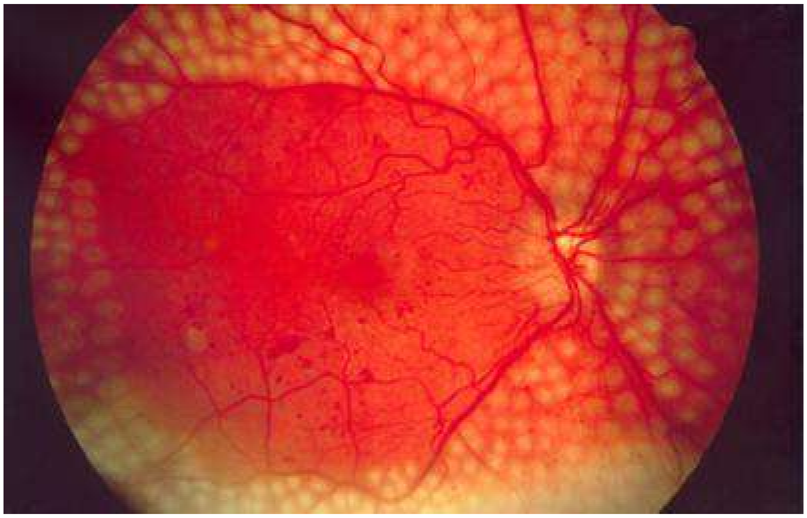
Back to top

Question 52 of 299

A 52-year-old man presents for a routine eye screening appointment. He has a past medical history of hypertension, type 2 diabetes mellitus and hyperlipidaemia. The eyes are examined using an ophthalmoscope and the following view is seen of the left eye:



What is the most common complication of the treatment that this patient has received?

- ○ Chronic eye pain
- ○ Development of floaters
- ○ Development of infection
- ○ Reduced central vision
- ○ Reduced night vision

Submit answer

Reference ranges

Score: **17.6%**

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52	-

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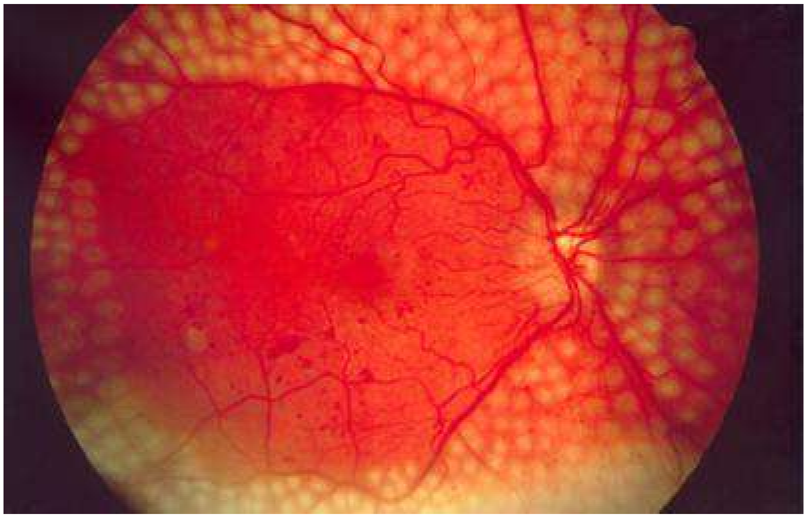
Question 52 of 299 ✓

A 52-year-old man presents for a routine eye screening appointment. He has a past medical history of hypertension, type 2 diabetes mellitus and hyperlipidaemia. The eyes are examined using an ophthalmoscope and the following view is seen of the left eye:



What is the most common complication of the treatment that this patient has received?

Chronic eye pain	2%
Development of floaters	20%
Development of infection	2%
Reduced central vision	20%
Reduced night vision	56%

The photo shows clear evidence of diabetic retinopathy - proliferation of small vessels is present, as well as haemorrhage. Importantly, this photo also shows evidence of panretinal laser photocoagulation (PRP) - the numerous circular yellow spots around the periphery are 'burn scars' from the treatment. PRP aims to prevent further proliferation of vessels. Of the complications listed here, the most common adverse effect associated with PRP is a **reduction in night vision**. This is because PRP leads to scarring of peripheral retinal tissue, where rods are situated (rods being responsible for vision in low light). As such, the peripheral visual field is also often reduced.

Chronic eye pain is incorrect. Some temporary pain may arise following the procedure, but this improves within 24-48 hours.

Development of floaters is incorrect. Floaters develop due to the underlying condition, and due to macular oedema that may result from diabetic eye disease. PRP itself is not generally associated with causing further floaters.

Development of infection is incorrect. PRP is not associated with the development of infection.

Reduced central vision is incorrect. As PRP targets the periphery, the central vision is typically spared.

👍 👎 Discuss (1) Improve

Next question >

Diabetic retinopathy ★

Diabetic retinopathy is the most common cause of blindness in adults aged 35-65 years-old.

Pathophysiology

Hyperglycaemia is thought to cause increased retinal blood flow and abnormal metabolism in the retinal vessel walls. This precipitates damage to endothelial cells and pericytes

Endothelial dysfunction leads to increased vascular permeability which causes the characteristic exudates seen on fundoscopy. Pericyte dysfunction predisposes to the formation of microaneurysms. Neovasculization is thought to be caused by the production of growth factors in response to retinal ischaemia

Classification

Patients are typically classified into those with non-proliferative diabetic retinopathy (NPDR), proliferative retinopathy (PDR) and maculopathy.

Non-proliferative diabetic retinopathy

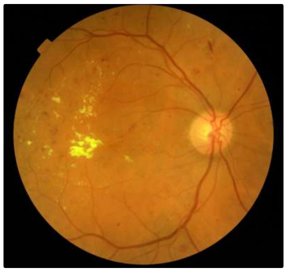
Mild NPDR
- 1 or more microaneurysm

Moderate NPDR

- microaneurysms
- blot haemorrhages
- hard exudates
- cotton wool spots ('soft exudates' - represent areas of retinal infarction), venous beading/looping and intraretinal microvascular abnormalities (IRMA) less severe than in severe NPDR

Severe NPDR

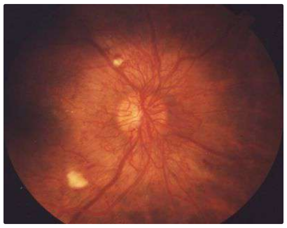
- blot haemorrhages and microaneurysms in 4 quadrants
- venous beading in at least 2 quadrants
- IRMA in at least 1 quadrant



Proliferative diabetic retinopathy

Key features

- retinal neovascularisation - may lead to vitrous haemorrhage
- fibrous tissue forming anterior to retinal disc
- more common in Type I DM, 50% blind in 5 years



Maculopathy

Key features

- based on location rather than severity, anything is potentially serious
- hard exudates and other 'background' changes on macula
- check visual acuity
- more common in Type II DM

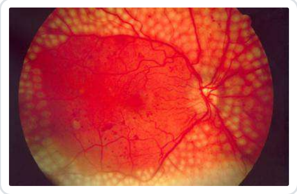
Management

All patients
- optimise glycaemic control, blood pressure and hyperlipidemia
- regular review by ophthalmology

Maculopathy
- if there is a change in visual acuity then intravitreal vascular endothelial growth factor (VEGF) inhibitors

Non-proliferative retinopathy
- regular observation
- if severe/very severe consider panretinal laser photocoagulation

Proliferative retinopathy
- panretinal laser photocoagulation
    - following treatment around 50% of patients develop a noticeable reduction in their visual fields due to the scarring of peripheral retinal tissue
    - other complications include a decrease in night vision (rods are predominantly responsible for vision in low light conditions, the majority of rod cells are located in the peripheral retina), a generalised decrease in visual acuity and macular oedema
- intravitreal VEGF inhibitors
    - often now used in combination with panretinal laser photocoagulation
    - examples include ranibizumab
    - strong evidence base suggests they both slow progression of proliferative diabetic retinopathy and improve visual acuity
- if severe or vitreous haemorrhage: vitreoretinal surgery



123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **20.5%**

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A 68-year-old man develops a left-sided hemiparesis and facial droop whilst out shopping with his wife at 14:35pm. His initial CT head scan is done at 15:40pm and reveals no intracranial haemorrhage or space occupying lesion. He is given alteplase in the emergency department at 15:50pm.

His post-thrombolysis CT head at 09:00am the following morning reveals a right middle cerebral artery infarction and no evidence of intracranial haemorrhage or haemorrhagic transformation. A 12 lead ECG done in the stroke unit shows atrial fibrillation of 110 beats per minute. He has a persisting dense left-sided weakness several hours post thrombolysis.

Which of the following drugs should be added next?

- ○ Clopidogrel
- ○ Aspirin
- ○ Warfarin
- ○ Unfractionated heparin
- ○ Rivaroxaban

Submit answer

Reference ranges

Score: **17.6%**

1 ✗
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3 ✗
4 ✓

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Question 53 of 299 ✗

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Which of the following drugs should be added next?

Clopidogrel	20%
Aspirin ✓	55%
Warfarin	3%
Unfractionated heparin	9%
Rivaroxaban ✗	13%

Aspirin 300mg orally for the initial two weeks should be commenced in patients with a disabling stroke and atrial fibrillation. Following this, these patients should be considered for anticoagulant treatment. The post-thrombolysis CT head revealed no acute intracranial haemorrhage or haemorrhagic transformation, therefore, it is safe to start aspirin.

Discuss (14) Improve

Next question ›

Stroke: management of acute ischaemic stroke

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
    - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
    - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:

- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.

- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
    - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
    - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.

- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6

hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 11 👎 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 16

Stroke and TIA guidelines

Age and Ageing 👍 21 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

2023 National Clinical Guideline for Stroke

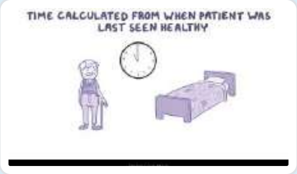
NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link

Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia
Acute Ischaemic Stroke

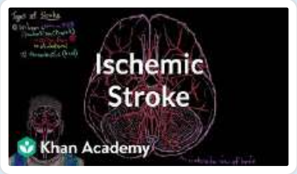
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

Khan Academy - YouTube 👍 1 👎 1

Report broken media

Score: **20.5%**

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A 67 year-old woman presents to hospital after a collapse at home. One week earlier she had seen her own doctor after feeling generally unwell with fever, myalgia, and coryza. These symptoms were attributed to a viral infection and have now resolved. However, for the past few days she has felt unsteady on her feet and has had to hold on to the furniture whilst walking around the house. For the last two days in particular she has also noticed that her vision has deteriorated, and she has been seeing double. Today she tried to hold onto the sofa whilst walking around the room, but misjudged the distance and lost her grip, causing her to fall to the floor. Her daughter who is also present adds that she feels her mothers speech is slurred compared to normal, and has been so for the past three days.

Her past medical history includes hypertension, hypothyroidism, type 2 diabetes, hyperlipidaemia, angina, and recurrent urinary tract infection. She takes ramipril, amlodipine, doxazosin, laevoythyroxine, metformin, gliclazide, simvastatin, atenolol, nicorandil, and nitrofurantoin. She does not smoke but admits to enjoying a glass of sherry on most nights. Usually she is independently mobile, without aids. She lives on her own and is self-caring.

On examination, there are some bruises on the left shoulder where she fell, but no suggestion of any fracture. Observations are normal and she is afebrile. Cardiovascular, respiratory, and abdominal examination is unremarkable. She is fully alert and oriented. Pupils are equal and reactive to light. Eye movements are grossly impaired in all directions. There is no facial asymmetry. Muscle power is normal in all limbs, but finger-nose pointing is impaired and she is unable to walk in a straight line. Reflexes are unobtainable. Sensation is normal.

What is the most likely diagnosis?

○ Wernickes encephalopathy

○ Viral cerebellitis

○ Myasthenia gravis

○ Miller Fisher syndrome

○ Posterior circulation stroke

Submit answer

Reference ranges

Score: **17.6%**

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Back to top

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What is the most likely diagnosis?

Wernickes encephalopathy	10%
Viral cerebellitis	22%
Myasthenia gravis	2%
Miller Fisher syndrome	55%
Posterior circulation stroke	12%

There has been a subacute deterioration in global cerebellar function, as suggested by gait ataxia, bilateral upper limb dysmetria, and dysarthria, progressing over a period of days. There is also complete ophthalmoplegia, and absence of reflexes. This combination of features occurring after a recent viral infection suggests a diagnosis of the Miller Fisher variant of Guillain-Barre syndrome. One might also expect to find descending weakness, which was not present in this case. Confirmation of the diagnosis is by lumbar puncture which demonstrates raised protein with normal cell count, and positive serum anti-GQ1b antibodies. Treatment is as for Guillain-Barre syndrome, with supportive care and intravenous immunoglobulin or plasmapheresis.

Wernickes encephalopathy presents with ataxia and ophthalmoplegia but also confusion, and typically occurs in malnourished alcoholics.

Viral cerebellitis is more common in children and would not explain the ophthalmoplegia and areflexia.

Myasthenia gravis would be unlikely to develop so rapidly, and though a cause of complex ophthalmoplegia, it would not explain the areflexia or the cerebellar signs.

Posterior circulation syndromes can cause acute cerebellar dysfunction but stroke of any sort is unlikely as this is a subacute presentation of neurological features not referable to one part of the brain.

👍 👎 Discuss (4) Improve

Next question >

Guillain-Barre syndrome ★

Guillain-Barre syndrome describes an immune-mediated demyelination of the peripheral nervous system often triggered by an infection (classically *Campylobacter jejuni*)

Pathogenesis
- cross-reaction of antibodies with gangliosides in the peripheral nervous system
- correlation between anti-ganglioside antibody (e.g. anti-GM1) and clinical features has been demonstrated
- anti-GM1 antibodies in 25% of patients

Miller Fisher syndrome
- variant of Guillain-Barre syndrome
- associated with ophthalmoplegia, areflexia and ataxia. The eye muscles are typically affected first

- usually presents as a descending paralysis rather than ascending as seen in other forms of Guillain-Barre syndrome
- anti-GQ1b antibodies are present in 90% of cases

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Medcomic 👍 18 👎 4

Guillain-Barre syndrome

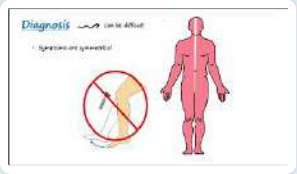
Suggest link Report broken link

Media

SYMPTOMS - BASED on NERVES AFFECTED
PARESTHESIA
LOSS of SENSATION
MUSCLE WEAKNESS & ABSENCE of REFLEXES
AUTONOMIC NERVES → VARIETY of SYMPTOMS

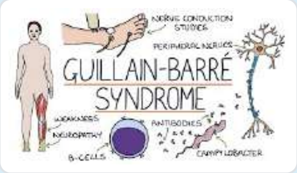
Guillain-Barre syndrome

Osmosis - YouTube 👍 0 👎 0



Guillain-Barre Syndrome (GBS) (Described Concisely)

PhysioPathoPharmaco - YouTube 👍 0 👎 0


NERVE CONDUCTION STUDIES
PERIPHERAL NERVES
GUILLAIN-BARRÉ SYNDROME
WEAKNESS
NEUROPATHY
B-CELLS
ANTIBODIES
CAMPYLOBACTER

Understanding Guillain-Barre Syndrome

Zero To Finals - YouTube 👍 0 👎 1

Report broken media

Score: **20.5%**

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A 44 year-old man presents to the neurology clinic. He complains of weakness in the right hand, which causes difficulty with tasks such as writing and dressing, and has also noted 'twitching' in his right forearm and hand. He cannot recall exactly when the symptoms first began but feels that they have certainly been present for at least six months, and that they have got worse.

He is otherwise fit and well. His only past medical history is well-controlled asthma. There is no family history of neurological disease.

Cranial nerve examination is normal. On examination of the right hand, there is weakness of extension of the the middle and ring fingers (2/5 on the MRC scale), but no discernible muscle wasting. Fasciculations are visible in the dorsal aspect of the forearm. On examination of the left hand, there is also some subtle weakness of extension of the ring finger (4/5 on the MRC scale). There is no weakness of wrist extension on either side and power in all other muscle groups is normal. For both hands, all the fingers extend when the wrist is passively moved into palmar flexion. All reflexes are normal and there are no sensory signs.

What is the most likely diagnosis?

- ○ Multifocal motor neuropathy
- ○ Chronic inflammatory demyelinating polyneuropathy
- ○ Motor neuron disease (amyotrophic lateral sclerosis)
- ○ Inclusion body myositis
- ○ Ruptured tendon of extensor digitorum

Submit answer

Reference ranges

Score: **17.6%**

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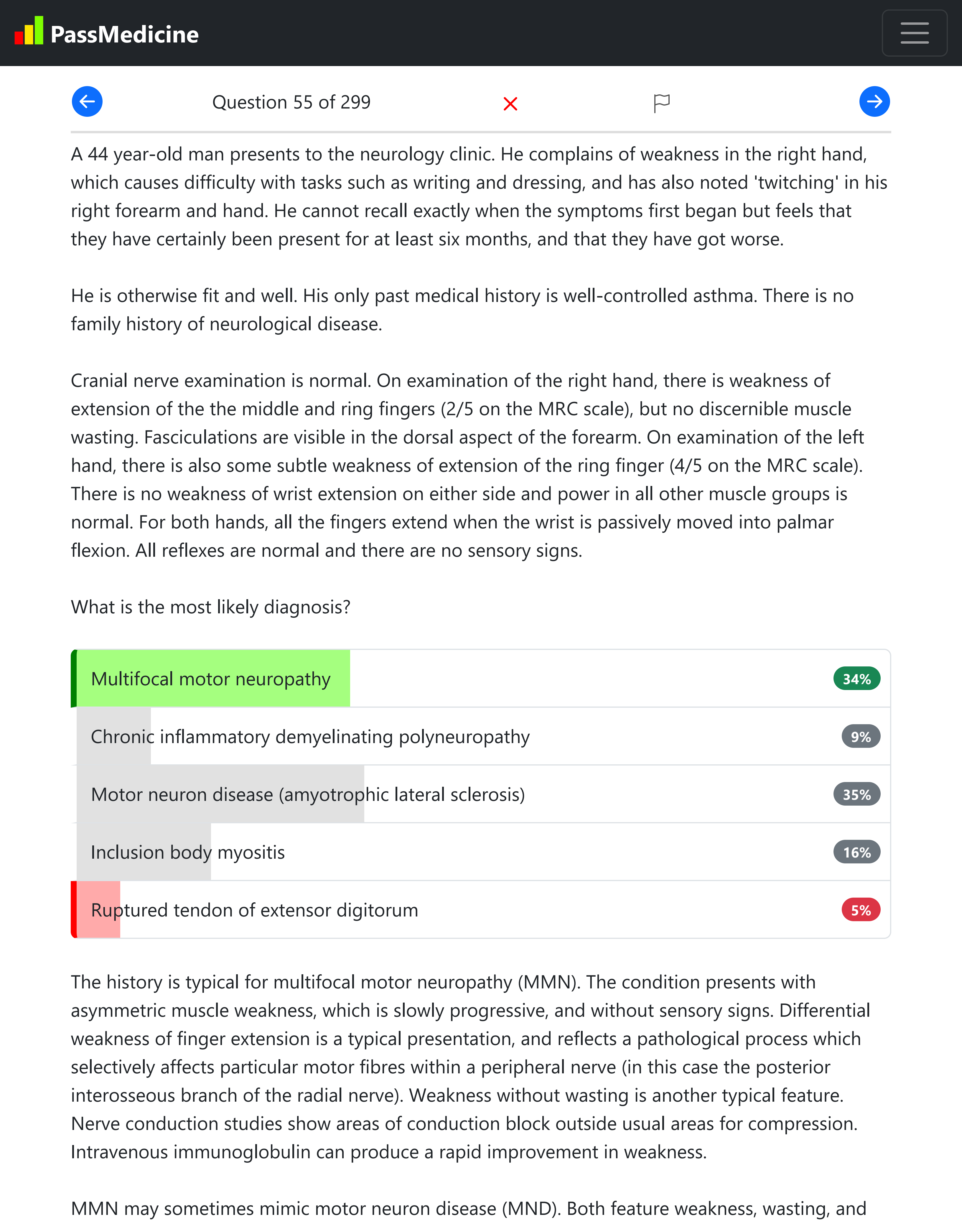

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What is the most likely diagnosis?

Multifocal motor neuropathy	34%
Chronic inflammatory demyelinating polyneuropathy	9%
Motor neuron disease (amyotrophic lateral sclerosis)	35%
Inclusion body myositis	16%
Ruptured tendon of extensor digitorum	5%

The history is typical for multifocal motor neuropathy (MMN). The condition presents with asymmetric muscle weakness, which is slowly progressive, and without sensory signs. Differential weakness of finger extension is a typical presentation, and reflects a pathological process which selectively affects particular motor fibres within a peripheral nerve (in this case the posterior interosseous branch of the radial nerve). Weakness without wasting is another typical feature. Nerve conduction studies show areas of conduction block outside usual areas for compression. Intravenous immunoglobulin can produce a rapid improvement in weakness.

MMN may sometimes mimic motor neuron disease (MND). Both feature weakness, wasting, and fasciculation, with relatively preserved reflexes, and normal sensation. However, certain features suggest one diagnosis over the other. For example, bulbar weakness is common in MND but rare in MMN. Depending on the form of MND, variable upper motor neuron signs may occur, contrary to MMN which is a purely lower motor neuron condition occurring in a distribution correlating with named peripheral nerves. Finally, whereas MND is usually fatal within 5 years, MMN is slowly progressive over decades and does not typically cause respiratory failure.

Chronic inflammatory demyelinating polyneuropathy(CIDP) typically causes a progressive sensory and motor neuropathy with loss of reflexes. CSF protein is elevation and nerve conduction studies show evidence of demyelination.

Inclusion body myositis characteristically causes weakness of finger flexion, as well as knee extension and ankle dorsiflexion. Affected muscles are wasted, and fasciculations do not occur as there is no denervation.

Rupture of extensor tendons often occurs in rheumatoid arthritis. The affected finger cannot be actively extended, and does not passively extend when the wrist is passively moved into palmar flexion.

👍 👎 Discuss (6) Improve

Next question >

Multifocal motor neuropathy ★

Acquired autoimmune demyelinating motor neuropathy

Associated with motor conduction block

Slowly progressive, distal motor neuropathy which progresses over many years

Anti-GM1 antibodies frequently raised

AI + Q 123

Next question >

B *I* A

Textbooks

High-yield textbook

Extended textbook

Score: **20.5%**

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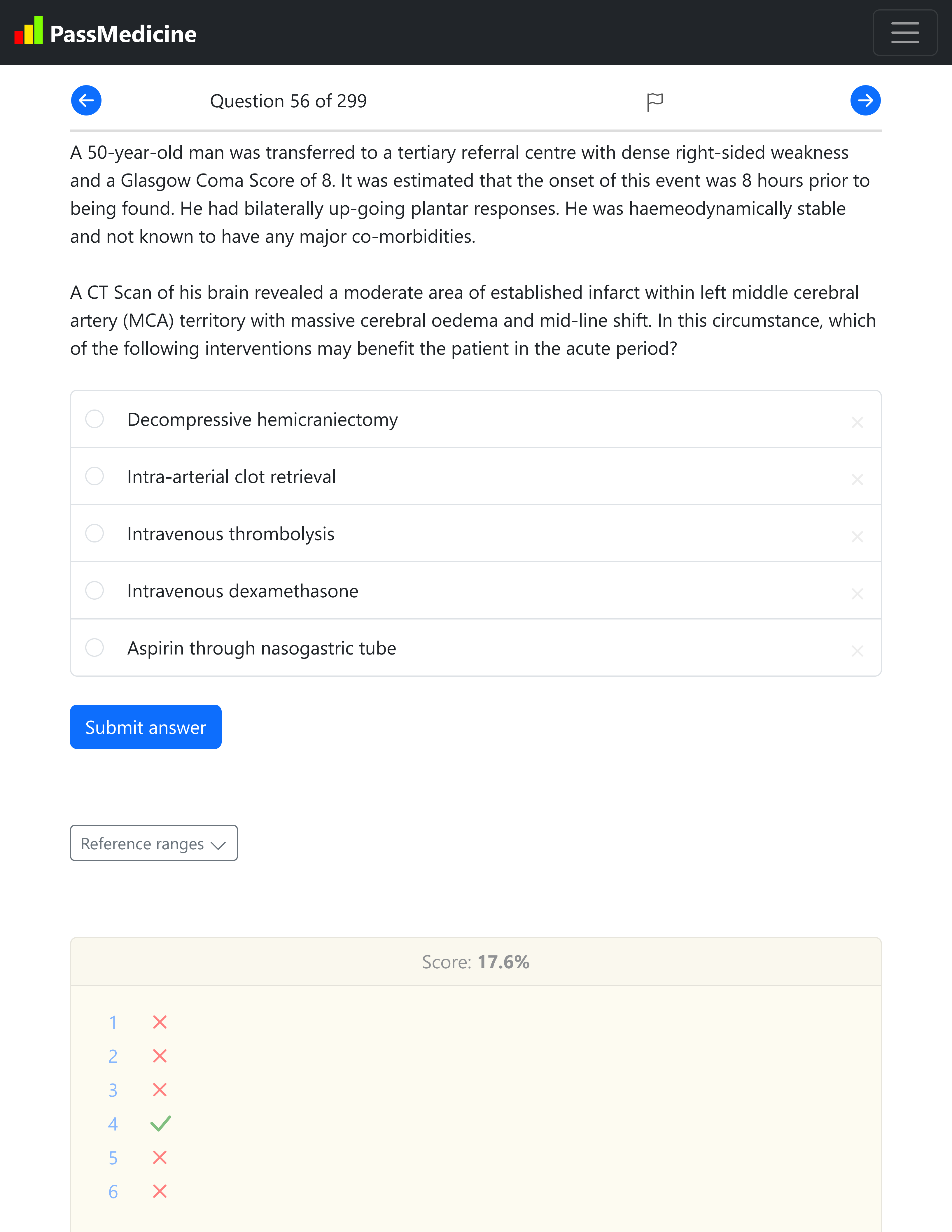

Question 56 of 299

A 50-year-old man was transferred to a tertiary referral centre with dense right-sided weakness and a Glasgow Coma Score of 8. It was estimated that the onset of this event was 8 hours prior to being found. He had bilaterally up-going plantar responses. He was haemeodynamically stable and not known to have any major co-morbidities.

A CT Scan of his brain revealed a moderate area of established infarct within left middle cerebral artery (MCA) territory with massive cerebral oedema and mid-line shift. In this circumstance, which of the following interventions may benefit the patient in the acute period?

- ○ Decompressive hemicraniectomy
- ○ Intra-arterial clot retrieval
- ○ Intravenous thrombolysis
- ○ Intravenous dexamethasone
- ○ Aspirin through nasogastric tube

Score: **17.6%**

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A CT Scan of his brain revealed a moderate area of established infarct within left middle cerebral artery (MCA) territory with massive cerebral oedema and mid-line shift. In this circumstance, which of the following interventions may benefit the patient in the acute period?

Decompressive hemicraniectomy	56%
Intra-arterial clot retrieval	17%
Intravenous thrombolysis	3%
Intravenous dexamethasone	21%
Aspirin through nasogastric tube	3%

For individuals aged up to 60 years who suffer an acute MCA territory ischaemic stroke complicated by massive cerebral oedema, surgical decompression by hemicraniectomy should be offered within 48 hours of stroke onset.

Discuss (7) Improve

Next question

Stroke: management of acute ischaemic stroke

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits

- blood pressure
  - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
  - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:
- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.
- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
  - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
  - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 11 👎 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 16

Stroke and TIA guidelines

Age and Ageing 👍 21 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

2023 National Clinical Guideline for Stroke

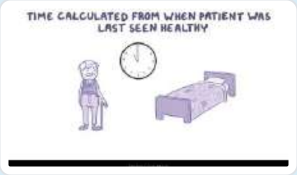
NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link

Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia

GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

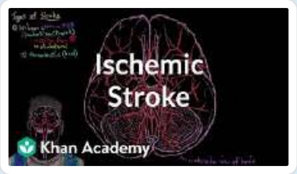
Radiology Channel - YouTube

👍 9 👎 0

Management of ischaemic stroke

[Stroke](#)

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
Khan Academy

[Iscchaemic stroke](#)

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

[Haemorrhagic stroke](#)

Khan Academy - YouTube 👍 1 👎 1

Report broken media

Score: **20.5%**

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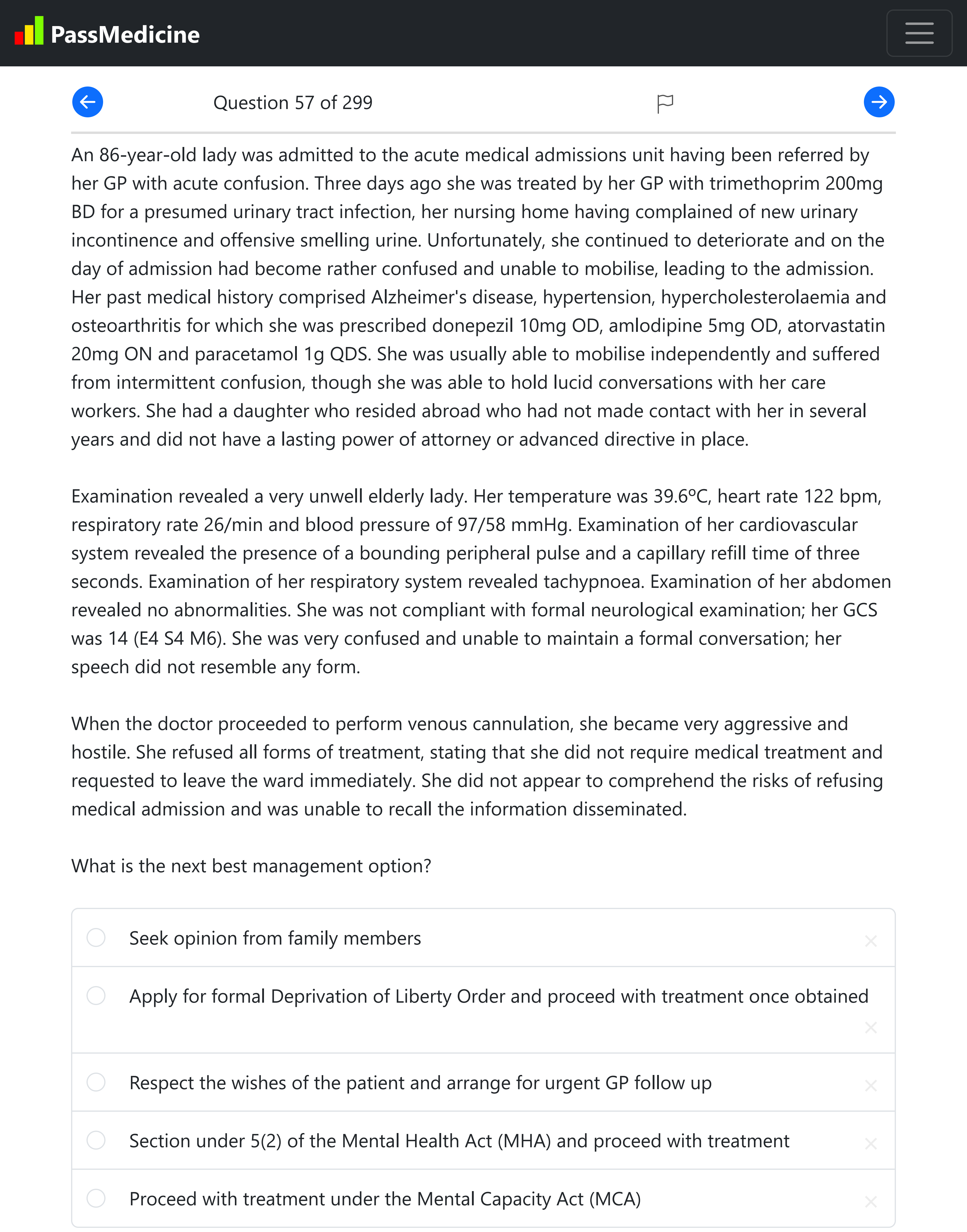

An 86-year-old lady was admitted to the acute medical admissions unit having been referred by her GP with acute confusion. Three days ago she was treated by her GP with trimethoprim 200mg BD for a presumed urinary tract infection, her nursing home having complained of new urinary incontinence and offensive smelling urine. Unfortunately, she continued to deteriorate and on the day of admission had become rather confused and unable to mobilise, leading to the admission. Her past medical history comprised Alzheimer's disease, hypertension, hypercholesterolaemia and osteoarthritis for which she was prescribed donepezil 10mg OD, amlodipine 5mg OD, atorvastatin 20mg ON and paracetamol 1g QDS. She was usually able to mobilise independently and suffered from intermittent confusion, though she was able to hold lucid conversations with her care workers. She had a daughter who resided abroad who had not made contact with her in several years and did not have a lasting power of attorney or advanced directive in place.

Examination revealed a very unwell elderly lady. Her temperature was 39.6ºC, heart rate 122 bpm, respiratory rate 26/min and blood pressure of 97/58 mmHg. Examination of her cardiovascular system revealed the presence of a bounding peripheral pulse and a capillary refill time of three seconds. Examination of her respiratory system revealed tachypnoea. Examination of her abdomen revealed no abnormalities. She was not compliant with formal neurological examination; her GCS was 14 (E4 S4 M6). She was very confused and unable to maintain a formal conversation; her speech did not resemble any form.

When the doctor proceeded to perform venous cannulation, she became very aggressive and hostile. She refused all forms of treatment, stating that she did not require medical treatment and requested to leave the ward immediately. She did not appear to comprehend the risks of refusing medical admission and was unable to recall the information disseminated.

What is the next best management option?

○ Seek opinion from family members

○ Apply for formal Deprivation of Liberty Order and proceed with treatment once obtained

○ Respect the wishes of the patient and arrange for urgent GP follow up

○ Section under 5(2) of the Mental Health Act (MHA) and proceed with treatment

○ Proceed with treatment under the Mental Capacity Act (MCA)

Submit answer

Reference ranges

Score: **17.6%**

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What is the next best management option?

Seek opinion from family members	3%
Apply for formal Deprivation of Liberty Order and proceed with treatment once obtained	23%
Respect the wishes of the patient and arrange for urgent GP follow up	3%
Section under 5(2) of the Mental Health Act (MHA) and proceed with treatment	17%
Proceed with treatment under the Mental Capacity Act (MCA)	53%

This lady has evidence of a life-threatening acute organic illness requiring immediate treatment, but is refusing treatment and lacks the capacity at present to make a decision regarding her medical care, and does not have an advanced directive of lasting power of attorney. Responsibility for decisions regarding medical care, therefore, is bestowed to the medical team, who are entitled to treat the patient under the Medical Capacity Act. Seeking a family opinion here is good practice but impractical here given the paucity of family contact and the emergency situation, and the Mental Health Act is not applicable here as the primary illness is most likely organic rather than relating to a mental health disorder. It is worth mentioning that under the MCA it is possible to obtain an emergency DOLS; a formal DOLS application must be made at the same time but in an emergency situation awaiting a formal DOLS should not prevent treatment.

👍 👎 Discuss (6) Improve

Next question ›

Mental Capacity Act ★

The Mental Capacity Act of 2005 came into force in 2007. It applies to adults over the age of 16 and sets out who can take decisions if a patient becomes incapacitated (e.g. following a stroke). Mental capacity includes the ability to make decisions affecting daily life, healthcare and financial issues.

The Act contains 5 key principles:

- A person must be assumed to have capacity unless it is established that he lacks capacity
- A person is not to be treated as unable to make a decision unless all practicable steps to help him to do so have been taken without success
- A person is not to be treated as unable to make a decision merely because he makes an unwise decision
- An act done, or decision made, under this Act for or on behalf of a person who lacks capacity must be done, or made, in his best interests
- Before the act is done, or the decision is made, regard must be had to whether the purpose for which it is needed can be as effectively achieved in a way that is less restrictive of the person's rights and freedom of action

Assessment of capacity

The Act sets out a clear test for assessing whether a person lacks capacity. It is a 'decision-specific' and 'time-specific' test. An adult can only be considered unable to make a particular decision if:

1. He or she has an 'impairment of, or disturbance in, the functioning of the mind or brain' whether permanent or temporary AND

2. He or she is unable to undertake any of the following
- a. understand the information relevant to the decision
- b. retain that information
- c. use or weigh that information as part of the process of making the decision
- d. communicate the decision made by talking, sign language or other means

No individual can be labelled 'incapable' simply as a result of a particular medical condition. Section 2 of the Act makes it clear that a lack of capacity cannot be assumed by a person's age, appearance, or any condition or aspect of a person's behaviour

Best interests

The following should be considered when assessing what is in someone's best interests:
- 1. Whether the person is likely to regain capacity and can the decision wait.
- 2. How to encourage and optimise the participation of the person in the decision.
- 3. The past and present wishes, feelings, beliefs, values of the person and any other relevant factors
- 4. Views of other relevant people

Lasting Powers of Attorney (LPAs)

The Act allows a person to appoint an attorney to act on their behalf if they should lose capacity in the future, replacing the current Enduring Power of Attorney (EPA). In addition to property and financial affairs the Act also allows people to empower an attorney make health and welfare decisions. The attorney only has the authority to make decisions about life-sustaining treatment if the LPA specifies that. Before it can be used an LPA must be registered with the Office of the Public Guardian

Advance decisions

Advance decisions can be drawn up by anybody with capacity to specify treatments they would not want if they lost capacity. They may be made verbally unless they specify refusing life-sustaining treatment (e.g. Ventilation) in which case they need to be written, signed and witnessed to be valid. Advance decisions cannot demand treatment

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

GMC 👍 2 👎 1

Mental capacity

Gov.uk 👍 3 👎 0

Mental Capacity Act 2005

Suggest link Report broken link

Score: **20.5%**

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A 70-year-old male is evaluated at the neurology outpatient department for an episode of left sided body weakness which lasted for around 30 minutes and resolved completely. He described the episode as an inability to move the left side of his body and an associated numbness and tingling sensation in the area involved. He remained conscious throughout and was able to communicate verbally with his family members during the episode.

He suffers from hypertension and has hypercholesterolaemia. He is also a smoker with a 40 pack year history and is known to be a heavy drinker.

He has also noted a tremor in both his hands which tends to improve after he has a drink. He also feels unsteady while walking and feels the need to grip something otherwise he may lose balance. His medication includes amlodipine 5mg daily and atorvastatin 20mg daily.

On examination, his blood pressure is 150/95mmHg and his pulse is 86bpm regular. A carotid bruit is audible over both sides of the neck. Neurological examination reveals impaired sensations in a glove and stocking distribution. The remaining clinical examination is normal.

Investigations reveal:

ECG: deep S waves in lead V1-V3 and tall R waves in V4-V6
CXR: Enlarged cardiac silhouette with flecks of calcification around the aorta

Carotid artery Doppler studies reveal 85% occlusion in the right external carotid. 50% occlusion in the right internal carotid. Left internal carotid is 80% occluded while there is a 60% occlusion in the left external carotid artery.

Which of the following is the most suitable treatment option in this patient?

- ○ Left internal carotid endarterectomy
- ○ Optimizing medical management
- ○ Right external carotid endarterectomy
- ○ Percutaneous stenting of the right internal carotid artery
- ○ Percutaneous stenting of the left internal carotid artery

Submit answer

Reference ranges

Score: **17.6%**

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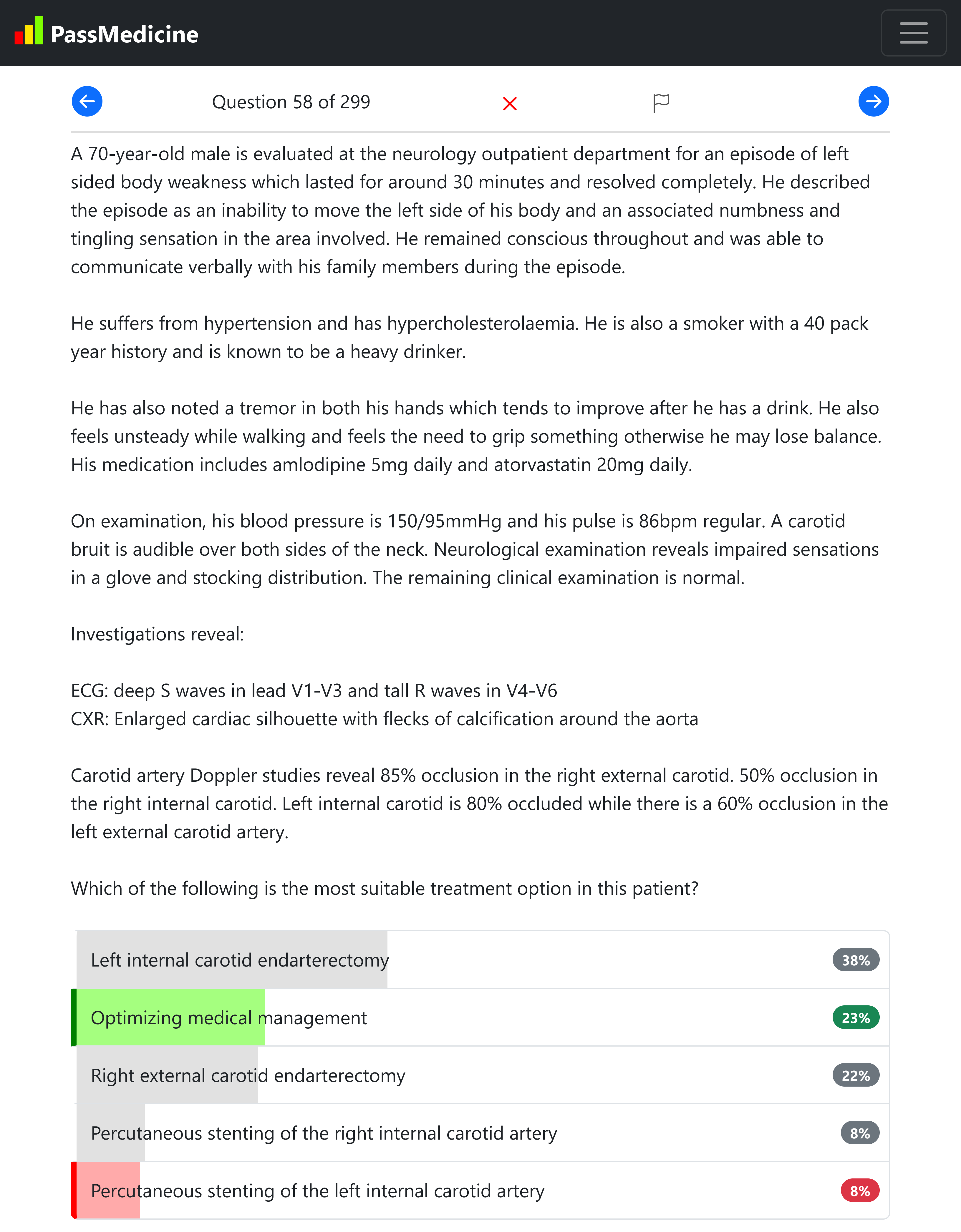

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Carotid artery Doppler studies reveal 85% occlusion in the right external carotid. 50% occlusion in the right internal carotid. Left internal carotid is 80% occluded while there is a 60% occlusion in the left external carotid artery.

Which of the following is the most suitable treatment option in this patient?

Left internal carotid endarterectomy	38%
Optimizing medical management	23%
Right external carotid endarterectomy	22%
Percutaneous stenting of the right internal carotid artery	8%
Percutaneous stenting of the left internal carotid artery	8%

In the aforementioned scenario, the patient has suffered from a right sided TIA and has recovered completely. The question aims to assess the candidates knowledge pertaining to intervention in patients with TIAs and carotid stenosis.

Carotid endarterectomy or endovascular stenting is recommended in patients with symptomatic stenosis of the affected vessel >70%. In the patient described above, the symptoms involve the left side of the body, hence the right carotid is involved. The degree of stenosis in the right internal carotid is 50 % and therefore the treatment of choice would be optimization of medical therapy with improved blood pressure control and antiplatelet agents rather than intervention.

The carotid stenosis in the left internal carotid is asymptomatic therefore not an indication for endarterectomy.

The external carotids do not form part of the circle of Willis and do not contribute to the blood supply of the brain.

The reference to his benign essential tremor is of no relevance to his clinical condition.

👍 | 👎 | Discuss (10) | Improve

Next question >

Stroke: management of acute ischaemic stroke ★

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
    - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
    - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded

- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:
- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.
- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
    - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
    - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy

Absolute	Relative
- Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

+ Q 123

Next question >

Textbooks

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Links

Royal College of Physicians 11 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 16

Stroke and TIA guidelines

Age and Ageing 👍 21 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

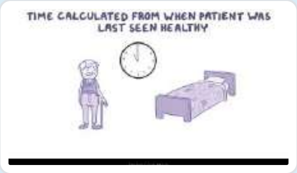
2023 National Clinical Guideline for Stroke

NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia
Acute Ischaemic Stroke

GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

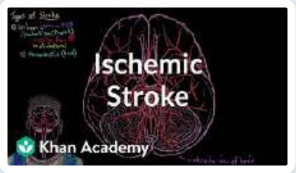
Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube

👍 2 👎 1


Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

Khan Academy - YouTube 👍 1 👎 1

Report broken media

Score: **20.5%**

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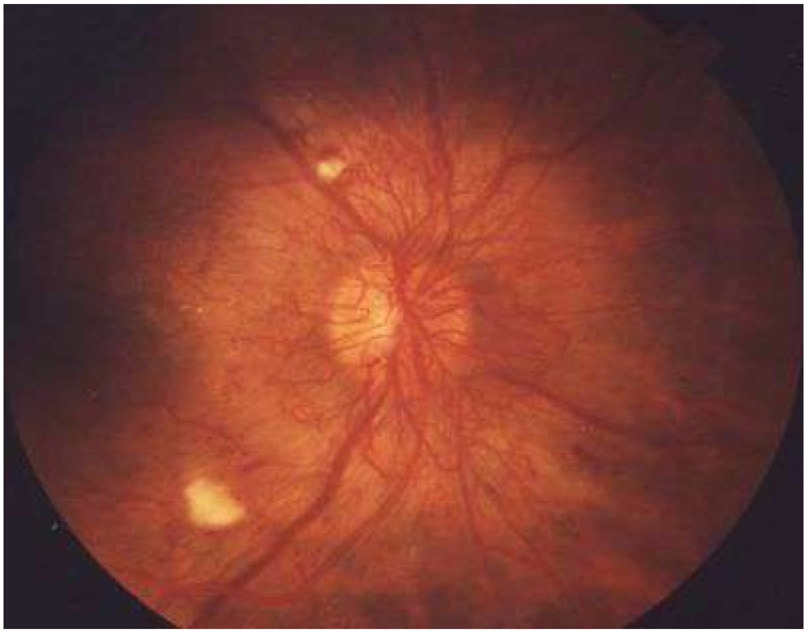
A 50-year-old woman presents to the optician with a progressive decline in visual acuity. The optician refers her for medical review. The fundoscopic examination shows:



Given the fundoscopic findings, what is the most appropriate treatment?

○ Intra-vitreal VEGF inhibitor therapy

○ Optimise glycaemic control

○ Pan-retinal laser photocoagulation

○ Urgent trabeculectomy

○ Vitreoretinal surgery

Submit answer

Reference ranges

Score: **17.6%**

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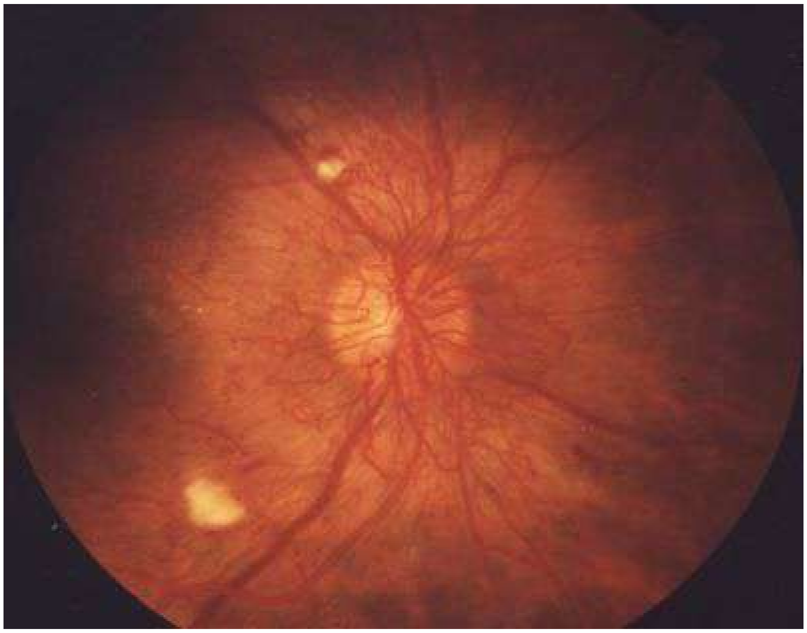


Given the fundoscopic findings, what is the most appropriate treatment?

Intra-vitreal VEGF inhibitor therapy	32%
Optimise glycaemic control	11%
Pan-retinal laser photocoagulation	51%
Urgent trabeculectomy	2%
Vitreoretinal surgery	5%

The fundoscopy shows extensive neovascularisation in all four quadrants obscuring the optic disc, which is suggestive of advanced proliferative diabetic retinopathy commonly seen in type I diabetes. The soft exudates, seen as 'cotton wool spots' on the left upper and lower quadrant, represent areas of retinal infarction, suggesting advanced diabetic retinopathy.

Pan-retinal laser photocoagulation is the most appropriate treatment for this patient with extensive neovascularisation to improve retinal oxygenation and reduce the risk of retinal or vitreous haemorrhage. Pan-retinal photocoagulation aims to prevent haemorrhage and further neovascularisation, not to regain lost vision.

Optimise glycaemic control should be considered in all patients with non-proliferative or proliferative diabetic retinopathy. However, this patient, with extensive neovascularisation and impending haemorrhage, requires urgent photocoagulation.

Urgent trabeculectomy is the surgical intervention used to reduce intraocular pressure in acute angle-closure glaucoma. Glaucoma leads to the classical fundoscopic appearance of cupping or increased vertical cup-to-disc ratio, which is not seen in this patient.

Intra-vitreal VEGF inhibitor therapy is recommended for patients with maculopathy or may be considered in combination with pan-retinal laser photocoagulation to treat severe proliferative diabetic retinopathy. While VEGF as an adjunctive therapy can help in the regression of neo-vascular vessels, definitive treatment with photocoagulation is essential to prevent impending retinal or vitreal haemorrhage.

Vitreoretinal surgery is recommended for severe or vitreous haemorrhage, which is often a complication of extensive neovascularisation. However, the fundoscopic findings in this patient do not suggest severe or vitreous haemorrhage, which may be seen as a red haze of haemorrhage into the vitreous associated with dot-blot haemorrhages, neovascularisation elsewhere or boat-shaped pre-retinal haemorrhages.

👍 👎 Discuss (5) Improve

Next question >

Diabetic retinopathy ★

Diabetic retinopathy is the most common cause of blindness in adults aged 35-65 years-old.

Pathophysiology

Hyperglycaemia is thought to cause increased retinal blood flow and abnormal metabolism in the retinal vessel walls. This precipitates damage to endothelial cells and pericytes

Endothelial dysfunction leads to increased vascular permeability which causes the characteristic exudates seen on fundoscopy. Pericyte dysfunction predisposes to the formation of microaneurysms. Neovasculization is thought to be caused by the production of growth factors in response to retinal ischaemia

Classification

Patients are typically classified into those with non-proliferative diabetic retinopathy (NPDR), proliferative retinopathy (PDR) and maculopathy.

Non-proliferative diabetic retinopathy

Mild NPDR

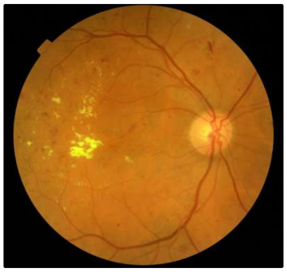
- 1 or more microaneurysm

Moderate NPDR

- microaneurysms
- blot haemorrhages
- hard exudates
- cotton wool spots ('soft exudates' - represent areas of retinal infarction), venous beading/looping and intraretinal microvascular abnormalities (IRMA) less severe than in severe NPDR

Severe NPDR

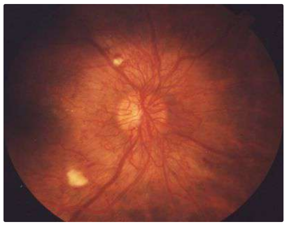
- blot haemorrhages and microaneurysms in 4 quadrants
- venous beading in at least 2 quadrants
- IRMA in at least 1 quadrant



Proliferative diabetic retinopathy

Key features

- retinal neovascularisation - may lead to vitrous haemorrhage
- fibrous tissue forming anterior to retinal disc
- more common in Type I DM, 50% blind in 5 years



Maculopathy

Key features

- based on location rather than severity, anything is potentially serious
- hard exudates and other 'background' changes on macula
- check visual acuity
- more common in Type II DM

Management

All patients

- optimise glycaemic control, blood pressure and hyperlipidemia
- regular review by ophthalmology

Maculopathy

- if there is a change in visual acuity then intravitreal vascular endothelial growth factor (VEGF) inhibitors

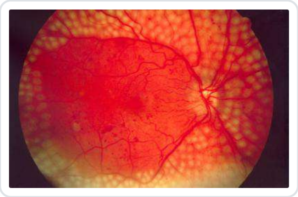
Non-proliferative retinopathy

- regular observation
- if severe/very severe consider panretinal laser photocoagulation

Proliferative retinopathy

- panretinal laser photocoagulation
    - following treatment around 50% of patients develop a noticeable reduction in their visual fields due to the scarring of peripheral retinal tissue
    - other complications include a decrease in night vision (rods are predominantly responsible for vision in low light conditions, the majority of rod cells are located in the peripheral retina), a generalised decrease in visual acuity and macular oedema
- intravitreal VEGF inhibitors
    - often now used in combination with panretinal laser photocoagulation
    - examples include ranibizumab
    - strong evidence base suggests they both slow progression of proliferative diabetic retinopathy and improve visual acuity

- if severe or vitreous haemorrhage: vitreoretinal surgery



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Next question

Textbooks

High-yield textbook

Extended textbook

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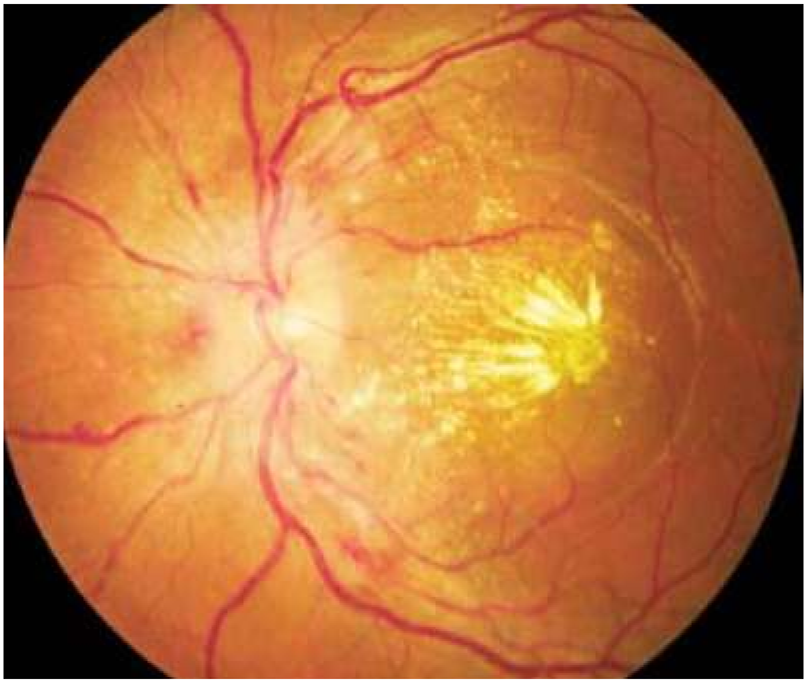
A 75-year-old man visited the optician for a progressive decline in vision in both eyes and a headache. The optician was concerned about his fundoscopic appearance and referred him to the emergency department for further workup. He has hypertension and diabetes and reports good compliance with his medications. He currently takes perindopril, amlodipine and hydrochlorothiazide for hypertension; metformin, linagliptin and dapagliflozin for diabetes management.



What is the next best step in his management?

- ○ Add basal-bolus insulin therapy
- ○ Escalate antihypertensive treatment
- ○ Monitor visual acuity weekly
- ○ Refer for urgent trabeculectomy
- ○ Start broad-spectrum antibiotics

Submit answer

Reference ranges

Score: **17.6%**

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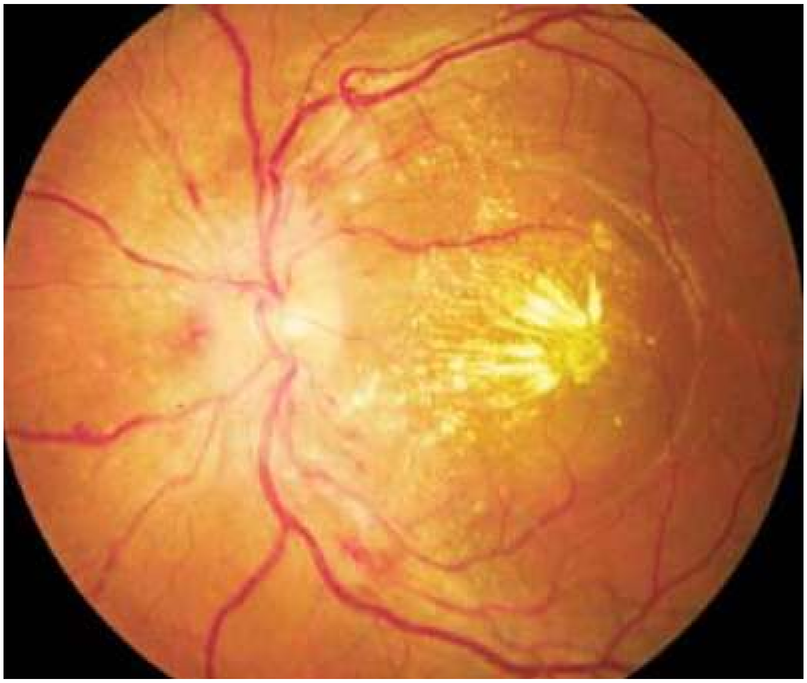
A 75-year-old man visited the optician for a progressive decline in vision in both eyes and a headache. The optician was concerned about his fundoscopic appearance and referred him to the emergency department for further workup. He has hypertension and diabetes and reports good compliance with his medications. He currently takes perindopril, amlodipine and hydrochlorothiazide for hypertension; metformin, linagliptin and dapagliflozin for diabetes management.



What is the next best step in his management?

Add basal-bolus insulin therapy	15%
Escalate antihypertensive treatment	48%
Monitor visual acuity weekly	8%
Refer for urgent trabeculectomy	28%
Start broad-spectrum antibiotics	1%

The flame and blot haemorrhages collected around the fovea result in a characteristic macular star pattern. The associated arteriolar narrowing and AV nipping are suggestive of hypertensive retinopathy. The more ominous sign of papilloedema indicates stage IV hypertensive retinopathy.

Escalate antihypertensive treatment is the next best step in his management. Stage IV hypertensive retinopathy is indicative of uncontrolled malignant hypertension. Prompt review and escalation of hypertensive treatment are vital in reducing ophthalmologic and systemic complications.

Add basal-bolus insulin may be considered if the patient has poorly controlled diabetes despite optimised oral therapy. Although the fundoscopic features suggest hypertensive rather than diabetic retinopathy, persistent hypertension can accelerate the progression of diabetic eye disease.

Monitor visual acuity weekly is inappropriate as this patient with stage IV hypertensive retinopathy requires prompt treatment to control blood pressure to reduce ophthalmologic and systemic complications.

Refer for urgent trabeculectomy , which is the most common surgical intervention for glaucoma, is not needed in this patient. Glaucoma leads to the classical fundoscopic appearance of cupping or increased vertical cup-to-disc ratio, which is not seen in this patient.

Start broad-spectrum antibiotics like azithromycin or ciprofloxacin would be the appropriate treatment for the cat-scratch disease, which is the most common cause of infectious neuroretinitis. Although macular star pattern and papilloedema may also be seen in neuroretinitis, the onset is typically acute and often limited to one eye.

👍 👎 💬 Discuss (1) Improve

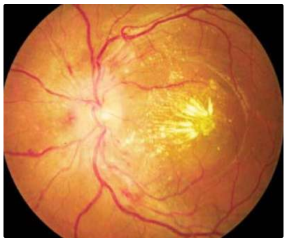
Next question ›

Hypertensive retinopathy ★

The table below shows the Keith-Wagener classification of hypertensive retinopathy

Stage	Features
I	Arteriolar narrowing and tortuosity Increased light reflex - silver wiring
II	Arteriovenous nipping
III	Cotton-wool exudates Flame and blot haemorrhages

Stage	Features
	• These may collect around the fovea resulting in a 'macular star'
IV	Papilloedema



123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

HYPERTENSIVE
RETINOPATHY

Hypertensive retinopathy stages

Last Second Medicine - YouTube 14 3

Report broken media

Score: **20.5%**

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Question 61 of 299

A 21-year-old man is referred to the ophthalmology clinic. He has a past medical history of type 1 diabetes mellitus which has been poorly controlled as he has recently started university and is living away from home for the first time. This has caused him to monitor his blood sugars less frequently as he is embarrassed about his medical problem and is keen to fit in. He has no visual symptoms but fundoscopy demonstrates microaneurysms and cotton wool spots. He has no other known medical problems and uses biphasic insulin. His observations are all within normal range visual acuity is normal.

Apart from advising and support in managing his diabetes, how should he be managed?

- ○ Referral for retinal laser treatment
- ○ Annual digital retinal photography
- ○ Six monthly digital retinal photography
- ○ Start ramipril
- ○ Start amlodipine

Submit answer

Reference ranges

Score: **17.6%**

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A 21-year-old man is referred to the ophthalmology clinic. He has a past medical history of type 1 diabetes mellitus which has been poorly controlled as he has recently started university and is living away from home for the first time. This has caused him to monitor his blood sugars less frequently as he is embarrassed about his medical problem and is keen to fit in. He has no visual symptoms but fundoscopy demonstrates microaneurysms and cotton wool spots. He has no other known medical problems and uses biphasic insulin. His observations are all within normal range visual acuity is normal.

Apart from advising and support in managing his diabetes, how should he be managed?

Referral for retinal laser treatment	24%
Annual digital retinal photography	33%
Six monthly digital retinal photography	34%
Start ramipril	8%
Start amlodipine	0%

The correct answer is annual digital retinal photography. This patient has developed pre-proliferative diabetic retinopathy and strict diabetic control is important. Apart from that, regular retinal photography can help find proliferative retinopathy in which case laser treatment will be needed. There is no evidence of hypertensive changes.

Diabetic retinopathy in type 1 diabetes

People with type 1 diabetes are at high risk of diabetic retinopathy. On diagnosis, they should be referred to screening services and from that point either undergo annual review or be referred to an ophthalmologist. If signs of proliferative retinopathy occur then the patient should be referred for retinal laser treatment.

Source:
'Type 1 Diabetes in Adults: Diagnosis and Management.' NICE Guideline [NG17]. National Institute of Care and Health Excellence, July 2016

Discuss (4) | Improve

Next question

Diabetic retinopathy

Diabetic retinopathy is the most common cause of blindness in adults aged 35-65 years-old.

Pathophysiology

Hyperglycaemia is thought to cause increased retinal blood flow and abnormal metabolism in the retinal vessel walls. This precipitates damage to endothelial cells and pericytes

Endothelial dysfunction leads to increased vascular permeability which causes the characteristic exudates seen on fundoscopy. Pericyte dysfunction predisposes to the formation of microaneurysms. Neovasculization is thought to be caused by the production of growth factors in response to retinal ischaemia

Classification

Patients are typically classified into those with non-proliferative diabetic retinopathy (NPDR), proliferative retinopathy (PDR) and maculopathy.

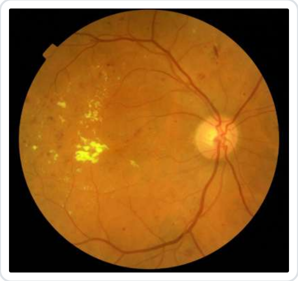
Non-proliferative diabetic retinopathy

Mild NPDR
- 1 or more microaneurysm

Moderate NPDR
- microaneurysms
- blot haemorrhages
- hard exudates
- cotton wool spots ('soft exudates' - represent areas of retinal infarction), venous beading/looping and intraretinal microvascular abnormalities (IRMA) less severe than in severe NPDR

Severe NPDR

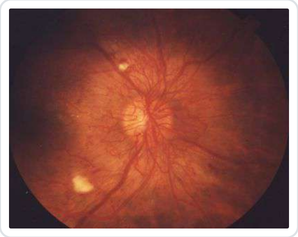
- blot haemorrhages and microaneurysms in 4 quadrants
- venous beading in at least 2 quadrants
- IRMA in at least 1 quadrant



Proliferative diabetic retinopathy

Key features

- retinal neovascularisation - may lead to vitrous haemorrhage
- fibrous tissue forming anterior to retinal disc
- more common in Type I DM, 50% blind in 5 years



Maculopathy

Key features

- based on location rather than severity, anything is potentially serious
- hard exudates and other 'background' changes on macula
- check visual acuity
- more common in Type II DM

Management

All patients

- optimise glycaemic control, blood pressure and hyperlipidemia
- regular review by ophthalmology

Maculopathy

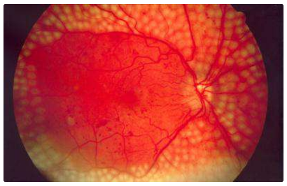
- if there is a change in visual acuity then intravitreal vascular endothelial growth factor (VEGF) inhibitors

Non-proliferative retinopathy

- regular observation
- if severe/very severe consider panretinal laser photocoagulation

Proliferative retinopathy

- panretinal laser photocoagulation
  - following treatment around 50% of patients develop a noticeable reduction in their visual fields due to the scarring of peripheral retinal tissue
  - other complications include a decrease in night vision (rods are predominantly responsible for vision in low light conditions, the majority of rod cells are located in the peripheral retina), a generalised decrease in visual acuity and macular oedema
- intravitreal VEGF inhibitors
  - often now used in combination with panretinal laser photocoagulation
  - examples include ranibizumab
  - strong evidence base suggests they both slow progression of proliferative diabetic retinopathy and improve visual acuity
- if severe or vitreous haemorrhage: vitreoretinal surgery



123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **20.5%**

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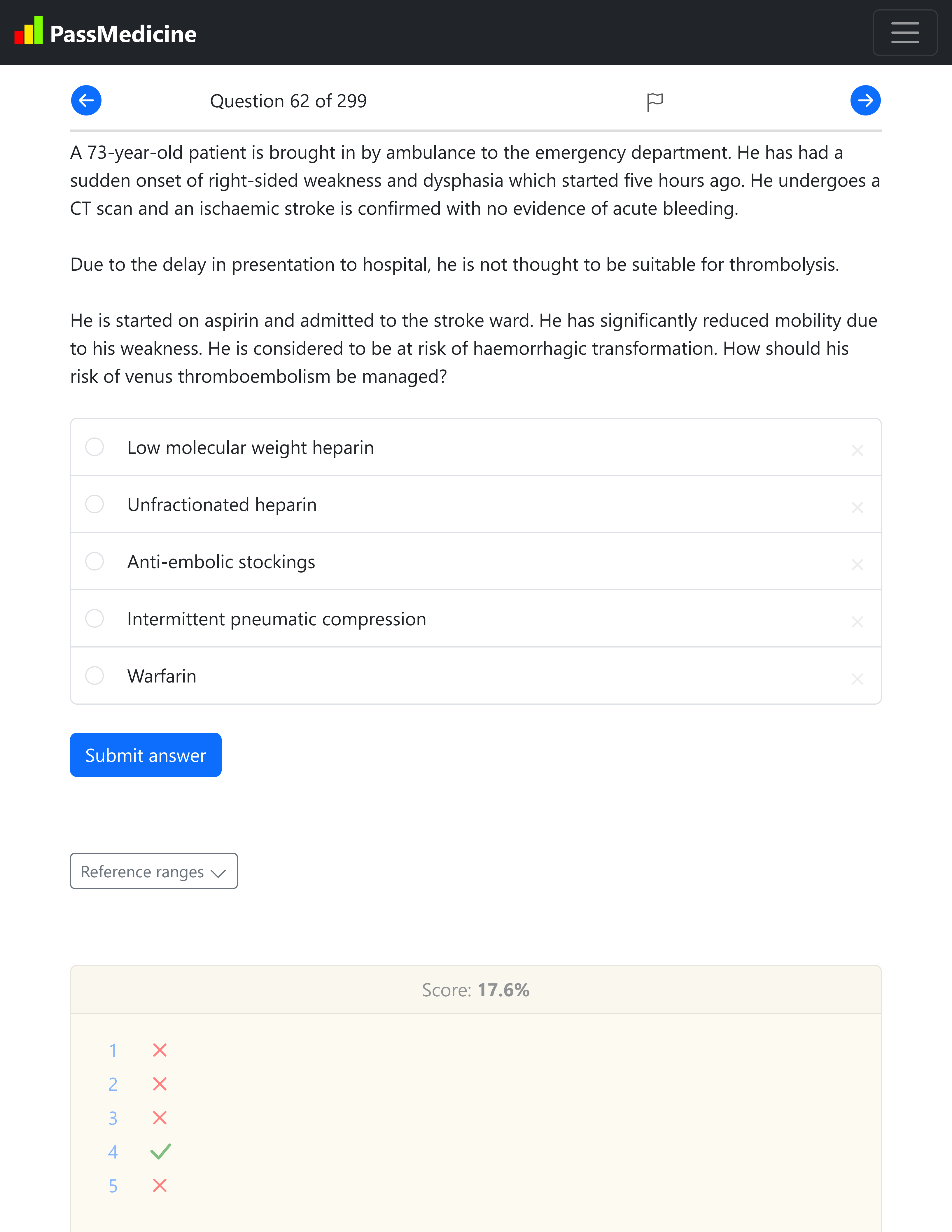

A 73-year-old patient is brought in by ambulance to the emergency department. He has had a sudden onset of right-sided weakness and dysphasia which started five hours ago. He undergoes a CT scan and an ischaemic stroke is confirmed with no evidence of acute bleeding.

Due to the delay in presentation to hospital, he is not thought to be suitable for thrombolysis.

He is started on aspirin and admitted to the stroke ward. He has significantly reduced mobility due to his weakness. He is considered to be at risk of haemorrhagic transformation. How should his risk of venus thromboembolism be managed?

○ Low molecular weight heparin ×

○ Unfractionated heparin ×

○ Anti-embolic stockings ×

○ Intermittent pneumatic compression ×

○ Warfarin ×

Submit answer

Reference ranges

Score: **17.6%**

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A 73-year-old patient is brought in by ambulance to the emergency department. He has had a sudden onset of right-sided weakness and dysphasia which started five hours ago. He undergoes a CT scan and an ischaemic stroke is confirmed with no evidence of acute bleeding.

Due to the delay in presentation to hospital, he is not thought to be suitable for thrombolysis.

He is started on aspirin and admitted to the stroke ward. He has significantly reduced mobility due to his weakness. He is considered to be at risk of haemorrhagic transformation. How should his risk of venus thromboembolism be managed?

Low molecular weight heparin	13%
Unfractionated heparin	7%
Anti-embolic stockings	22%
Intermittent pneumatic compression	57%
Warfarin	1%

The correct answer is intermittent pneumatic compression. Patients admitted with stroke are very likely to be at high risk of developing a thromboembolic event due to reduced mobility, but low molecular weight heparin is associated with haemorrhagic transformation. NICE advises that patients admitted within three days of an acute stroke should have intermittent pneumatic compression considered. This should be provided for 30 days or until the patient is mobile or discharged.

Source:

'Venous thromboembolism: reducing the risk for patients in hospital' Clinical guideline [CG92]. The National Institute for Health and Care Excellence, January 2010.

Discuss (5) Improve

Next question ›

Stroke: management of acute ischaemic stroke ★

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:
- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
  - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
  - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:
- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.
- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:

    - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
    - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	**Relative**
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with

intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 11 👎 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 16

Stroke and TIA guidelines

Age and Ageing 👍 21 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

2023 National Clinical Guideline for Stroke

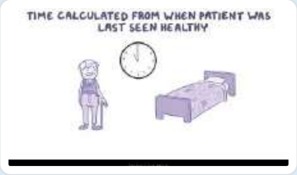
NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link

Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia.org
Acute Ischaemic Stroke

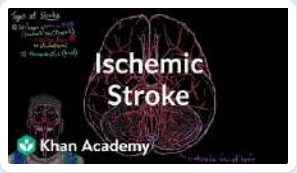
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

Khan Academy - YouTube 👍 1 👎 1

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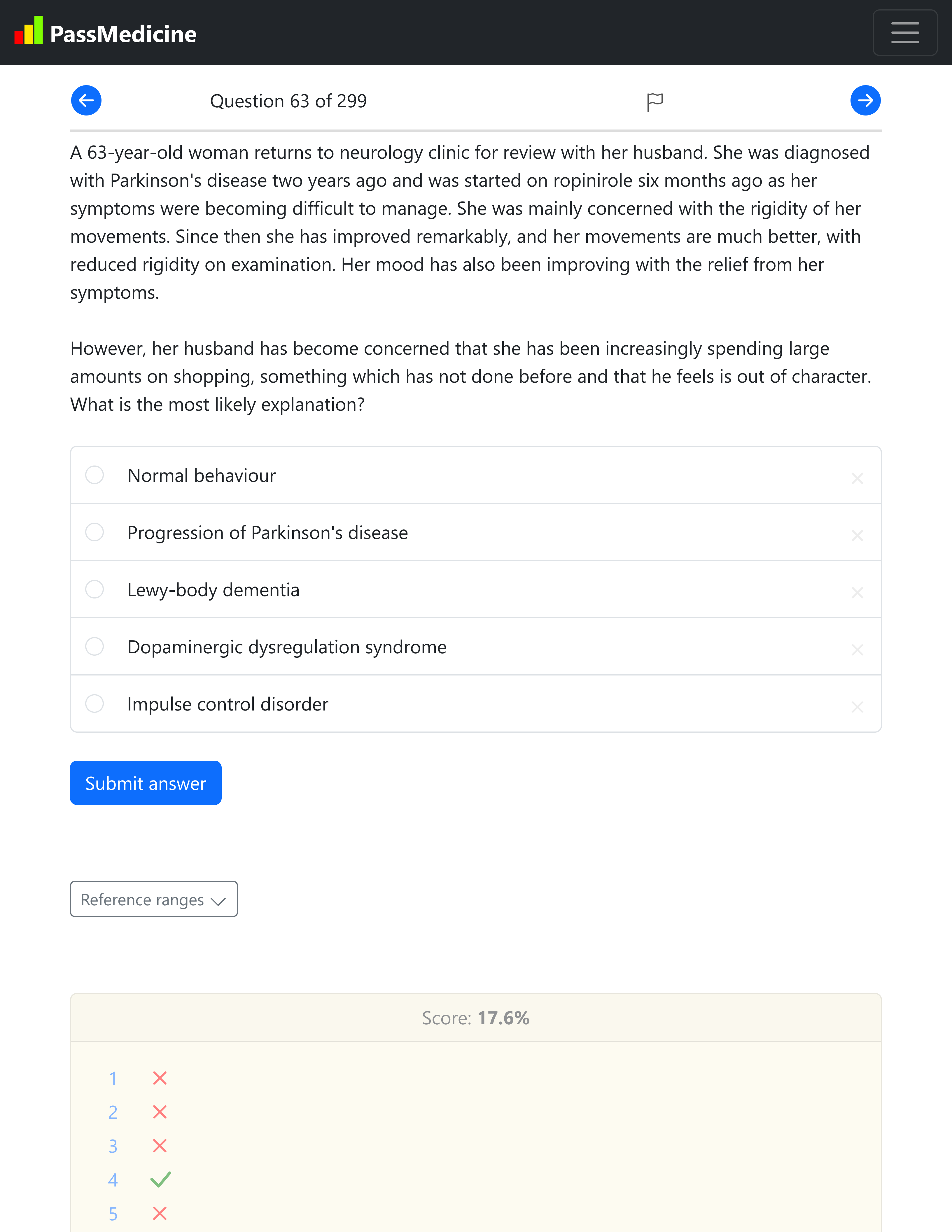

Question 63 of 299

A 63-year-old woman returns to neurology clinic for review with her husband. She was diagnosed with Parkinson's disease two years ago and was started on ropinirole six months ago as her symptoms were becoming difficult to manage. She was mainly concerned with the rigidity of her movements. Since then she has improved remarkably, and her movements are much better, with reduced rigidity on examination. Her mood has also been improving with the relief from her symptoms.

However, her husband has become concerned that she has been increasingly spending large amounts on shopping, something which has not done before and that he feels is out of character. What is the most likely explanation?

- ○ Normal behaviour
- ○ Progression of Parkinson's disease
- ○ Lewy-body dementia
- ○ Dopaminergic dysregulation syndrome
- ○ Impulse control disorder

Score: **17.6%**

1	✗
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3	✗
4	✓
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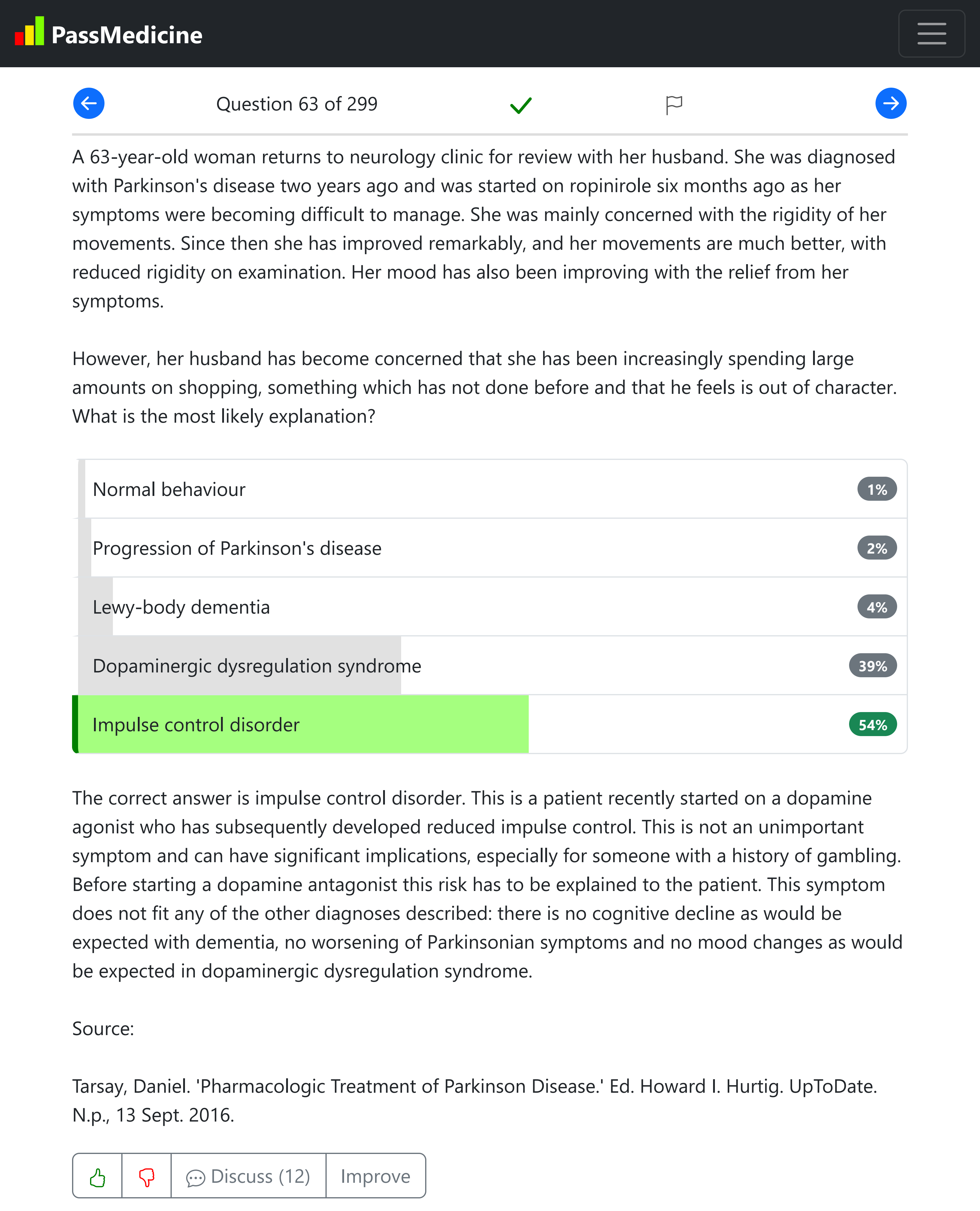


Question 63 of 299 ✓

A 63-year-old woman returns to neurology clinic for review with her husband. She was diagnosed with Parkinson's disease two years ago and was started on ropinirole six months ago as her symptoms were becoming difficult to manage. She was mainly concerned with the rigidity of her movements. Since then she has improved remarkably, and her movements are much better, with reduced rigidity on examination. Her mood has also been improving with the relief from her symptoms.

However, her husband has become concerned that she has been increasingly spending large amounts on shopping, something which has not done before and that he feels is out of character. What is the most likely explanation?

Normal behaviour	1%
Progression of Parkinson's disease	2%
Lewy-body dementia	4%
Dopaminergic dysregulation syndrome	39%
Impulse control disorder ✓	54%

The correct answer is impulse control disorder. This is a patient recently started on a dopamine agonist who has subsequently developed reduced impulse control. This is not an unimportant symptom and can have significant implications, especially for someone with a history of gambling. Before starting a dopamine antagonist this risk has to be explained to the patient. This symptom does not fit any of the other diagnoses described: there is no cognitive decline as would be expected with dementia, no worsening of Parkinsonian symptoms and no mood changes as would be expected in dopaminergic dysregulation syndrome.

Source:

Tarsay, Daniel. 'Pharmacologic Treatment of Parkinson Disease.' Ed. Howard I. Hurtig. UpToDate. N.p., 13 Sept. 2016.

Next question >

Parkinson's disease: management ★

Parkinsons disease should only be diagnosed, and management initiated, by a specialist with expertise in movement disorders. However, it is important for all doctors to be aware of the medications used in Parkinson's given the prevalence of this condition. NICE published guidelines in 2017 regarding the management of Parkinson's disease.

For first-line treatment:

- if the motor symptoms are affecting the patient's quality of life: levodopa
- if the motor symptoms are not affecting the patient's quality of life: dopamine agonist (non-ergot derived), levodopa or monoamine oxidase B (MAO-B) inhibitor

Whilst all drugs used to treat Parkinson's can cause a wide variety of side-effects NICE produced tables to help with decision making:

	Levodopa	**Dopamine agonists**	**MAO-B inhibitors**
Motor symptoms	More improvement in motor symptoms	Less improvement in motor symptoms	Less improvement in motor symptoms
Activities of daily living	More improvement in activities of daily living	Less improvement in activities of daily living	Less improvement in activities of daily living
Motor complications	More motor complications	Fewer motor complications	Fewer motor complications
Adverse events	Fewer specified adverse events*	More specified adverse events*	Fewer specified adverse events*

* excessive sleepiness, hallucinations and impulse control disorders

If a patient continues to have symptoms despite optimal levodopa treatment or has developed dyskinesia then NICE recommend the addition of a dopamine agonist, MAO-B inhibitor or catechol-O-methyl transferase (COMT) inhibitor as an adjunct. Again, NICE summarise the main points in terms of decision making:

	Dopamine agonists	MAO-B inhibitors	COMT inhibitors	Amantadine
Motor symptoms	Improvement in motor symptoms	Improvement in motor symptoms	Improvement in motor symptoms	No evidence of improvement in motor symptoms
Activities of daily living	Improvement in activities of daily living	Improvement in activities of daily living	Improvement in activities of daily living	No evidence of improvement in activities of daily living
Off time	More off-time reduction	Off-time reduction	Off-time reduction	No studies reporting this outcome
Adverse events	Intermediate risk of adverse events	Fewer adverse events	More adverse events	No studies reporting this outcome
Hallucinations	More risk of hallucinations	Lower risk of hallucinations	Lower risk of hallucinations	No studies reporting this outcome

Specific points regarding Parkinson's medication

NICE reminds us of the risk of acute akinesia or neuroleptic malignant syndrome if medication is not taken/absorbed (for example due to gastroenteritis) and advise against giving patients a 'drug holiday' for the same reason.

Impulse control disorders have become a significant issue in recent years. These can occur with any dopaminergic therapy but are more common with:
- dopamine agonist therapy
- a history of previous impulsive behaviours
- a history of alcohol consumption and/or smoking

If excessive daytime sleepiness develops then patients should not drive. Medication should be adjusted to control symptoms. Modafinil can be considered if alternative strategies fail.

If orthostatic hypotension develops then a medication review looking at potential causes should be done. If symptoms persist then midodrine (acts on peripheral alpha-adrenergic receptors to increase arterial resistance) can be considered.

Consider glycopyrronium bromide to manage drooling of saliva in people with Parkinson's disease.

Further information regarding specific anti-Parkinson's medication

Levodopa

- nearly always combined with a decarboxylase inhibitor (e.g. carbidopa or benserazide)
    - this prevents the peripheral metabolism of levodopa to dopamine outside of the brain and hence can reduce side effects
- common adverse effects:
    - dry mouth
    - anorexia
    - palpitations
    - postural hypotension
    - psychosis
- some adverse effects are due to the difficulty in achieving a steady dose of levodopa
    - end-of-dose wearing off: symptoms often worsen towards the end of dosage interval. This results in a decline of motor activity
    - 'on-off' phenomenon: large variations in motor performance, with normal function during the 'on' period, and weakness and restricted mobility during the 'off' period
    - dyskinesias at peak dose: dystonia, chorea and athetosis (involuntary writhing movements)
    - these effects may worsen over time with - clinicians therefore may limit doses until necessary
- it is important not to acutely stop levodopa, for example, if a patient is admitted to hospital
    - if a patient with Parkinson's disease cannot take levodopa orally, they can be given a dopamine agonist patch as rescue medication to prevent acute dystonia

Dopamine receptor agonists

- e.g. bromocriptine, ropinirole, cabergoline, apomorphine
- ergot-derived dopamine receptor agonists (bromocriptine, cabergoline) have been associated with pulmonary, retroperitoneal and cardiac fibrosis. The Committee on Safety of Medicines advice that an echocardiogram, ESR, creatinine and chest x-ray should be obtained prior to treatment and patients should be closely monitored
- patients should be warned about the potential for dopamine receptor agonists to cause impulse control disorders and excessive daytime somnolence
- more likely than levodopa to cause hallucinations in older patients. Nasal congestion and postural hypotension are also seen in some patients

MAO-B (Monoamine Oxidase-B) inhibitors

- e.g. selegiline
- inhibits the breakdown of dopamine secreted by the dopaminergic neurons

Amantadine

- mechanism is not fully understood, probably increases dopamine release and inhibits its uptake at dopaminergic synapses
- side-effects include ataxia, slurred speech, confusion, dizziness and livedo reticularis

COMT (Catechol-O-Methyl Transferase) inhibitors

- e.g. entacapone, tolcapone
- COMT is an enzyme involved in the breakdown of dopamine, and hence may be used as an adjunct to levodopa therapy
- used in conjunction with levodopa in patients with established PD

Antimuscarinics

- block cholinergic receptors
- now used more to treat drug-induced parkinsonism rather than idiopathic Parkinson's disease
- help tremor and rigidity
- e.g. procyclidine, benzotropine, trihexyphenidyl (benzhexol)

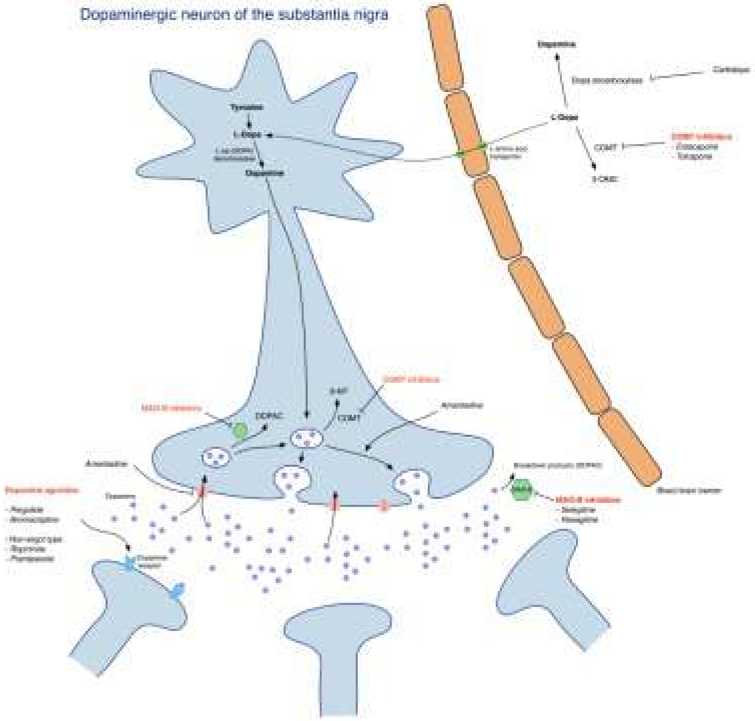

Dopaminergic neuron of the substantia nigra
Tyrosine
L-Dopa
Dopamine
Dopamine
L-Dopa
COMT
3-OMD
DOPAC
3-MT
COMT
Amantadine
Amantadine

Image sourced from Wikipedia

Diagram showing the mechanism of action of Parkinson's drugs

123

Next question

Textbooks

High-yield textbook

Extended textbook

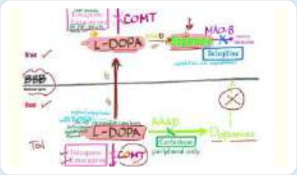
Links

NICE 👍 23 👎 12

2017 Parkinson's disease guidelines

Suggest link Report broken link

Media


COMT
MAO-B
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The use of Levodopa/ Carbidopa in the treatment of Parkinson's disease

Medicosis Perfectionalis - YouTube 👍 13 👎 0

PARKINSON'S
DISEASE
PHARMACOLOGY

Parkinson's disease pharmacology

Soton Brain Hub - YouTube

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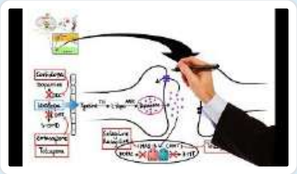
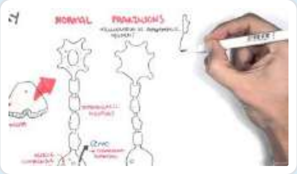


Pharmacology - DRUGS FOR PARKINSON'S DISEASE

Speed Pharmacology - YouTube 👍 8 👎 1


NORMAL
PARKINSON'S

Pharmacology - Parkinson's Disease

Armando Hasudungan - YouTube 👍 16 👎 2

PARKINSON'S DISEASE
Osmosis.org

Parkinson's disease

Osmosis - YouTube 👍 4 👎 1

PARKINSON'S DISEASE
SHUFFLING GAIT
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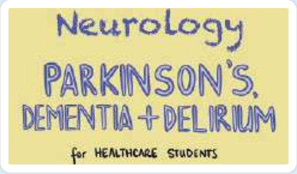
Understanding Parkinson's Disease

Rhesus Medicine - YouTube 👍 2 👎 1


Neurology
PARKINSON'S, DEMENTIA + DELIRIUM
for HEALTHCARE STUDENTS

Parkinson's disease

MedFlix - YouTube 👍 1 👎 2

Report broken media

Score: **20.5%**

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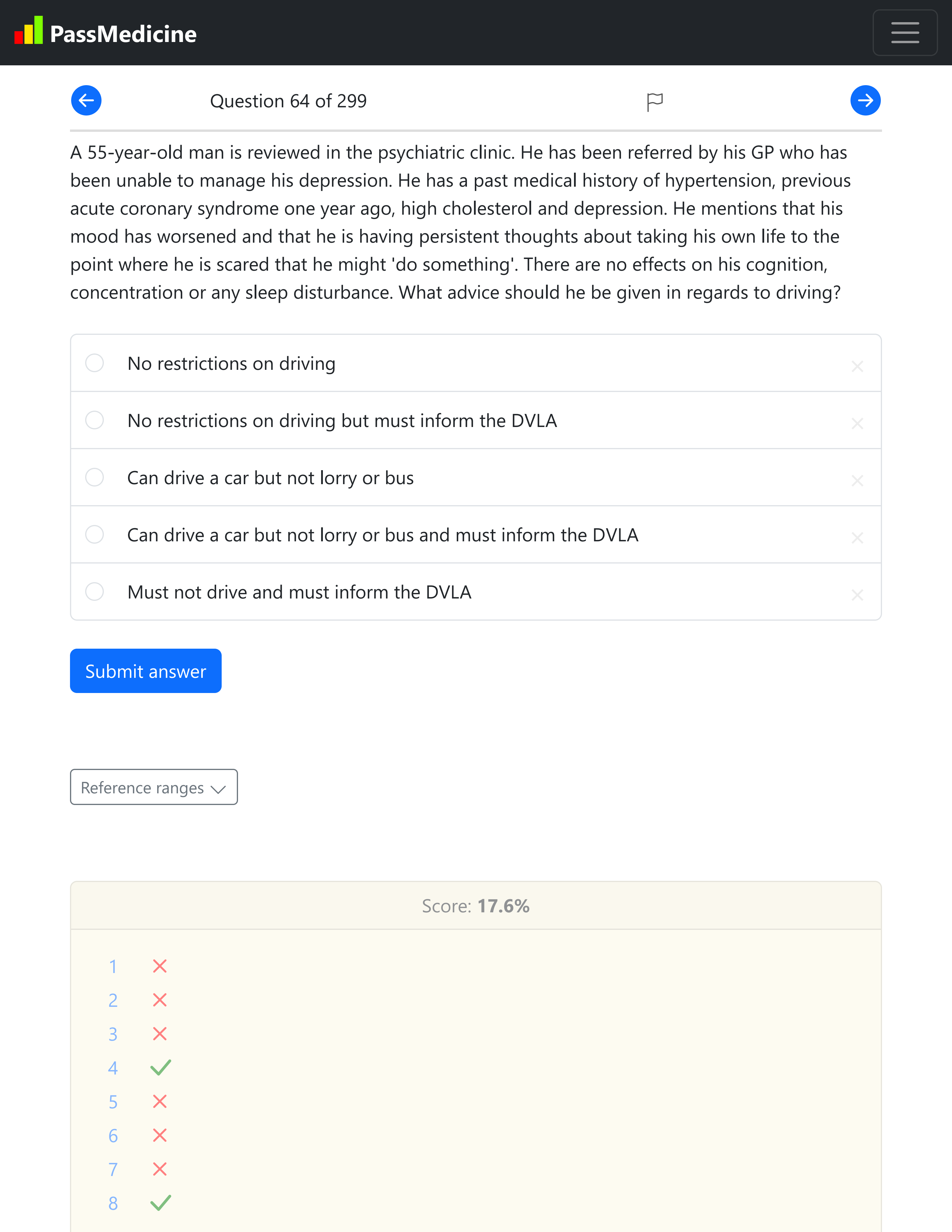

Question 64 of 299

A 55-year-old man is reviewed in the psychiatric clinic. He has been referred by his GP who has been unable to manage his depression. He has a past medical history of hypertension, previous acute coronary syndrome one year ago, high cholesterol and depression. He mentions that his mood has worsened and that he is having persistent thoughts about taking his own life to the point where he is scared that he might 'do something'. There are no effects on his cognition, concentration or any sleep disturbance. What advice should he be given in regards to driving?

- ○ No restrictions on driving ×
- ○ No restrictions on driving but must inform the DVLA ×
- ○ Can drive a car but not lorry or bus ×
- ○ Can drive a car but not lorry or bus and must inform the DVLA ×
- ○ Must not drive and must inform the DVLA ×

Score: **17.6%**

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No restrictions on driving	21%
No restrictions on driving but must inform the DVLA	13%
Can drive a car but not lorry or bus	5%
Can drive a car but not lorry or bus and must inform the DVLA	10%
Must not drive and must inform the DVLA	51%

The correct answer is that he must not drive and must inform the DVLA. He is a patient with depression and active suicidal thoughts and therefore should not drive.

Discuss (5) Improve

Next question

DVLA: psychiatric disorders

The below rules apply to group 1 vehicles (car and motorcycle), the group 2 (bus and lorry) rules are stricter.

Specific rules

- Severe anxiety or depression with any of the following: significant memory problems, significant concentration problems, agitation, behavioural disturbance or suicidal thoughts: must not drive and must notify the DVLA
- Acute psychotic disorder: must not drive during acute illness and must notify the DVLA
- Hypomania or mania: must not drive during acute illness and must notify the DVLA

- Schizophrenia: must not drive during acute illness and must notify the DVLA
- Pervasive developmental disorders and ADHD: may be able to drive but must inform the DVLA
- Mild cognitive impairment: may drive and need not inform the DVLA
- Dementia: may be able to drive but must inform the DVLA
- Mild learning disability: may be able to drive but must inform the DVLA
- Severe disability: must not drive and must notify the DVLA
- Personality disorders: may be able to drive but must inform the DVLA

Source:

'Assessing Fitness to Drive a Guide for Medical Professionals.' Driver and Vehicle Licensing Agency (2016): n. pag. Web. data/file/526635/assessing-fitness-to-drive-a-guide-for-medical-professionals.pdf>

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

DVLA 👍 0 👎 0

Fitness to drive guidelines

Suggest link Report broken link

Score: **20.5%**

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Question 65 of 299

A 72-year-old male presents to the Parkinson's clinic with his wife, reporting increasing frequency and duration of 'off' periods. The couple finds these episodes extremely debilitating and occurs up to 11 times a day. The patient was diagnosed with Parkinson's disease 12 years ago. As a relatively young patient on diagnosis, he was commenced on ropinirole, which he continued for 5 years, before being prescribed Sinemet 6 times a day and entacapone for the following 7 years. Over past 2 years, the 'off' episodes have gradually increased in frequency in addition to the development of very mild involuntary jaw movements. He is very low in mood and has presented to the emergency department with two episodes of attempted paracetamol overdoses. He would like a more effective treatment. What would you recommend?

- ○ Deep brain stimulation
- ○ Subcutaneous apomorphine
- ○ Reintroduce ropinirole at higher doses
- ○ Palliative care involvement
- ○ Trihexyl

Submit answer

Reference ranges

Score: **17.6%**

1 ✗
2 ✗
3 ✗
4 ✓
5 ✗

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Deep brain stimulation	36%
Subcutaneous apomorphine	27%
Reintroduce ropinirole at higher doses	19%
Palliative care involvement	3%
Trihexyl	15%

The treatment of 'off' symptoms is particularly important in patients who have been diagnosed with Parkinson's disease for a long time. The introduction of ropinirole would not help off periods. Trihexyl is an anti-cholinergic therapy for PD patients where tremor is the predominant symptom. In the context of suicidal behaviour, the patient would not be a candidate for deep brain stimulation, which for unknown reasons, increases the risk of suicide. Subcutaneous apomorphine is a reasonable option, administered as an infusion after a test bolus, which can be used to alleviate but not cure off symptoms.

Discuss (11) | Improve

Next question >

Parkinson's disease: management

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- ergot-derived dopamine receptor agonists (bromocriptine, cabergoline) have been associated with pulmonary, retroperitoneal and cardiac fibrosis. The Committee on Safety of Medicines advice that an echocardiogram, ESR, creatinine and chest x-ray should be obtained prior to treatment and patients should be closely monitored
- patients should be warned about the potential for dopamine receptor agonists to cause impulse control disorders and excessive daytime somnolence
- more likely than levodopa to cause hallucinations in older patients. Nasal congestion and postural hypotension are also seen in some patients

MAO-B (Monoamine Oxidase-B) inhibitors

- e.g. selegiline
- inhibits the breakdown of dopamine secreted by the dopaminergic neurons

Amantadine

- mechanism is not fully understood, probably increases dopamine release and inhibits its uptake at dopaminergic synapses
- side-effects include ataxia, slurred speech, confusion, dizziness and livedo reticularis

COMT (Catechol-O-Methyl Transferase) inhibitors

- e.g. entacapone, tolcapone
- COMT is an enzyme involved in the breakdown of dopamine, and hence may be used as an adjunct to levodopa therapy
- used in conjunction with levodopa in patients with established PD

Antimuscarinics

- block cholinergic receptors
- now used more to treat drug-induced parkinsonism rather than idiopathic Parkinson's disease
- help tremor and rigidity
- e.g. procyclidine, benzotropine, trihexyphenidyl (benzhexol)

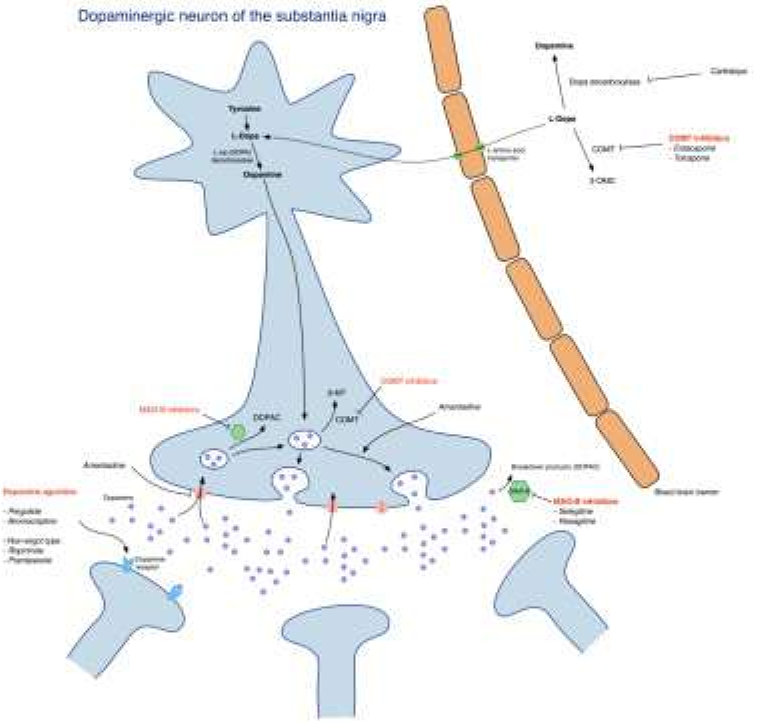

Dopaminergic neuron of the substantia nigra
Tyrosine
L-Dopa
Dopamine
Dopamine
L-Dopa
COMT
3-OMD
DOPAC
3-MT
COMT
Amantadine
Amantadine
Blood brain barrier

Image sourced from Wikipedia

Diagram showing the mechanism of action of Parkinson's drugs

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

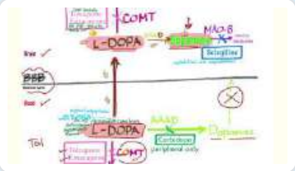
NICE 👍 23 👎 12

2017 Parkinson's disease guidelines

Suggest link

Report broken link

Media


COMT
MAO-B
L-DOPA
BBB
L-DOPA
COMT
Carbidopa

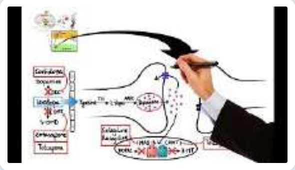
The use of Levodopa/ Carbidopa in the treatment of Parkinson's disease

Medicosis Perfectionalis - YouTube 👍 13 👎 0

PARKINSON'S
DISEASE
PHARMACOLOGY

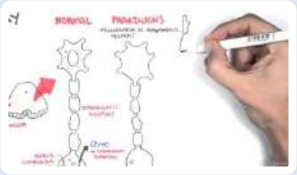
Parkinson's disease pharmacology

Soton Brain Hub - YouTube 👍 4 👎 0



Pharmacology - DRUGS FOR PARKINSON'S DISEASE

Speed Pharmacology - YouTube 👍 8 👎 1


NORMAL
PARKINSON'S

Pharmacology - Parkinson's Disease

Armando Hasudungan - YouTube 👍 16 👎 2

PARKINSON'S
DISEASE
Osmosis.org

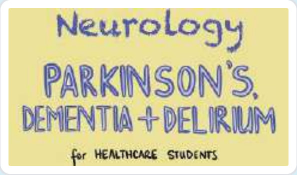
Parkinson's disease

Osmosis - YouTube 👍 4 👎 1

α-SYNUCLEIN
PARKINSON'S
DISEASE
SHUFFLING GAIT
PILL ROLLING TREMOR
COG-WHEEL RIGIDITY
DaT SCAN

Understanding Parkinson's Disease

Rhesus Medicine - YouTube 👍 2 👎 1


Neurology
PARKINSON'S,
DEMENTIA + DELIRIUM
for HEALTHCARE STUDENTS

Parkinson's disease

MedFlix - YouTube 👍 1 👎 2

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Score: **20.3%**

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Back to top

A 62-year-old male presents to the emergency department via blue light ambulance 90 minutes after sudden onset right sided weakness, expressive and receptive dysphasia. He is known to be T2DM, hypertensive on 3 agents and has a 40 pack year smoking history. On examination, you demonstrate a dense 0 out of 5 right hemiparesis. The patient has a complete expressive and receptive dysphasia, which you confirm to be new following a collateral history from his wife. The patient scores an NIHSS score of 7. He has no history of any recent surgery or head trauma. He is not on an anticoagulant and has no past medical history of coagulation disorders. His CT head demonstrates no areas of haemorrhage and a likely evolving area of ischaemic in the left middle cerebral artery territory. The radiologist calculated his ASPECT score to be 8. His observations on arrival are: temperature 36.7 degrees, heart rate 90/min and regular, blood pressure 220/150 mmHg, sats 99% on air, respiratory rate 20/min. What is the most appropriate first-line treatment?

- ○ IV thrombolysis ×
- ○ Aspirin 300mg OD ×
- ○ PO amlodipine 2.5mg ×
- ○ MRI head with diffusion weighting sequences ×
- ○ IV labetalol ×

Submit answer

Reference ranges

Score: **17.6%**

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IV thrombolysis	29%
Aspirin 300mg OD	20%
PO amlodipine 2.5mg	2%
MRI head with diffusion weighting sequences	3%
IV labetalol	45%

The patient has multiple vascular risk factors with a sudden onset negative speech and motor syndrome this is strongly suggestive of an acute vascular event. The patient has a likely left MCA territory ischaemic infarct and is within the time window for thrombolysis (<4.5 hours in accordance with ECASS 3 trial results[1]). The size of his infarct is also sufficiently small (calculated by ASPECT score, thrombolysis possible for all scores over 7) and his NIHSS score within range.

Thrombolysis is however not indicated in patients with blood pressures over 185/110 mmHg. Time is brain in the context of acute stroke the longer the delay to cerebral arterial reperfusion, the greater the size of infarct and subsequent functional deficits. The blood pressure is thus lowered rapidly with intravenous labetalol and not oral calcium channel blockers in this case.

1. Hacke W, Kaste M, Bluhmki E et al. Thrombolysis with alteplase 3 to 4.5 hours after acute ischemic stroke. N Engl J Med. 2008;359(13):1317

👍 👎 Discuss (18) Improve

Next question ›

Stroke: management of acute ischaemic stroke ★

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
  - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
  - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:

- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.

- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
    - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
    - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

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Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 11 👎 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 16

Stroke and TIA guidelines

Age and Ageing 👍 21 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

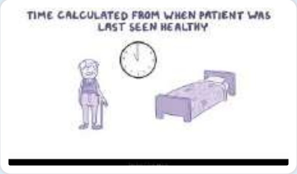
2023 National Clinical Guideline for Stroke

NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia
Acute Ischaemic Stroke

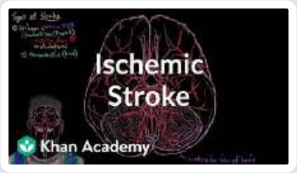
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

Khan Academy - YouTube 👍 1 👎 1

Report broken media

Score: **20.3%**

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A 59-year-old male presents with 4-day history of sudden onset visual disturbance in his right eye. He reports it to be his first episode with no previous history of visual problems. His past medical history includes type 2 diabetes mellitus, hypertension and raised BMI. He is an active smoker of 40 pack years.

On examination, a right relative afferent papillary defect is detected. Pupils were equal in size. A visual field defect is demonstrated in the inferior nasal field of the right eye without a precise quadrantanopia or altitudinal pattern. Temporal arteries are non-tender and not thickened. Visual acuity on Snellen chart in left eye was 6/6, 6/18 in right. Colour vision on Ishihara plates were 17/17 on left, 5/17 on right. Fundoscopy is unremarkable. Examination of the upper and lower limbs are unremarkable, no language deficits are noted. Auscultation revealed normal heart sounds and no bruits.

His blood tests are as follows:

Hb	154 g/l
Platelets	190 * 10^9/l
WBC	7.8 * 10^9/l
ESR 5 mm/hr	

Na^+	141 mmol/l
K^+	3.9 mmol/l
Urea	5.6 mmol/l
Creatinine	80 µmol/l

What is the most likely diagnosis?

○ Right retinal infarct ×

○ Right temporal arteritis ×

○ Right non-arteritic ischaemic optic neuropathy ×

○ Left middle cerebral artery ischaemic stroke ×

○ Left occipital ischaemic stroke ✗

Submit answer

Reference ranges ⌵

Score: **17.6%**

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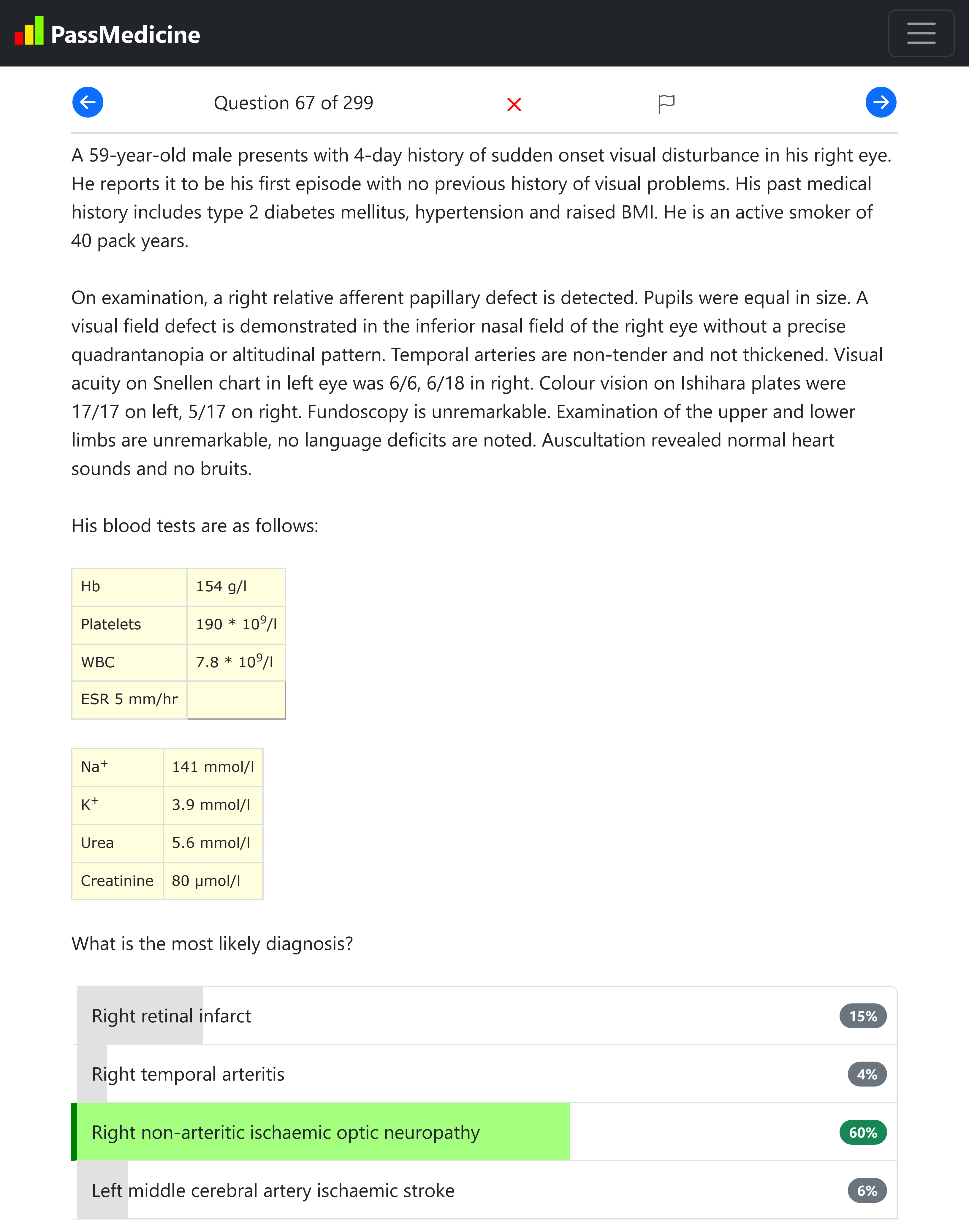

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On examination, a right relative afferent papillary defect is detected. Pupils were equal in size. A visual field defect is demonstrated in the inferior nasal field of the right eye without a precise quadrantanopia or altitudinal pattern. Temporal arteries are non-tender and not thickened. Visual acuity on Snellen chart in left eye was 6/6, 6/18 in right. Colour vision on Ishihara plates were 17/17 on left, 5/17 on right. Fundoscopy is unremarkable. Examination of the upper and lower limbs are unremarkable, no language deficits are noted. Auscultation revealed normal heart sounds and no bruits.

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ESR 5 mm/hr	

Na^+	141 mmol/l
K^+	3.9 mmol/l
Urea	5.6 mmol/l
Creatinine	80 µmol/l

What is the most likely diagnosis?

Right retinal infarct	15%
Right temporal arteritis	4%
Right non-arteritic ischaemic optic neuropathy	60%
Left middle cerebral artery ischaemic stroke	6%

Left occipital ischaemic stroke 15%

Visual field defects are a common presentation in clinical practice and MRCP part 2. The first feature to notice here is the monocular presentation, which indicates the lesion must be anterior to the optic chiasm. Next, fundoscopy is unremarkable, which would be unusual for a retinal infarct, most likely secondary to central or branch retinal artery occlusion.

Typically, an acute retinal infarct should reveal a classic cherry red spot, pale fundus and possible visualisation of the embolus. On locating the lesion to be at the optic nerve, the cause must be established: typically is this optic neuritis, arteritic or non-arteritic anterior ischaemic optic neuropathy. Optic neuropathy typically produces a correlated deficit in visual acuity with colour vision, allowing differentiation from optic neuritis, where colour vision is frequently better preserved.

Lastly, this patient has a normal ESR, is under 60 and has no local or systemic features of vasculitis, particularly temporal arteritis. In addition, he has multiple vascular risk factors, making a non-arteritic ischaemic anterior optic neuropathy most likely. Management involves optimisation of vascular risk factors and there is limited evidence for a tapering dose of oral prednisolone. Most patients can expect to improve by 3 lines of a Snellen chart by 6 months.

Discuss (8) Improve

Next question

Sudden loss of vision

Sudden loss is a frightening symptom for patients. It may represent an ongoing issue or only be temporary. The term transient monocular visual loss (TMVL) describes a sudden, transient loss of vision that lasts less than 24 hours.

The most common causes of a sudden painless loss of vision are as follows:
- ischaemic/vascular (e.g. thrombosis, embolism, temporal arteritis etc). This includes recognised syndromes e.g. occlusion of central retinal vein and occlusion of central retinal artery
- vitreous haemorrhage
- retinal detachment
- retinal migraine

Ischaemic/vascular

- often referred to as 'amaurosis fugax'
- wide differential including large artery disease (atherothrombosis, embolus, dissection), small artery occlusive disease (anterior ischemic optic neuropathy, vasculitis e.g. temporal arteritis), venous disease and hypoperfusion
- may represent a form of transient ischaemic attack (TIA). It should therefore be treated in a similar fashion, with aspirin 300mg being given
- altitudinal field defects are often seen: 'curtain coming down'
- ischaemic optic neuropathy is due to occlusion of the short posterior ciliary arteries, causing damage to the optic nerve

Central retinal vein occlusion

- incidence increases with age, more common than arterial occlusion
- causes: glaucoma, polycythaemia, hypertension
- severe retinal haemorrhages are usually seen on fundoscopy

Central retinal artery occlusion

- due to thromboembolism (from atherosclerosis) or arteritis (e.g. temporal arteritis)
- features include afferent pupillary defect, 'cherry red' spot on a pale retina

Vitreous haemorrhage

- causes: diabetes, bleeding disorders, anticoagulants
- features may include sudden visual loss, dark spots

Retinal detachment

- features of vitreous detachment, which may precede retinal detachment, include flashes of light or floaters (see below)

Differentiating posterior vitreous detachment, retinal detachment and vitreous haemorrhage

Posterior vitreous detachment	**Retinal detachment**	**Vitreous haemorrhage**
Flashes of light (photopsia) - in the peripheral field of vision Floaters, often on the temporal side of the central vision	Dense shadow that starts peripherally progresses towards the central vision A veil or curtain over the field of vision Straight lines appear curved Central visual loss	Large bleeds cause sudden visual loss Moderate bleeds may be described as numerous dark spots Small bleeds may cause floaters

+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

The Royal Australian College of General Practitioners 👍 16 👎 20

Sudden loss of vision review

Patient.info 👍 14 👎 11

Retinal artery occlusions

American Academy of Ophthalmology 👍 16 👎 10

Central Retinal Vein Occlusion

American Academy of Ophthalmology 👍 15 👎 7

Central Retinal Artery Occlusion

Suggest link Report broken link

Score: **20.3%**

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A 69-year-old male presenting with a 4-month history of progressive 'droopiness' of his left eyelid. He reports that in the last 2 weeks, he has been unable to lift his eyelid at all, which he says is fortunate considering he gets double vision now when he lifts his eyelid with his fingers. He underwent a renal transplant in 1988, which continues to function well, and previously had a squamous cell and malignant melanoma resected 2 and 5 years ago respectively.

On examination, you note complete ptosis, loss of vertical eye movements and loss of adduction in his left eye. On head tilt to the right, he is unable to depress left eye. His right eye demonstrates full eye movements. The left pupil is unreactive and larger in diameter compared to the right. There is also loss of sensation at the left nasal skin fold and left forehead with sparing of his left chin. He has no facial weakness, hearing loss or palatal asymmetry. He denies headache or retroorbital pain.

Where is the likely lesion?

Left ocular neuromuscular junction	6%
🟩 Left cavernous sinus	51%
Left superior orbital fissure	28%
Cerebral aqueduct	1%
🟥 Left midbrain	14%

The patient has deficits of his oculomotor nerve with parasympathetic involvement, involvement of the trochlear nerve and also the first and second branch of the trigeminal nerve, hence sparing of his left chin as supplied by the mandibular branch of the trigeminal nerve. Loss of pupillary reflexes in the left eye is a result of third nerve involvement and not necessarily retinal or optic nerve pathology. Ability to abduct the left eye suggests the abducens nerve is intact. The only anatomical location involving all these structures is the left cavernous sinus.

The possible cause for this cavernous sinus syndrome is interesting: the insidious gradual course goes against an acute cavernous sinus thrombus. A significant, expanding internal carotid aneurysm, which runs inside the cavernous sinus with the parasympathetic nerves wrapped around it, is a possibility. However, the key is the recognition that the patient is chronically immunosuppressed due to his renal transplant, increasing his risk of opportunistic infections such as fungi and also dermatological malignancies.

Discuss (8) | Improve

Next question ›

Third nerve palsy ★

Features
- eye is deviated 'down and out'
- ptosis
- pupil may be dilated (sometimes called a 'surgical' third nerve palsy)

Causes
- diabetes mellitus
- vasculitis e.g. temporal arteritis, SLE
- false localizing sign* due to uncal herniation through tentorium if raised ICP
- posterior communicating artery aneurysm
  - pupil dilated
  - often associated pain
- cavernous sinus thrombosis
- Weber's syndrome: ipsilateral third nerve palsy with contralateral hemiplegia -caused by midbrain strokes
- other possible causes: amyloid, multiple sclerosis

*this term is usually associated with sixth nerve palsies but it may be used for a variety of neurological presentations

+ Q 123

Next question ›

Textbooks

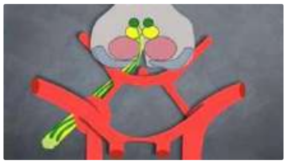
High-yield textbook

Extended textbook

Media

Third Cranial Nerve Palsy

U-M Kellogg Eye Center - YouTube 👍 6 👎 0



3rd nerve palsy

Individual - YouTube 👍 6 👎 1

OphthoBook.com

Cranial nerve palsies

Tim Root - YouTube 👍 5 👎 2

Report broken media

Score: **20.3%**

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A 66-year-old female is brought into Emergency Department by her distraught husband, who reports that she has not been out of bed for 3 days and seems unable to recognise him. He reports no recent fevers or rigors, she has not complained of cough, dysuria, sore throat or diarrhoea and vomiting prior to being unwell. She is well known to local neurologists: she is undergoing off-label rituximab for difficult to manage relapsing-remitting progressive multiple sclerosis, with previous white matter lesions in her corpus callosum, periventricular white matter, and lower thoracic transverse myelitis.

Over the past 6 years, she had continued to relapse despite interferon beta, natalizumab, mitoxantrone and cyclophosphamide. At baseline, she is weak in her lower limbs and is able to stand only with assistance of a turning apparatus, then able to walk about 3 steps. She is normally cognitively intact and fully orientated, normally responsible for organising the couples finances. Her past medical history includes type 1 diabetes mellitus, autoimmune thyroiditis and previous deep vein thrombosis 5 years ago (warfarin since discontinued). Her medication history includes lantus, novomix, levothyroxine and 3 monthly infusions of rituximab, which had stably maintained her MS for 4 years.

On examination, she is not orientated to time or place and appears extremely irritable. Halfway through the examination, she becomes extremely tearful. There is a full range of neck movements and no photophobia.

Neurological examination is difficult due to patient cooperation: pupils are equal and reactive, there is a full range of eye movements and no obvious facial asymmetry. Power in her upper limbs were at least 3/5, lower limbs are unable to counteract gravity. Both plantar responses are upgoing. Her bloods are as follows:

Hb	134 g/l
Platelets	268 * 10^9/l
WBC	7.8 * 10^9/l

Na^+	137 mmol/l
K^+	4.4 mmol/l
Urea	4.5 mmol/l
Creatinine	60 µmol/l
CRP	5 mg/l
HIV	negative

Glucose	7.4 mmol/l

A lumbar puncture was performed:

WBC	16 /mm^3
RBC	150 /mm^3
Protein	0.71 g/l
Glucose	3.5 mmol/l
PCR and gram staining	awaited

What is the most appropriate management step?

○ Intravenous aciclovir ×

○ Intravenous methylprednisolone ×

○ Intravenous immunoglobulin ×

○ Intravenous rituximab infusion ×

○ Psychiatric input ×

Submit answer

Reference ranges ⌄

Score: **17.6%**

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A lumbar puncture was performed:

WBC	16 /mm^3
RBC	150 /mm^3
Protein	0.71 g/l
Glucose	3.5 mmol/l
PCR and gram staining	awaited

What is the most appropriate management step?

Intravenous aciclovir	33%
Intravenous methylprednisolone	39%
Intravenous immunoglobulin	15%
Intravenous rituximab infusion	2%
Psychiatric input	10%

This is a difficult question regarding the side effect of rituximab and opportunistic infections. The neurological signs are difficult to interpret. Firstly, there are no hard clinical signs that her MS has deteriorated: her plantars are likely to be upgoing at baseline due to previous transverse myelitis while a symmetrical bilateral mild reduction in power is expected as a result of decompensation during acute illness. As a result, further aggressive immunosuppression would not be appropriate.

Rituximab results in B cell depletion through incompletely understood processes. An acute deterioration in cognition and agitation, in the context of an immunosuppressed, should raise strong suspicions of encephalitis or meningitis. The patient might not raise a strong immune response, hence relatively modest number of white cells in CSF and CRP, but the raised CSF protein would be consistent with viral encephalitis. A CSF:serum glucose of around 1:2 lowers the risk of bacterial CNS infection. The patient should be covered for possible herpes simplex or varicella-zoster encephalitis with intravenous aciclovir while awaiting CSF PCR.

👍 👎 Discuss (11) Improve

Next question ›

Multiple sclerosis: management ★

Treatment in multiple sclerosis is focused on reducing the frequency and duration of relapses. There is no cure.

Acute relapse

High-dose steroids (e.g. oral or IV methylprednisolone) may be given for 5 days to shorten the length of an acute relapse. It should be noted that steroids shorten the duration of a relapse and do not alter the degree of recovery (i.e. whether a patient returns to baseline function)

Disease modifying drugs

A number of drugs have been shown to reduce the risk of relapse in patients with MS. Typical indications for disease-modifying drugs include:
- relapsing-remitting disease + 2 relapses in past 2 years + able to walk 100m unaided
- secondary progressive disease + 2 relapses in past 2 years + able to walk 10m (aided or unaided)

Drug options for reducing the risk of relapse in MS
- natalizumab
  - a recombinant monoclonal antibody that antagonises alpha-4 beta-1-integrin found on the surface of leucocytes
  - inhibit migration of leucocytes across the endothelium across the blood-brain barrier
  - generally considered to have the strongest evidence base for preventing relapse of the disease-modifying and hence is often used first-line
  - given intravenously
- ocrelizumab
  - humanized anti-CD20 monoclonal antibody
  - like natalizumab, it is considered a high-efficacy drug that is often used first-line
  - given intravenously
- fingolimod
  - sphingosine 1-phosphate (S1P) receptor modulator
  - prevents lymphocytes from leaving lymph nodes
  - oral formulations are available
- beta-interferon
  - not considered to be as effective as alternative disease-modifying drugs
  - given subcutaneously/intramuscularly
- glatiramer acetate

    - immunomodulating drug - acts as an 'immune decoy'
    - given subcutaneously
    - along with beta-interferon considered an 'older drug' with less effectiveness compared to monoclonal antibodies and S1P) receptor modulators

Some specific problems

Fatigue

- once other problems (e.g. anaemia, thyroid or depression) have been excluded NICE recommend a trial of amantadine
- other options include mindfulness training and CBT

Spasticity

- baclofen and gabapentin are first-line. Other options include diazepam, dantrolene and tizanidine
- physiotherapy is important
- cannabis and botox are undergoing evaluation

Bladder dysfunction

- may take the form of urgency, incontinence, overflow etc
- guidelines stress the importance of getting an ultrasound first to assess bladder emptying - anticholinergics may worsen symptoms in some patients
- if significant residual volume → intermittent self-catheterisation
- if no significant residual volume → anticholinergics may improve urinary frequency

Oscillopsia (visual fields appear to oscillate)

- gabapentin is first-line

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Multiple Sclerosis Society 👍 6 👎 14

Primary care guidelines

NICE 👍 15 👎 7

2014 Multiple Sclerosis guidelines

Suggest link Report broken link

Media

Multiple sclerosis

Podmedics - YouTube 👍 4 👎 0

Multiple Sclerosis

Multiple sclerosis

Soton Brain Hub - YouTube 👍 3 👎 0

DEMYELINATION
T-CELLS
PLAQUES
MULTIPLE SCLEROSIS
OLIGO CLONAL BANDS
RELAPSING-REMITTING

Multiple sclerosis

Rhesus Medicine - YouTube

👍 1 👎 0

MULTIPLE SCLEROSIS

Multiple sclerosis - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 7 👎 1

Report broken media

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A 57-year-old male presents with a 3 months history of increasing clumsiness in his hands and arms. He has a complicated past medical history: 22 years ago, he underwent a renal transplant after a progressive deterioration in his renal function following diagnosis with autosomal dominant polycystic kidney disease aged 25. His transplant has functioned well since but the patient has since undergone two resections of squamous cell carcinomas and one serious lengthy hospital admission for a systemic fungal infection. He stopped working a year ago as a wine merchant, after complaining that he was no longer able to differentiate 'the smells of his wines as he got older'.

On examination, he is alert and orientated. Questioning was challenging due to his hearing impairment, despite bilateral hearing aids. He scored 17/30 on a mini-mental examination. Pupils were reactive with a full range of eye movements. Facial power and sensation were normal, with symmetrical palatal elevation and no tongue deviation. He was profoundly deaf bilaterally. Tone, power and sensation were normal, reflexes were present with downgoing plantars. However, you note significant bilateral finger-nose dysmetria and heel-shin mal co-ordination.

Which investigation is likely to produce the unifying diagnosis?

○ Lumbar puncture for cerebrospinal fluid including 14-3-3, S100

○ EEG

○ MRI head

○ Neurogenetic testing

○ Nerve conduction studies and EMG

Submit answer

Reference ranges

Score: **17.6%**

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Which investigation is likely to produce the unifying diagnosis?

Lumbar puncture for cerebrospinal fluid including 14-3-3, S100	29%
EEG	1%
MRI head	32%
Neurogenetic testing	34%
Nerve conduction studies and EMG	3%

The patient has presented with anosmia, bilateral symmetrical ataxia, cognitive impairment and sensorineural hearing loss, classical symptoms of superficial siderosis, which would be demonstrated on MRI head, with siderosis typically prominent in the posterior fossa. The underlying cause for iron deposition is thought to be due to chronic or previous intracerebral haemorrhages. In the case of this patient, the association of polycystic kidney disease with brain aneurysms are likely to be significant. In other patients, previous neurosurgery or head trauma can similarly result in siderosis. There is no current treatment for superficial siderosis but case reports have demonstrated possible improvements with lipid-soluble iron chelators.

Discuss (19) Improve

Next question

Superficial siderosis

Superficial siderosis describes the chronic deposition of iron in neurons of the central nervous system. The most common cause is chronic bleeding secondary to either a subarachnoid haemorrhage or subdural haemorrhage.

Features
- sensorineural hearing loss
- ataxia
- dementia
- anosmia
- anisocoria

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **20.3%**

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Question 71 of 299

A 78-year-old male presents to you with progressive weakness in his arms. He first saw his GP about 4 months ago complaining of a weakness in his right wrist and was told he had carpal tunnel syndrome, for which he was placed on a waiting list to see the orthopaedic surgeons. Over the next 4 months, he noticed progressive weakness in his right wrist and in bending his right elbow. He noted his right hand was becoming increasingly wasted. Two months ago, he noted similar weakness in his left wrist. He reports no changes to sensation, normal speech and swallowing. On examination, cranial nerves were unremarkable.

Both hands were markedly wasted with significant loss of musculature and fasciculations bilaterally. Reflexes were muted in both upper limbs with negative Hoffman's sign. Sensory examination in the upper limbs were unremarkable, he was unable to perform the finger-nose examination. Examination of the lower limbs was also unremarkable but the patient felt a subjective weakness in both hip flexion. MRI imaging of the patients head and spine demonstrated no significant lesions. Nerve conduction studies demonstrated f waves and H-reflexes, multiple focal areas of demyelination and motor neuropathic blocks.

What is the most appropriate management?

○ Intravenous cyclophosphamide

○ Intravenous immunoglobulin

○ Intravenous methylprednisolone

○ Plasma exchange

○ Riluzole

Submit answer

Reference ranges

Score: **17.6%**

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A 78-year-old male presents to you with progressive weakness in his arms. He first saw his GP about 4 months ago complaining of a weakness in his right wrist and was told he had carpal tunnel syndrome, for which he was placed on a waiting list to see the orthopaedic surgeons. Over the next 4 months, he noticed progressive weakness in his right wrist and in bending his right elbow. He noted his right hand was becoming increasingly wasted. Two months ago, he noted similar weakness in his left wrist. He reports no changes to sensation, normal speech and swallowing. On examination, cranial nerves were unremarkable.

Both hands were markedly wasted with significant loss of musculature and fasciculations bilaterally. Reflexes were muted in both upper limbs with negative Hoffman's sign. Sensory examination in the upper limbs were unremarkable, he was unable to perform the finger-nose examination. Examination of the lower limbs was also unremarkable but the patient felt a subjective weakness in both hip flexion. MRI imaging of the patients head and spine demonstrated no significant lesions. Nerve conduction studies demonstrated f waves and H-reflexes, multiple focal areas of demyelination and motor neuropathic blocks.

What is the most appropriate management?

Intravenous cyclophosphamide	2%
🟩 Intravenous immunoglobulin	29%
Intravenous methylprednisolone	26%
Plasma exchange	3%
🟥 Riluzole	40%

The patient has presented with a pure motor peripheral neuropathy with multifocal areas of demyelination and motor block, with f waves and H-reflexes suggestive of late motor responses to electrical stimuli, suggestive of multifocal motor neuropathy with conduction block (MMN-CB). MMN-CB is an immune-mediated disorder, commonly associated with anti-GM1 antibodies, typically presenting with asymmetric bi-brachial motor weakness without sensory or bulbar involvement, allowing the key distinction from motor neurone disease. MMN-CB is not responsive to steroids or plasma exchange, unlike chronic demyelinating polyneuropathy (CIDP), which typically involves sensory nerves too.

Cyclophosphamide may have some efficacy but is generally not used in MMN-CB due to its toxicity profile.

Riluzole is a drug for motor neurone disease.

👍 👎 Discuss (4) Improve

Next question >

Multifocal motor neuropathy with conduction block ★

Multifocal motor neuropathy with conduction block (MMN-CB) is an acquired immune-mediated demyelinating neuropathy. Features of MMN-CB may resemble motor neuron disease but in contrast MMN-CB may respond well to intravenous immunoglobulin.

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Score: **20.3%**

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Question 72 of 299

A 45-year-old female presents with small multiple ischaemic infarcts in the right cerebellar hemisphere confirmed on MRI, associated with small haemorrhages within two areas of infarct. A subsequent CT angiogram 24 hours into the admission demonstrated a right vertebral artery dissection with a free flowing thrombus visualised. What is the optimal management?

- ○ IV heparin infusion
- ○ 300mg aspirin 14 days followed by clopidogrel 75mg
- ○ Load with warfarin
- ○ Treatment dose low molecular heparin
- ○ Hold off antiplatelet and anticoagulation

Submit answer

Reference ranges

Score: **17.6%**

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Question 72 of 299

A 45-year-old female presents with small multiple ischaemic infarcts in the right cerebellar hemisphere confirmed on MRI, associated with small haemorrhages within two areas of infarct. A subsequent CT angiogram 24 hours into the admission demonstrated a right vertebral artery dissection with a free flowing thrombus visualised. What is the optimal management?

✓ IV heparin infusion	24%
300mg aspirin 14 days followed by clopidogrel 75mg	31%
Load with warfarin	2%
Treatment dose low molecular heparin	16%
✗ Hold off antiplatelet and anticoagulation	27%

It is generally accepted that visualised thrombus requires anticoagulation to prevent it increasing in size and consequent possible embolic showering. However, the patient is still in the hyperacute phase with haemorrhagic transformation within small areas of infarct. The optimal management in this scenario is to offer anticoagulation with the greatest reversibility should intracranial haemorrhage occurs: intravenous heparin is tricky to manage but can be reversed the fastest compared to subcutaneous low molecular weight heparin and warfarin. The patient will require close hourly neurological monitoring and urgent repeat imaging if GCS decreases or new or deteriorating focal neurology presents.

Discuss (10) | Improve

Next question

Stroke: management of acute ischaemic stroke

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
    - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
    - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:

- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.

- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
    - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
    - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and

- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):
- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:
- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled
- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%
- it should be performed as soon as possible within 7 days

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 11 👎 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 16

Stroke and TIA guidelines

Age and Ageing 👍 21 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

2023 National Clinical Guideline for Stroke

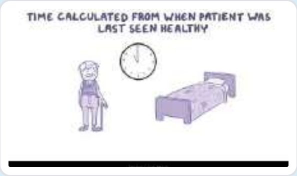
NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link

Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia
Acute Ischaemic Stroke

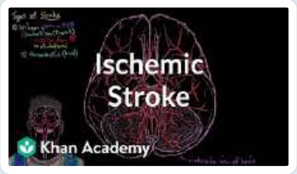
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

Khan Academy - YouTube 👍 1 👎 1

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Score: **20.3%**

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A 65-year-old lady was referred to the neurology clinic for assessment. She reported a 3-month history of progressive weakness. She had initially noticed difficulty opening jars but reported that over the past month she had also had difficulty walking up stairs. She did not have any pain and reported no changes in sensation. Past medical history included osteoporosis, type 2 diabetes mellitus and hypertension.

On neurological examination there were no fasciculations, the tone was normal and the sensation was intact. Power was reduced in finger flexion (3/5), wrist flexion (4/5), knee extension (3/5) and hip flexion (4/5) bilaterally. Other movements were relatively spared. Upper limb reflexes were present but diminished, but the knee jerk was absent. The plantar response was flexor bilaterally. There was no tenderness over any muscle groups. Cranial nerve examination was unremarkable.

Blood results are shown below:

Haemoglobin	122 g/l
White cell count	8.2 x 10^9/l
Platelets	376 x 10^9/l
C reactive protein	7 mg/l
Erythrocyte sedimentation rate	39 mm/hr
Creatine kinase	272 (24-170 U/l)

What is the most likely diagnosis?

○ Diabetic amyotrophy ×

○ Inclusion body myositis ×

○ Motor neuron disease ×

○ Myasthenia gravis ×

○ Polymyositis ×

Submit answer

Reference ranges

Score: **17.6%**

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Erythrocyte sedimentation rate	39 mm/hr
Creatine kinase	272 (24-170 U/l)

What is the most likely diagnosis?

Diabetic amyotrophy	12%
Inclusion body myositis	54%
Motor neuron disease	14%
Myasthenia gravis	3%
Polymyositis	17%

Inclusion body myositis is the most common primary myopathy in this age group. It manifests as a slowly progressive weakness, usually affecting finger and wrist flexion initially. Lower limb weakness may also occur. Reflexes are usually diminished as in other myopathies. Creatine kinase levels are usually normal or only mildly raised, in contrast to polymyositis (where creatine kinase levels are usually markedly elevated). Muscles are often tender in polymyositis, and the distal muscles are usually not affected until the disease is advanced.

Motor neuron disease is an important differential to consider, but the lack of upper motor neuron signs and fasciculations goes against this. There is no description of fatigability that would lead one to consider myasthenia gravis, and no evidence of bulbar, facial or ocular weakness. Diabetic amyotrophy is characterised by painful wasting of the proximal lower limb muscles.

👍 👎 Discuss (3) Improve

Next question ›

Inclusion body myositis ★

Overview
- cause of myopathy
- associated with cytoplasmic inclusions on muscle biopsy

Features
- typically affects older males
- can affect both proximal and distal muscles
- characteristically affects quadriceps and finger/wrist flexors are usually more severely affected than extensor counterparts

+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Media

INCLUSION BODY MYOPATHY

Inclusion body myositis

Osmosis - YouTube 👍 7 👎 2

Report broken media

Score: **20.3%**

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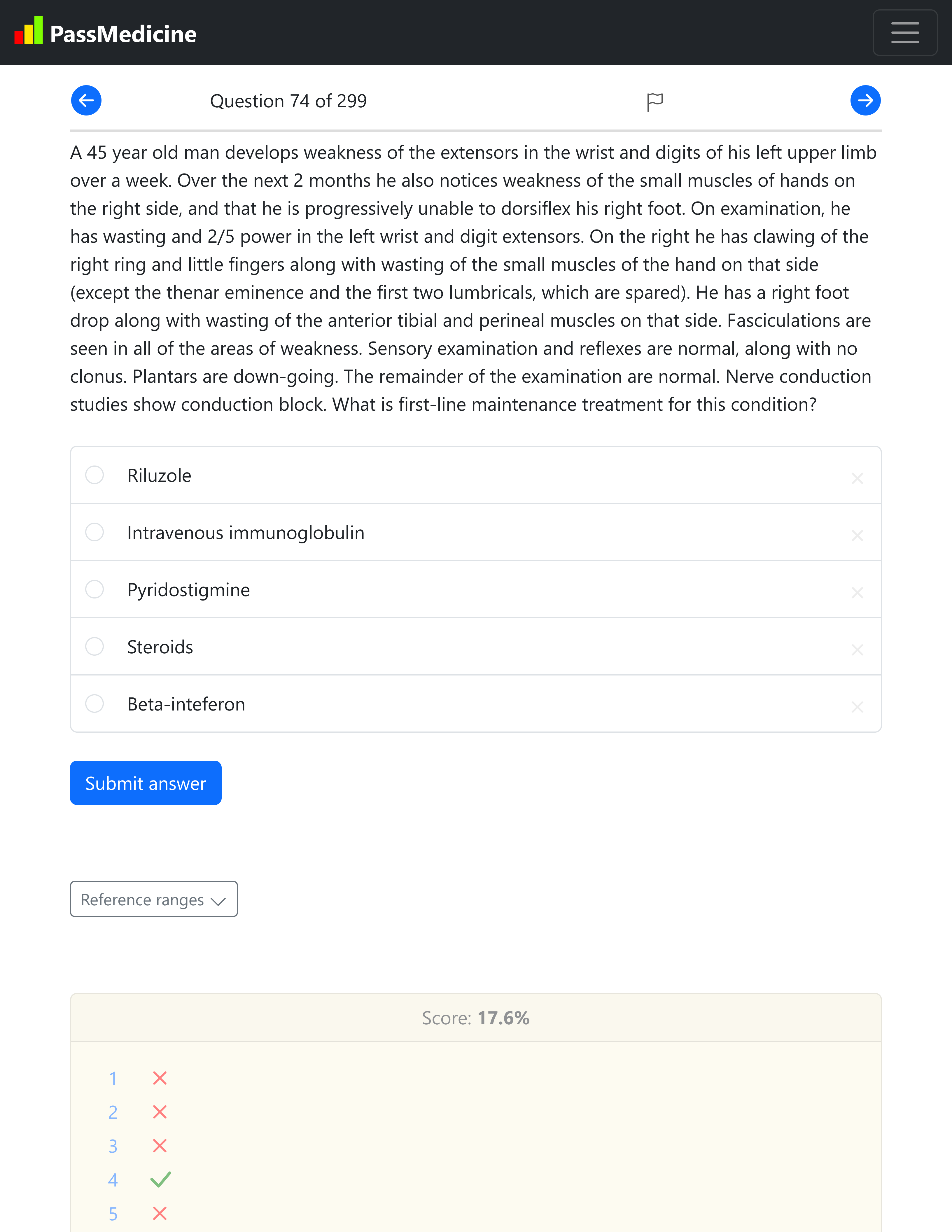

A 45 year old man develops weakness of the extensors in the wrist and digits of his left upper limb over a week. Over the next 2 months he also notices weakness of the small muscles of hands on the right side, and that he is progressively unable to dorsiflex his right foot. On examination, he has wasting and 2/5 power in the left wrist and digit extensors. On the right he has clawing of the right ring and little fingers along with wasting of the small muscles of the hand on that side (except the thenar eminence and the first two lumbricals, which are spared). He has a right foot drop along with wasting of the anterior tibial and perineal muscles on that side. Fasciculations are seen in all of the areas of weakness. Sensory examination and reflexes are normal, along with no clonus. Plantars are down-going. The remainder of the examination are normal. Nerve conduction studies show conduction block. What is first-line maintenance treatment for this condition?

- ○ Riluzole
- ○ Intravenous immunoglobulin
- ○ Pyridostigmine
- ○ Steroids
- ○ Beta-inteferon

Score: **17.6%**

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Riluzole	55%
Intravenous immunoglobulin	24%
Pyridostigmine	4%
Steroids	14%
Beta-inteferon	4%

The answer here is the condition called multifocal motor neuropathy with conduction block (MMNCB). These patients are usually younger to middle-aged men who develop focal arm weakness in the distribution of a named nerve. It usually happens quite suddenly (e.g. over a week) and they are often mistaken as a stroke at first presentation. However, over several months additional named motor nerves become involved asymmetrically such that MMNCB may eventually look like motor neurone disease (MND) - the principle differential here (specifically the lower motor neurone progressive muscular atrophy form rather than the mixed upper/lower motor neurone ALS form). For exam purposes, the key difference between these two conditions is the nerve conduction studies. MMNCB shows conduction block. MND does not. MMNCB is a demyelinating condition, much in the same way Guillain Barre or chronic inflammatory demyelinating polyneuropathy (CIDP) are. However, in MMNCB this demyelination is in segments of a nerve rather than affecting the whole nerve. Therefore, the action potentials (as well as being slowed because of demyelination) actually don't get past the areas of conduction block. The importance of doing nerve conduction studies is to decide whether a motor neuropathy is axonal (i.e. the axon itself getting damaged) or demyelinating. The demyelinating ones tend to be more easily and similarly treatable. MMNCB, Guillain Barre, and CIDP are all good examples of demyelinating neuropathies, all of which therefore respond to intravenous immunoglobulin (IVIG).

Note that the nerves involved in this particular case are the left radial nerve, right ulnar nerve, and right common perineal nerve.

Reference: Andrew Tarulli. Neurology: A Clinician's Approach. Cambridge University Press; 1 edition (18 Nov. 2010)

👍 👎 Discuss (18) Improve

Next question >

Multifocal motor neuropathy ★

Acquired autoimmune demyelinating motor neuropathy

Associated with motor conduction block

Slowly progressive, distal motor neuropathy which progresses over many years

Anti-GM1 antibodies frequently raised

AI + Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Score: **20.3%**

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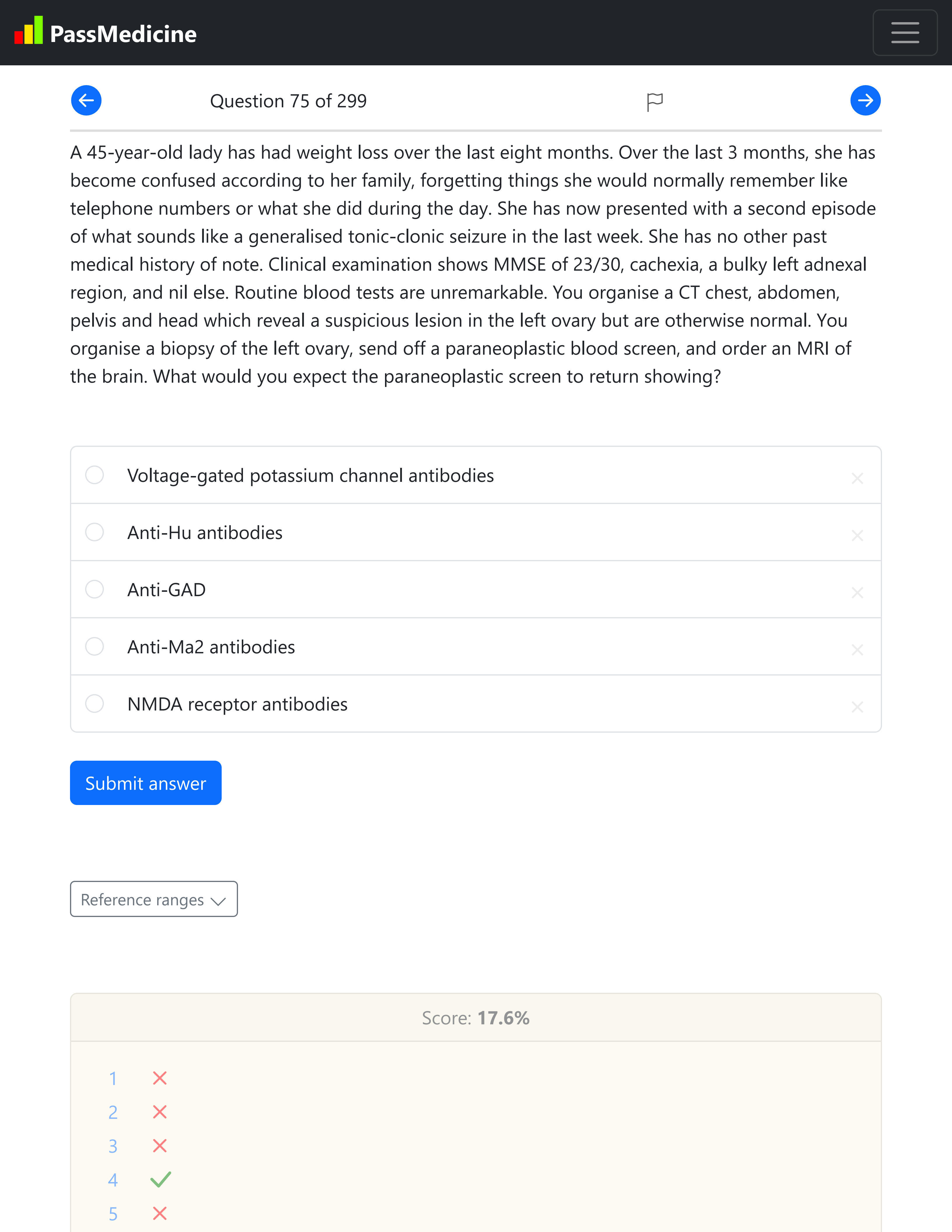

A 45-year-old lady has had weight loss over the last eight months. Over the last 3 months, she has become confused according to her family, forgetting things she would normally remember like telephone numbers or what she did during the day. She has now presented with a second episode of what sounds like a generalised tonic-clonic seizure in the last week. She has no other past medical history of note. Clinical examination shows MMSE of 23/30, cachexia, a bulky left adnexal region, and nil else. Routine blood tests are unremarkable. You organise a CT chest, abdomen, pelvis and head which reveal a suspicious lesion in the left ovary but are otherwise normal. You organise a biopsy of the left ovary, send off a paraneoplastic blood screen, and order an MRI of the brain. What would you expect the paraneoplastic screen to return showing?

- ○ Voltage-gated potassium channel antibodies
- ○ Anti-Hu antibodies
- ○ Anti-GAD
- ○ Anti-Ma2 antibodies
- ○ NMDA receptor antibodies

Score: **17.6%**

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Question 75 of 299 ✓

A 45-year-old lady has had weight loss over the last eight months. Over the last 3 months, she has become confused according to her family, forgetting things she would normally remember like telephone numbers or what she did during the day. She has now presented with a second episode of what sounds like a generalised tonic-clonic seizure in the last week. She has no other past medical history of note. Clinical examination shows MMSE of 23/30, cachexia, a bulky left adnexal region, and nil else. Routine blood tests are unremarkable. You organise a CT chest, abdomen, pelvis and head which reveal a suspicious lesion in the left ovary but are otherwise normal. You organise a biopsy of the left ovary, send off a paraneoplastic blood screen, and order an MRI of the brain. What would you expect the paraneoplastic screen to return showing?

Voltage-gated potassium channel antibodies	4%
Anti-Hu antibodies	27%
Anti-GAD	7%
Anti-Ma2 antibodies	5%
NMDA receptor antibodies	57%

The diagnosis is limbic encephalitis. Limbic encephalitis represents a group of autoimmune conditions characterised by inflammation of the limbic system and other parts of the brain. People with limbic encephalitis typically presents with a subacute development of memory impairment, confusion, and alteration of consciousness, often accompanied by seizures and temporal lobe signal change on MRI.

It is important to diagnose the underlying autoimmune aetiology of limbic encephalitis as this might point you toward the underlying diagnosis of cancer. This is to say that limbic encephalitis may often present as a paraneoplastic syndrome, although not always. The antibodies that cause limbic encephalitis and their associated malignancies are:

NMDA receptor antibodies = ovarian cancer
Voltage-gated potassium channel antibodies = Thymoma or small cell lung cancer
Anti-Hu antibodies = Small cell lung cancer
Anti-GAD = Thymoma

anti-Ma2 antibodies = Germ-cell tumours of testis, non-small cell lung cancer

👍 👎 Discuss (6) Improve

Next question >

Autoimmune encephalitis ★

Autoimmune encephalitis (AE) is a group of rare and potentially severe neurological disorders characterized by inflammation of the brain caused by an aberrant immune response. The immune system mistakenly targets and attacks healthy brain tissue, resulting in various neurological and psychiatric symptoms.

Types of autoimmune encephalitis include:

- autoimmune limbic encephalitis (paraneoplastic and non-paraneoplastic)
- Rasmussen's encephalitis
- anti-NMDAR (NR1) encephalitis
- glycine-receptor mediated encephalitis
- Bickerstaff brainstem encephalitis

Features

- Cognitive dysfunction: Memory impairment, confusion, and disorientation
- Seizures: Focal or generalized
- Movement disorders: Dystonia, chorea, or myoclonus
- Psychiatric symptoms: Anxiety, depression, hallucinations, or psychosis
- Autonomic dysfunction: Fluctuating blood pressure, heart rate irregularities, or temperature dysregulation
- Sleep disturbances: Insomnia or hypersomnia

Investigation

- Detection of specific autoantibodies in serum or cerebrospinal fluid (CSF)
- Neuroimaging: MRI to identify areas of inflammation or other abnormalities
- Electroencephalogram (EEG): To detect seizure activity or other abnormal brain wave patterns
- Additional tests: Screening for underlying malignancies, infections, or other autoimmune diseases

Management

- High-dose corticosteroids

- Intravenous immunoglobulin
- Plasma exchange (plasmapheresis)

123

Next question

Textbooks

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Extended textbook

Score: **20.3%**

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A 47 year old female with relapsing-remitting multiple sclerosis (MS) describes increasing difficulty with fatigue, and has had to give up her work as a medical secretary. She has always been careful about ensuring a balanced diet, gentle exercise and early treatment of infection. On review and questioning her mood is good. Her most recent bloods are shown below:

Hb	120g/dl
Platelets	150 * 10^9/l
WBC	5.2 * 10^9/l

Na	130 mmol/l
K	3.8 mmol/l
Urea	6.0 mmol/l
Creatinine	68 µmol/l

B12, folate and TFTs are normal.

What is the best course of action?

Advise that she increase caffeine intake	2%
Advise that increasing exercise may benefit	16%
Trial of vitamin D	24%
Trial of amantadine	49%
Trial of ropinirole	9%

The first step in her management should be to exclude anaemia, thyroid dysfunction, sleep or mood disturbance. With the above results and knowledge that she is living a well balanced lifestyle, the next step would be a trial of amantadine.

Amantadine is an anti-viral agent that slows viral replication. It is also known to have some dopaminergic effects; the mechanism of action for reducing fatigue in MS however, remains unclear. It is currently recommended by NICE as a potential treatment, although a Cochrane review in 2006 was inconclusive.

Increasing caffeine intake and levels of exercise would not be recommended in this patient. Similarly, vitamin D has not be proven to be of benefit in MS. Ropinirole is a dopamine agonist used in parkinsons disease but not in MS.

👍 👎 Discuss (6) Improve

Next question >

Multiple sclerosis: management ★

Treatment in multiple sclerosis is focused on reducing the frequency and duration of relapses. There is no cure.

Acute relapse

High-dose steroids (e.g. oral or IV methylprednisolone) may be given for 5 days to shorten the length of an acute relapse. It should be noted that steroids shorten the duration of a relapse and do not alter the degree of recovery (i.e. whether a patient returns to baseline function)

Disease modifying drugs

A number of drugs have been shown to reduce the risk of relapse in patients with MS. Typical indications for disease-modifying drugs include:

- relapsing-remitting disease + 2 relapses in past 2 years + able to walk 100m unaided
- secondary progressive disease + 2 relapses in past 2 years + able to walk 10m (aided or unaided)

Drug options for reducing the risk of relapse in MS

- natalizumab
    - a recombinant monoclonal antibody that antagonises alpha-4 beta-1-integrin found on the surface of leucocytes
    - inhibit migration of leucocytes across the endothelium across the blood-brain barrier
    - generally considered to have the strongest evidence base for preventing relapse of the disease-modifying and hence is often used first-line
    - given intravenously
- ocrelizumab

    - humanized anti-CD20 monoclonal antibody
    - like natalizumab, it is considered a high-efficacy drug that is often used first-line
    - given intravenously
- fingolimod
    - sphingosine 1-phosphate (S1P) receptor modulator
    - prevents lymphocytes from leaving lymph nodes
    - oral formulations are available
- beta-interferon
    - not considered to be as effective as alternative disease-modifying drugs
    - given subcutaneously/intramuscularly
- glatiramer acetate
    - immunomodulating drug - acts as an 'immune decoy'
    - given subcutaneously
    - along with beta-interferon considered an 'older drug' with less effectiveness compared to monoclonal antibodies and S1P) receptor modulators

Some specific problems

Fatigue

- once other problems (e.g. anaemia, thyroid or depression) have been excluded NICE recommend a trial of amantadine
- other options include mindfulness training and CBT

Spasticity

- baclofen and gabapentin are first-line. Other options include diazepam, dantrolene and tizanidine
- physiotherapy is important
- cannabis and botox are undergoing evaluation

Bladder dysfunction

- may take the form of urgency, incontinence, overflow etc
- guidelines stress the importance of getting an ultrasound first to assess bladder emptying - anticholinergics may worsen symptoms in some patients
- if significant residual volume → intermittent self-catheterisation
- if no significant residual volume → anticholinergics may improve urinary frequency

Oscillopsia (visual fields appear to oscillate)

- gabapentin is first-line

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Multiple Sclerosis Society 👍 6 👎 14

Primary care guidelines

NICE 👍 15 👎 7

2014 Multiple Sclerosis guidelines

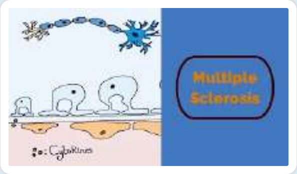
Suggest link

Report broken link

Media

Multiple sclerosis

Podmedics - YouTube 👍 4 👎 0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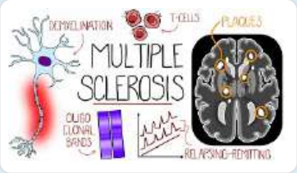
DEMYELINATION
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MULTIPLE SCLEROSIS
OLIGO CLONAL BANDS
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Multiple sclerosis

Rhesus Medicine - YouTube 👍 1 👎 0

MULTIPLE SCLEROSIS

Multiple sclerosis - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 7 👎 1

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Score: **20.3%**

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A 26-year-old female from the travelling community presents to A&E following a seizure. History from her family suggests that she was suffering with headaches over the last 24 hours and this morning was feverish and vomiting. Her partner says she was unwell 1 month ago with a fever and whole body rash that spontaneously resolved. There is no other significant past medical history.

On examination she appears drowsy. She has a left sided hemiparesis with bilateral nystagmus. Fundoscopy reveals papiloedema. There are no skin rashes.

What is the most likely underlying diagnosis?

Meningococcal septicaemia	16%
Meningitis secondary to herpes simplex virus	17%
Progressive multifocal leukoencephalopathy (PML)	13%
Multiple sclerosis	1%
Acute disseminated encephalomyelitis (ADEM)	53%

In the case given there is no history of preceding neurological symptoms and the acute onset of the neurology points away from the diagnosis of MS. In the examination findings it is specified there is no rash making meningococcal septicaemia very unlikely. PML is only seen with significant immunosupression and presents with a subacute picture. This leaves HSV as a plausible diagnosis however ADEM secondary to measles is more likely given the previous full body rash in a member of the travelling community who may not have been vaccinated.

Discuss (5) Improve

Next question >

Acute disseminated encephalomyelitis ★

Acute disseminated encephalomyelitis (ADEM) is an autoimmune demyelinating disease of the central nervous system. It may also be termed post-infectious encephalomyelitis. The aetiology is not fully understood and it can occur following infection with a bacterial or viral pathogen. Common infections include measles, mumps, rubella and varicella, however this list is not exhaustive.

After a lag time of between a few days to 2 months, there is an acute onset of multifocal neurological symptoms with rapid deterioration. Non-specific signs such as headache, fever, nausea and vomiting may also accompany the onset of illness. Motor and sensory deficits are frequent and there may also be brainstem involvement including oculomotor defects.

There are no specific biomarkers for the diagnosis of ADEM. MRI imaging may show areas of supra and infra-tentorial demyelination. Management involves intravenous glucocorticoids and the consideration of IVIG where this fails.

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20.3%**

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A 65-year-old man, originally from Pakistan is referred to the rapid access chest clinic by his GP with a one month history of weight loss (of roughly 5 kg) and a dry, irritating cough. Over the past two days he has started to have loss of sensation in both of his feet and lower legs.

Examination reveals heart sounds 1 and 2 present with no added sounds, reduced air entry in the mid and lower left zones of the chest, a non-tender liver edge that extends 3cm below the costal margin and loss of sensation for pain, light touch and temperature in both feet up to the mid shin.

A chest X-ray is performed which shows a unilateral hilar mass, but no other consolidation or features. His past medical history includes mild hypertension and diabetes mellitus which is diet-controlled. His only medication is enalapril for his hypertension. Bloods reveal no obvious abnormality.

What is the most likely diagnosis?

Squamous cell carcinoma	11%
🟩 Small cell carcinoma	54%
Adenocarcinoma	8%
Tuberculosis	13%
🟥 Sarcoidosis	15%

Small cell carcinoma is the correct diagnosis in this case. The presentation of rapid onset neurological symptoms (sensory neuropathy) alongside constitutional symptoms and a hilar mass on chest X-ray is highly suggestive of small cell lung cancer (SCLC) with paraneoplastic syndrome. SCLC is strongly associated with paraneoplastic syndromes, particularly Lambert-Eaton myasthenic syndrome and peripheral neuropathies. The rapid progression of symptoms is also characteristic of SCLC, which typically has a more aggressive course than other lung cancers.

Squamous cell carcinoma typically presents as a central mass and can cause hilar lymphadenopathy, but it is less commonly associated with paraneoplastic syndromes. While it can present with constitutional symptoms like weight loss, the neurological manifestations would be unusual for this type.

Adenocarcinoma is typically peripherally located in the lung and would be less likely to present as a hilar mass. While it can metastasise to cause neurological symptoms, these would more commonly be due to direct spread rather than paraneoplastic phenomena.

Tuberculosis, while common in patients from Pakistan and capable of causing hilar lymphadenopathy, typically presents with more systemic symptoms such as night sweats and haemoptysis. The neurological symptoms would be unusual for TB unless there was direct CNS involvement, which would typically present differently.

Sarcoidosis can present with hilar lymphadenopathy but is typically bilateral rather than unilateral. While neurological involvement can occur in sarcoidosis, it typically affects the cranial nerves rather than causing a peripheral neuropathy. The weight loss and rapid progression would also be unusual for sarcoidosis.

Discuss (6) | Improve

Next question

Paraneoplastic syndromes affecting nervous system

Lambert-Eaton myasthenic syndrome

- associated with small cell lung cancer (also breast and ovarian)
- antibody directed against pre-synaptic voltage gated calcium channel in the peripheral nervous system
- can also occur independently as autoimmune disorder

Anti-Hu

- associated with small cell lung carcinoma and neuroblastomas
- sensory neuropathy - may be painful
- cerebellar syndrome
- encephalomyelitis

Anti-Yo

- associated with ovarian and breast cancer
- cerebellar syndrome

Anti-GAD antibody

- associated with breast, colorectal and small cell lung carcinoma
- stiff person's syndrome or diffuse hypertonia

Anti-Ri

- associated with breast and small cell lung carcinoma
- ocular opsoclonus-myoclonus

Purkinje cell antibody
- peripheral neuropathy in breast cancer

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Mayo Clinic 👍 4 👎 3

Paraneoplastic Syndromes: An Approach to Diagnosis and Treatment

Suggest link

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Media

OPSOCLONUS-
MYOCLONUS
SYNDROME
Osmosis.org

Opsoclonus-myoclonus syndrome

Osmosis - YouTube

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A 64 year old man is referred to you from a psychiatrist for a second opinion. He initially presented with a 3 month history of low mood, apathy and suicidal ideation. In addition he was asked to retire early from his job as an accountant as he was performing poorly at work. He is also sleeping an average of 14 hours per day.

When the psychiatrist assessed him he noted abnormal jerky movements in the lower limbs as well as a broad based gait. An MMSE was performed and he scored 15/30. This is corroborated by your examination and you also note hyperreflexia in the lower limbs and nystagmus.

He has no history of cognitive impairment or any psychiatric history. There is no family history of any neurological or psychiatric conditions and his only past medical history is an appendectomy 20 years ago which was complicated by a large intraperitoneal bleed.

Which findings are you most likely to find on investigation?

Increased T2 and FLAIR signal intensity in the putamen and head of the caudate on T2 weighted MRI, 14-3-3 protein on CSF	57%
CAG trinucleotide repeats on the short arm of chromosome 4	14%
Increased T2 and FLAIR signal intensity in the putamen and head of caudate on T2 weighted MRI and oligoclonal bands on CSF	11%
CAG trinucleotide repeats on the short arm of chromosome 12	11%
Cortical atrophy, most prominent in the frontal lobes	6%

The diagnosis here is Sporadic Creutzfeld Jakob Disease. CJD is characterised by a rapidly progressive dementia along with neuropsychiatric changes. Death usually occurs within a year of diagnosis. Most common psychiatric symptoms are depression and apathy although euphoria, anxiety and emotional lability can also be seen. Myoclonus, ataxia, nystagmus and hyperreflexia are the most common neurological symptoms.

A represents the most likely findings for sporadic CJD. Sporadic CJD normally has a longer clinical course than nvCJD and is normally seen in older people (although nvCJD is possible in this individual as he may have has a blood transfusion 20).

B represents a finding seen in Huntingtons chorea. The lack of family history makes this diagnosis less likely. The oligoclonal bands in C are seen in association with Multiple Sclerosis not CJD. D is not a recognised investigation finding and E would be more consistent with a diagnosis of Picks Disease. The very rapid onset of symptoms and presence of myoclonic jerks make this answer less likely.

👍 👎 💬 Discuss (9) Improve

Next question >

Creutzfeldt-Jakob disease ★

Creutzfeldt-Jakob disease (CJD) is rapidly progressive neurological condition caused by prion proteins. These proteins induce the formation of amyloid folds resulting in tightly packed beta-pleated sheets resistant to proteases.

Features
- dementia (rapid onset)
- myoclonus

Investigation
- CSF is usually normal
- EEG: biphasic, high amplitude sharp waves (only in sporadic CJD)
- MRI: hyperintense signals in the basal ganglia and thalamus

Sporadic CJD
- accounts for 85% of cases
- 10-15% of cases are familial
- mean age of onset is 65 years

New variant CJD
- younger patients (average age of onset = 25 years)
- psychological symptoms such as anxiety, withdrawal and dysphonia are the most common presenting features
- the 'prion protein' is encoded on chromosome 20 - it's role is not yet understood
- methionine homozygosity at codon 129 of the prion protein is a risk factor for developing CJD - all patients who have so far died have had this

- median survival = 13 months

Other prion diseases

- kuru
- fatal familial insomnia
- Gerstmann Straussler-Scheinker disease

123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

PRIONS
(SPONGIFORM
ENCEPHALOPATHY)

Prions (Spongiform encephalopathy)

Osmosis - YouTube 👍 7 👎 0

Report broken media

Score: **20.3%**

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He tells you that he has a long history of alcohol dependence lasting for 30 years drinking approximately 10 units of alcohol a day on average. He tells you that in the past he has tried to stop drinking alcohol but this has caused admission to hospital due to seizures. He is particularly worried because he has cut down on alcohol since the hallucinations because he is worried that he is going mad. He is now only drinking 2 units a day and has not drunk any alcohol for 24 hours. On one previous occasion where he abstained from alcohol, he said that he had hallucinations and had to be admitted to hospital for a few days and put on a drip and was told that he almost died.

Na^+	144 mmol/l
K^+	3.6 mmol/l
Urea	14.1 mmol/l
Creatinine	119 µmol/l

Bilirubin	36 µmol/l
ALP	199 u/l
ALT	92 u/l
γGT	271 u/l
Albumin	36 g/l

He has a family history of alcohol dependence and depression but no other psychiatric problems. His medications include Omeprazole, Vitamin B, Thiamine and Diazepam.

On examination his GCS is 15, there is no tremor or sweating Pulse 80 regular BP 138 / 74 chest clear, abdo soft non tender, no peripheral focal neurology MMSE 28/30 He is commenced on chlordiazepoxide and observed for 24 hours. His GCS remains at 15 and his repeat physical examination remains unchanged and the hallucinations are still present

What is the most likely diagnosis?

○ Late onset schizophrenia ×

○ Delerium tremens ×

○ Alcohol withdrawal syndrome ×

○ Alcoholic hallucinosis ×

○ Hepatic encephalopathy grade 2 ×

Submit answer

Reference ranges ⌄

Score: **20.3%**

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What is the most likely diagnosis?

Late onset schizophrenia	12%
Delerium tremens	16%
Alcohol withdrawal syndrome	16%
Alcoholic hallucinosis	50%
Hepatic encephalopathy grade 2	6%

Alcoholic hallucinosis is a rare condition that can occur during intoxication or withdrawal, but occurs without a clouding of consciousness. Characteristically there are auditory hallucinations which can be vague at first but then can develop into clear voices with a derogotary or persecutory content. The hallucinations normally resolve after less than 6 months.

Delirium tremens will present with a clouding of consciousness and a fluctuating or reduced GCS should be seen when the patient is observed. Also in Delirium Tremens, visual hallucinations are more common, with Lilliputian hallucinations being characteristic of this condition.

Alcohol withdrawal syndrome does not typically cause auditory hallucinations. In addition there is no evidence of a withdrawal syndrome as his pulse is normal there is no sweating or tremulousness. It is possible he has been self medicating with diazepam during his period of abstinence from alcohol.

Schizophrenia is unlikely as a diagnosis as the symptoms are too acute. In addition, affective changes and delusions and family and personal history would be more likely with a diagnosis of schizophrenia.

Hepatic encephalopathy is not the diagnosis. Although there are deranged LFTS, there is no reduction in GCS and there is no liver flap .In grade 2 encephalopathy there should be definite impairment of concentration and attention. However, this is not seen in this individual as evidenced by the high score on the MMSE

👍 👎 Discuss (5) Improve

Next question ›

Alcoholic hallucinosis ★

Alcoholic hallucinosis is a psychiatric disorder considered separate from alcohol withdrawal, Wernicke's/Korsakoff's

Defined as
- a psychosis of less than 6 months duration
- auditory hallucinations, often of persecutory or derogatory nature
- occurs in clear consciousness

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20.9%**

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Question 81 of 299

A 65-year-old man was referred to neurology outpatient clinic with a six month history of double vision. He had first noticed this when reading before going to bed but had more recently been occurring earlier during the day whenever the patient concentrated on an activity. The patients wife also reported that she had been struggling to hear the patient when he spoke to her and that on several occasions the patients eyelids had drooped during late in the day. The patient denied experiencing any weakness in his arms or legs but stated that his general mobility was normally very limited due to osteoarthritis of both his knees. Other past medical history included hypercholesterolaemia, hypertension and diverticular disease. Regular medications included bendroflumethiazide 2.5 mg OD, simvastatin 40 mg OD, co-dydramol as required. The patient was a retired builder and lived alone with his wife. He was an ex-smoker who rarely consumed alcohol.

Examination showed normal pupillary reflexes, visual acuity and visual fields. No ptosis was observed. Assessment of eye movements demonstrated a complex ophthalmoplegia with diplopia in all directions of gaze. There was significant weakness of bilateral facial muscles with bilateral involvement of the forehead. Examination of the tongue and palette was normal. Peripheral nerve examination was unremarkable except for evidence of fatiguable muscle weakness in the upper limbs.

Chest x-ray: clear lung fields with no effusion; no cardiomegaly; possible mediastinal widening

Haemoglobin	13.5 g / dL
White cell count	8.9 * 10^9/l
Platelets	409 * 10^9/l
Urea	4.7 mmol / L
Creatinine	95 micromol / L
Sodium	137 mmol / L
Potassium	4.8 mmol / L
Calcium (adjusted)	2.5 mmol / L

What is the single most sensitive investigation for the likely diagnosis?

○ Repetitive nerve stimulation neurophysiology ×

○ Serum acetylcholine receptor antibodies ×

○ Serum muscle specific tyrosine kinase antibodies ×

○ CT mediastinum ×

○ Single fibre electromyography ×

Submit answer

Reference ranges ⌄

Score: **20.3%**

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Calcium (adjusted)	2.5 mmol / L

What is the single most sensitive investigation for the likely diagnosis?

Repetitive nerve stimulation neurophysiology	21%
Serum acetylcholine receptor antibodies	34%

Serum muscle specific tyrosine kinase antibodies	8%
CT mediastinum	6%
Single fibre electromyography	31%

The patient is presenting with symptoms and signs consistent with myasthenia gravis. All of the above investigations would be appropriate in this context but have variable sensitivities for myasthenia gravis as listed below:

- Single fibre electromyography: sensitivity 92-100 %
- Repetitive nerve stimulation neurophysiology: sensitivity 70 %
- Serum acetylcholine receptor antibodies: sensitivity 85 %
- Serum muscle specific tyrosine kinase antibodies: sensitivity 40-70 %

The Edrophonium (Tensilon) test has a high sensitivity for myasthenia gravis but is no longer used due to risk of fatal cardiac arrhythmia. Around 10 % of patients with myasthenia gravis also have a thymoma and therefore CT scanning is an important part of assessment of these patients. However, this investigation will no yield a diagnosis.

Spillane J, Higham E, Kullman D. Myasthenia gravis. BMJ 2012;345:e8497.

👍 👎 Discuss (3) Improve

Next question ›

Myasthenia gravis ★

Myasthenia gravis is an autoimmune disorder resulting in insufficient functioning acetylcholine receptors. Antibodies to acetylcholine receptors are seen in 85-90% of cases*. Myasthenia is more common in women (2:1)

The key feature is muscle fatigability - muscles become progressively weaker during periods of activity and slowly improve after periods of rest:

- extraocular muscle weakness: diplopia
- proximal muscle weakness: face, neck, limb girdle
- ptosis
- dysphagia

Associations

- thymomas in 15%
- autoimmune disorders: pernicious anaemia, autoimmune thyroid disorders, rheumatoid, SLE
- thymic hyperplasia in 50-70%

Investigations

- single fibre electromyography: high sensitivity (92-100%)
- CT thorax to exclude thymoma
- CK normal
- antibodies to acetylcholine receptors
  - positive in around 85-90% of patients
  - n the remaining patients, about about 40% are positive for anti-muscle-specific tyrosine kinase antibodies
- Tensilon test: IV edrophonium reduces muscle weakness temporarily - not commonly used any more due to the risk of cardiac arrhythmia

Management

- long-acting acetylcholinesterase inhibitors
  - pyridostigmine is first-line
- immunosuppression is usually not started at diagnosis, but the majority of patients eventually require it in addition to long-acting acetylcholinesterase inhibitors:
  - prednisolone initially
  - azathioprine, cyclosporine, mycophenolate mofetil may also be used
- thymectomy

Management of myasthenic crisis

- plasmapheresis
- intravenous immunoglobulins

*antibodies are less commonly seen in disease limited to the ocular muscles

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

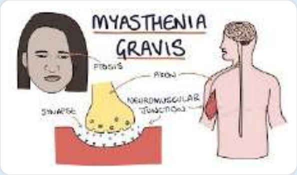
Links

Postgraduate Medical Journal 👍 13 👎 3

Review of myasthenia gravis

Suggest link Report broken link

Media


MYASTHENIA GRAVIS
PTOSIS
AXON
NEUROMUSCULAR JUNCTION
SYNAPSE

Myasthenia gravis

Zero to Finals - YouTube 👍 12 👎 0

NEUROLOGY
MYASTHENIA GRAVIS

Myasthenia gravis

Armando Hasudungan - YouTube 👍 1 👎 1

MYASTHENIA GRAVIS

Myasthenia gravis - causes, symptoms, treatment, pathology

Osmosis - YouTube 👍 2 👎 2

Report broken media

Score: **20.9%**

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The patient reported feeling progressively more short of breath since the operation, particularly when he had tried to lie down to sleep. He denied any cough, chest pain, leg swelling or palpitations. Prior to the operation the patient had been generally well although he had found that he frequently experienced double vision when reading especially in the evening. He also had noticed some difficulties when chewing tough foods in recent weeks. Past medical history was unremarkable and the patient took no regular medications.

Examination revealed a regular pulse, no elevation of jugular venous pressure and normal heart sounds. Both calves were soft and non-tender. The patient had a shallow respiratory effort and was unable to speak in full sentences. Chest expansion was reduced bilaterally, chest was resonant with vesicular breath sounds throughout. The patient had bilateral weakness of facial muscles and ptosis on prolonged upward gaze. Power of neck flexion and extension was reduced, graded as 4/5.

Basic observations:

- Blood pressure: 120 / 76 mmHg
- Heart rate: 115 beats / min
- Respiratory rate: 32 breaths / min
- Temperature: 36.8ºC

Portable CXR: technically poor film due to poor inspiratory effort; clear lung fields; no pleural effusion; no upper lobe blood diversion; no free air under diaphragm.

Arterial blood gas analysis (35 % O2)

pH	7.29
PaCO2	6.6 kPa
PaO2	8.7 kPa
Bicarbonate	18 mmol / L (reference 20.0-26.0)
Lactate	2.1 mmol / L

Bedside forced vital capacity: 1.9 L

What is the most important next step in management?

- ○ Pyridostigmine ×
- ○ Referral to intensive care unit ×
- ○ Reduce percentage of supplemental oxygen ×
- ○ High-dose corticosteroids ×
- ○ Intravenous immunoglobulin ×

Submit answer

Reference ranges ∨

Score: **20.3%**

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A 65-year-old man underwent an elective inguinal hernia repair. Due to the list running late into the evening, the patient was admitted for an overnight stay. During the night after the operation the patient was observed to have an increasing oxygen requirement and the following morning was referred to the oncall medical registrar.

The patient reported feeling progressively more short of breath since the operation, particularly when he had tried to lie down to sleep. He denied any cough, chest pain, leg swelling or palpitations. Prior to the operation the patient had been generally well although he had found that he frequently experienced double vision when reading especially in the evening. He also had noticed some difficulties when chewing tough foods in recent weeks. Past medical history was unremarkable and the patient took no regular medications.

Examination revealed a regular pulse, no elevation of jugular venous pressure and normal heart sounds. Both calves were soft and non-tender. The patient had a shallow respiratory effort and was unable to speak in full sentences. Chest expansion was reduced bilaterally, chest was resonant with vesicular breath sounds throughout. The patient had bilateral weakness of facial muscles and ptosis on prolonged upward gaze. Power of neck flexion and extension was reduced, graded as 4/5.

Basic observations:

- Blood pressure: 120 / 76 mmHg
- Heart rate: 115 beats / min
- Respiratory rate: 32 breaths / min
- Temperature: 36.8°C

Portable CXR: technically poor film due to poor inspiratory effort; clear lung fields; no pleural effusion; no upper lobe blood diversion; no free air under diaphragm.

Arterial blood gas analysis (35 % O2)

pH	7.29
PaCO2	6.6 kPa
PaO2	8.7 kPa
Bicarbonate	18 mmol / L (reference 20.0-26.0)
Lactate	2.1 mmol / L

Bedside forced vital capacity: 1.9 L

What is the most important next step in management?

Pyridostigmine	17%
Referral to intensive care unit	51%
Reduce percentage of supplemental oxygen	2%
High-dose corticosteroids	3%
Intravenous immunoglobulin	27%

The patient has symptoms and signs of previously unrecognised myasthenia gravis. The physiological stress of surgery has precipitated a myasthenic crisis associated with type 2 respiratory failure, tachycardia, tachypnoea and reduced forced vital capacity. The priority in management is to refer the patient to an intensive care unit for respiratory support.

Corticosteroids and pyridostigmine are mainstays of treatment of myasthenia gravis with intravenous immunoglobulin also used in severe cases. However, these treatments cannot be expected to restore respiratory muscle function rapidly in this emergency situation.

The patient has no history of obstructive lung disease with type 2 respiratory failure due instead to failure of ventilation due to respiratory muscle weakness. Therefore, reducing percentage of supplemental oxygen would not be beneficial and would cause additional hypoxia.

Spillane J, Higham E, Kullman D. Myasthenia gravis. BMJ 2012;345:e8497.

👍 👎 Discuss (10) Improve

Next question ›

Myasthenia gravis: exacerbating factors ★

The most common exacerbating factor is exertion resulting in fatigability, which is the hallmark feature of myasthenia gravis.. Symptoms become more marked during the day

The following drugs may exacerbate myasthenia:

- penicillamine
- quinidine, procainamide
- beta-blockers
- lithium
- phenytoin
- antibiotics: gentamicin, macrolides, quinolones, tetracyclines

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians (Edin) 👍 4 👎 2

Myasthenia gravis review

Myasthenia Gravis Association 👍 9 👎 3

Drugs which may aggravate myasthenia gravis

Suggest link

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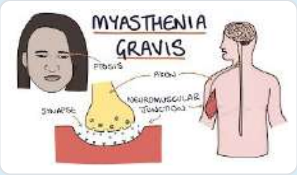
Media

MYASTHENIA GRAVIS

Myasthenia gravis - causes, symptoms, treatment, pathology

Osmosis - YouTube 👍 1 👎 0


MYASTHENIA GRAVIS
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NEUROMUSCULAR JUNCTION
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Myasthenia gravis

Zero to Finals - YouTube 👍 2 👎 1

Report broken media

Score: **20.9%**

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A 48-year-old woman presented to the emergency department with a severe headache. Symptoms had started early that day while the patient had been walking around her office with the headache reaching maximal intensity within a few minutes. The pain was felt across the entirety of the patient's head and was much improved when she lay down flat. There were no associated symptoms and the patient had been constitutionally well in the preceding days.

The patient had no past medical history and was nulliparous. She was a non-smoker who consumed 15 units of alcohol per week.

Clinical examination demonstrated no evidence of focal neurological deficit and no signs of meningism. Simple analgesia given in the emergency department had limited impact on the patient's headache.

CT brain with venogram: no evidence of intra-axial or extra-axial bleeding; no space occupying lesion; no hydrocephalus; no evidence of venous sinus thrombosis

Lumbar puncture: opening pressure 5 mmHg; red cells 8 mm^3; white cells 1 / mm^3; no xanthrochromia

What is the next best investigation to confirm the likely diagnosis?

- ○ Digital subtraction myelography
- ○ Cerebral angiography
- ○ CT brain with contrast
- ○ MRI whole spine with STIR
- ○ MRI brain with gadolinium

Submit answer

Reference ranges

Score: **20.3%**

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Lumbar puncture: opening pressure 5 mmHg; red cells 8 mm^3; white cells 1 / mm^3; no xanthrochromia

What is the next best investigation to confirm the likely diagnosis?

Digital subtraction myelography	12%
Cerebral angiography	38%
CT brain with contrast	6%
MRI whole spine with STIR	11%
MRI brain with gadolinium ✓	34%

The clinical presentation and initial investigations are suggestive of spontaneous intracranial hypotension. The key aspect of the history is the strong postural relationship to pain with the headache worsening within a few seconds of upright posture and easing within a minute of lying horizontal. Spontaneous intracranial hypotension can cause either a headache of insidious or rapid onset. A low opening pressure on lumbar puncture (< 6 cmCSF) is one of the diagnostic criteria.

Diagnosis is confirmed by MRI with gadolinium that demonstrates distinctive dural gadolinium enhancement and downward displacement of brain on sagittal views.

In some cases of spontaneous intracranial hypotension, investigations such as MRI whole spine with STIR sequences or digital subtraction myelography may be used to identify the location of CSF leak in order to guide blood patch treatment.

Scott S, Davenport R. Low pressure headaches caused by spontaneous intracranial hypotension. BMJ 2014;349:6219.

Discuss (9) Improve

Next question

Spontaneous intracranial hypotension

Spontaneous intracranial hypotension is a very rare cause of headaches that results from a CSF leak. The leak is typically from the thoracic nerve root sleeves.

Risk factors include connective tissue disorders such as Marfan's syndrome.

Key features
- strong postural relationship with the headache generally much worse when upright. Patients may, therefore, be bed-bound

Investigations
- MRI with gadolinium: typically shows pachymeningeal enhancement

Management
- usually conservative
- if this fails an epidural blood patch may be tried

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **20.9%**

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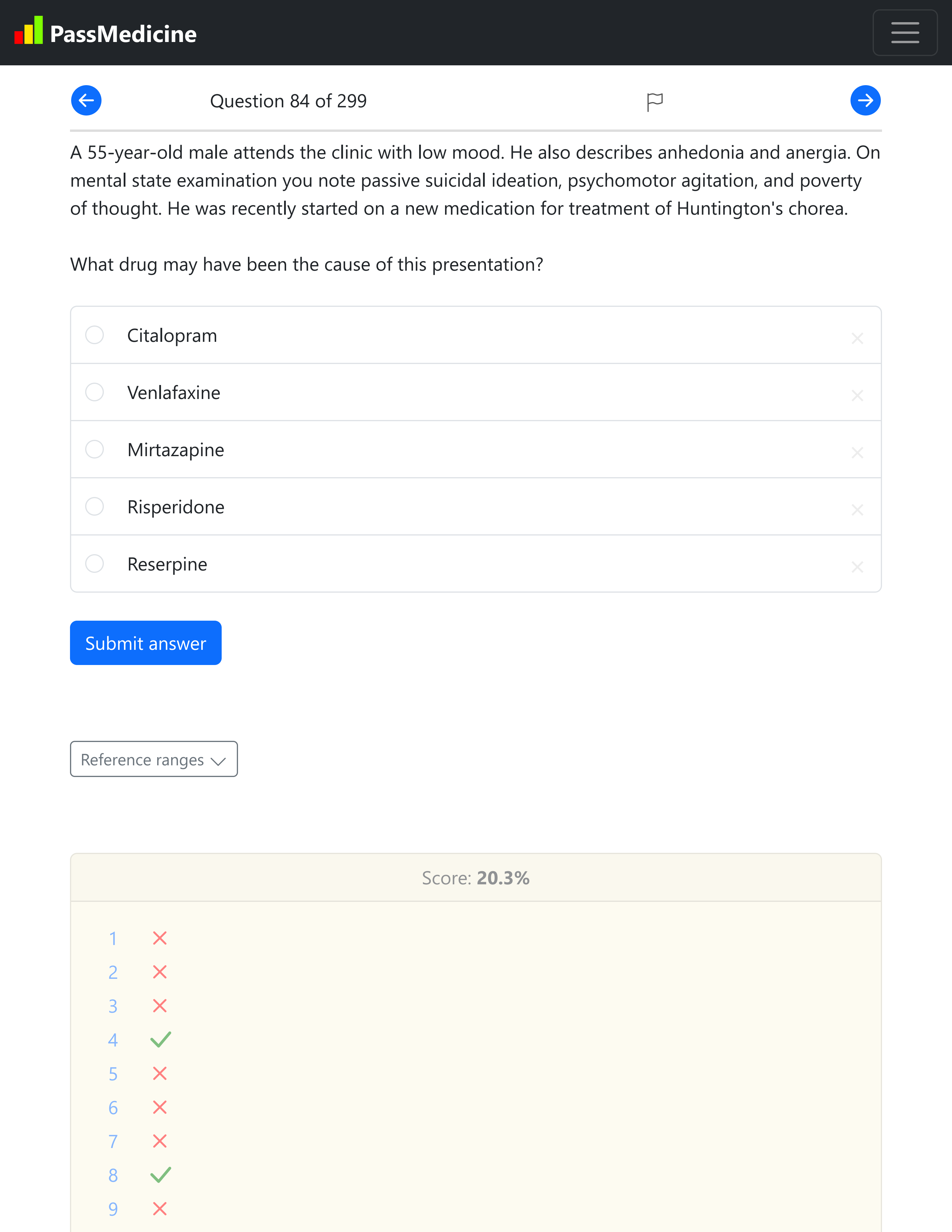

Question 84 of 299

A 55-year-old male attends the clinic with low mood. He also describes anhedonia and anergia. On mental state examination you note passive suicidal ideation, psychomotor agitation, and poverty of thought. He was recently started on a new medication for treatment of Huntington's chorea.

What drug may have been the cause of this presentation?

○ Citalopram

○ Venlafaxine

○ Mirtazapine

○ Risperidone

○ Reserpine

Score: **20.3%**

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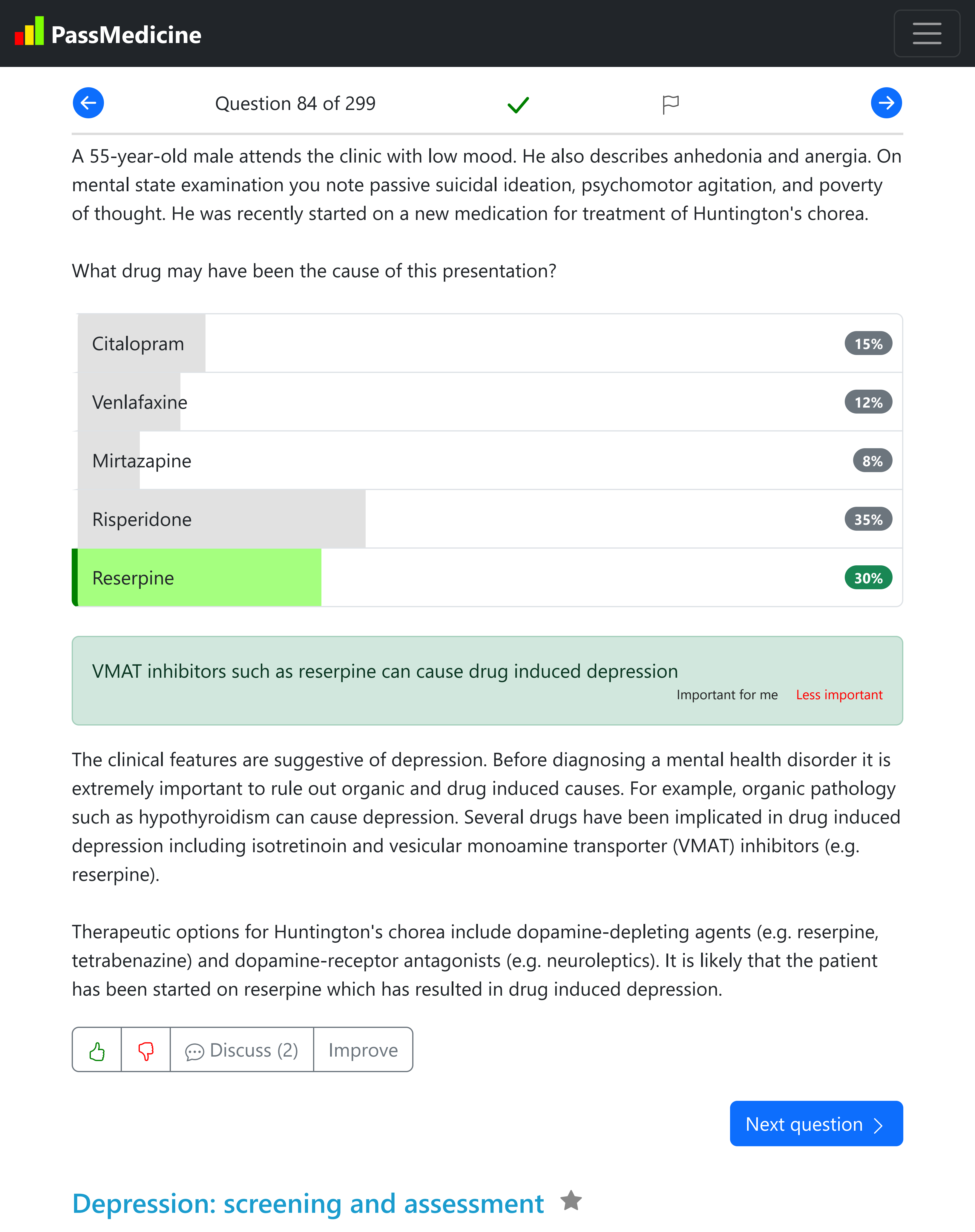

A 55-year-old male attends the clinic with low mood. He also describes anhedonia and anergia. On mental state examination you note passive suicidal ideation, psychomotor agitation, and poverty of thought. He was recently started on a new medication for treatment of Huntington's chorea.

What drug may have been the cause of this presentation?

Citalopram	15%
Venlafaxine	12%
Mirtazapine	8%
Risperidone	35%
Reserpine	30%

VMAT inhibitors such as reserpine can cause drug induced depression

The clinical features are suggestive of depression. Before diagnosing a mental health disorder it is extremely important to rule out organic and drug induced causes. For example, organic pathology such as hypothyroidism can cause depression. Several drugs have been implicated in drug induced depression including isotretinoin and vesicular monoamine transporter (VMAT) inhibitors (e.g. reserpine).

Therapeutic options for Huntington's chorea include dopamine-depleting agents (e.g. reserpine, tetrabenazine) and dopamine-receptor antagonists (e.g. neuroleptics). It is likely that the patient has been started on reserpine which has resulted in drug induced depression.

Depression: screening and assessment

Screening

The following two questions can be used to screen for depression
- 'During the last month, have you often been bothered by feeling down, depressed or hopeless?'
- 'During the last month, have you often been bothered by having little interest or pleasure in doing things?'

A 'yes' answer to either of the above should prompt a more in depth assessment.

Assessment

There are many tools to assess the degree of depression including the Hospital Anxiety and Depression (HAD) scale and the Patient Health Questionnaire (PHQ-9).

Hospital Anxiety and Depression (HAD) scale
- consists of 14 questions, 7 for anxiety and 7 for depression
- each item is scored from 0-3
- produces a score out of 21 for both anxiety and depression
- severity: 0-7 normal, 8-10 borderline, 11+ case
- patients should be encouraged to answer the questions quickly

Patient Health Questionnaire (PHQ-9)
- asks patients 'over the last 2 weeks, how often have you been bothered by any of the following problems?'
- 9 items which can then be scored 0-3
- includes items asking about thoughts of self-harm

Traditionally, depression severity has been grouped under 4 categories (subthreshold, mild, moderate and severe). The updated NICE guideline uses a simpler 2 category definition of depression: less severe or more severe depression.

Less severe depression encompasses subthreshold and mild depression, and more severe depression encompasses moderate and severe depression. Thresholds on validated scales were used in this guideline as an indicator of severity
- a score < 16 on the PHQ-9: less severe depression
- a score of ≥ 16 on the PHQ-9: severe depression

The DSM-5 also provides criteria for diagnosing major depressive disorder (MDD), commonly referred to as depression.

Major Depressive Disorder (MDD) DSM-5 Criteria:

Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

- Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad, empty, hopeless) or observation made by others (e.g., appears tearful). (Note: In children and adolescents, can be irritable mood.)
- Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation).
- Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. (Note: In children, consider failure to make expected weight gain.)
- Insomnia or hypersomnia nearly every day.
- Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).
- Fatigue or loss of energy nearly every day.
- Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).
- Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).
- Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 23 👎 10

PHQ 9 form

Suggest link Report broken link

Media

LOW MOOD
ANHEDONIA
SEROTONIN
MAJOR DEPRESSIVE DISORDER
ELECTROCONVULSION

Depression Explained (Major Depressive Disorder)

Rhesus Medicine - YouTube 👍 0 👎 1

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Score: **20.9%**

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Question 85 of 299

A 68-year-old man presents with a weak left leg. He states that he first noticed it a few weeks ago. On examination you note weakness of left ankle plantar flexion.

Nerve conduction studies of the tibial nerve are as follows:

Conduction velocity	25 metres per second (normal >50)
Amplitude	21mV (normal >17)

What is the most likely cause?

- ○ Uraemia
- ○ Multiple myeloma
- ○ HIV
- ○ Vitamin B12 deficiency
- ○ Diabetes mellitus

Submit answer

Reference ranges

Score: **20.3%**

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Nerve conduction studies of the tibial nerve are as follows:

Conduction velocity	25 metres per second (normal >50)
Amplitude	21mV (normal >17)

What is the most likely cause?

Uraemia	9%
Multiple myeloma	22%
HIV	9%
Vitamin B12 deficiency	39%
Diabetes mellitus	22%

When interpreting NCS, a reduced conduction velocity but normal amplitude is indicative of demyelinating pathology

Important for me Less important

Peripheral neuropathies can result from axonal degeneration, demyelination, or a combination of the two. Generally, axonal neuropathies are caused by metabolic, toxic or heritable causes. Demyelinating neuropathies are generally caused by autoimmune processes, paraproteinaemia or the result of an inherited disorder of myelin It is very difficult to distinguish between the two on clinical grounds, hence why nerve conduction studies (NCS) are very useful for making the distinction.

When interpreting NCS, a reduced amplitude but normal conduction velocity is indicative of axonal pathology. Causes of a predominant sensory axonal peripheral neuropathy include diabetes mellitus (commonest cause), B12 deficiency, uraemia, carcinoma (paraneoplastic) and HIV.

The NCS in this case confirm a demyelinating process. Multiple myeloma is the only given option which causes demyelination. The other options are causes of an axonal peripheral neuropathy.

Discuss (14) Improve

Next question

Nerve conduction studies

Nerve conduction studies (NCS) are useful in determining between axonal and demyelinating pathology

Axonal
- normal conduction velocity
- reduced amplitude

Demyelinating
- reduced conduction velocity
- normal amplitude

+ Q 123

Next question

Textbooks

High-yield textbook

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Score: **20.9%**

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Question 86 of 299

A 54-year-old man presents with seizures. His wife states that they recently returned from Kenya. Observations are as follows: heart rate 96 beats per minute, blood pressure 130/85 mmHg, respiratory rate 16 breaths per minute, SpO2 96% (on air), temperature 38.6ºC.

An MRI scan is performed:

MRI head	General oedema of the medial temporal lobes on T1

What is the most likely pathogen?

- ○ Coxsackie
- ○ Herpes simplex virus type 1
- ○ Herpes simplex virus type 2
- ○ Varicella zoster virus
- ○ West Nile virus

Submit answer

Reference ranges

Score: **20.3%**

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Question 86 of 299

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An MRI scan is performed:

MRI head	General oedema of the medial temporal lobes on T1

What is the most likely pathogen?

Coxsackie	3%
Herpes simplex virus type 1	69%
Herpes simplex virus type 2	15%
Varicella zoster virus	1%
West Nile virus	12%

CT head showing temporal lobe changes - think herpes simplex encephalitis

Important for me Less important

Herpes simplex virus type 1 is correct. The clinical ad radiological features are consistent with herpes simplex encephalitis. HSV-1 is responsible for 95% of cases of viral encephalitis in adults.

Coxsackie is incorrect. Coxsackie or Echovirus groups of enteroviruses are the most common cause of viral meningitis. The patient in this clinical case has clinical features of encephalitis (e.g. seizures) rather than meningitis (headache, neck stiffness, and photophobia).

Herpes simplex virus type 2 is incorrect. It is herpes simplex virus type 1 which is more commonly implicated in viral encephalitis.

Varicella zoster virus is incorrect. Although this remains within the differential diagnosis, the most common cause of viral encephalitis is herpes simplex virus type 1 making this the preferred answer.

West Nile virus is incorrect. Although this virus remains within the differential diagnosis given the travel history, it is a rare infection in travellers, and thus a less likely diagnosis. However, a consult with infectious diseases and appropriate testing should be conducted in order to exclude it.

👍 👎 Discuss (1) Improve

Next question >

Herpes simplex encephalitis ★

Herpes simplex (HSV) encephalitis is a common topic in the exam. The virus characteristically affects the temporal lobes - questions may give the result of imaging or describe temporal lobe signs e.g. aphasia.

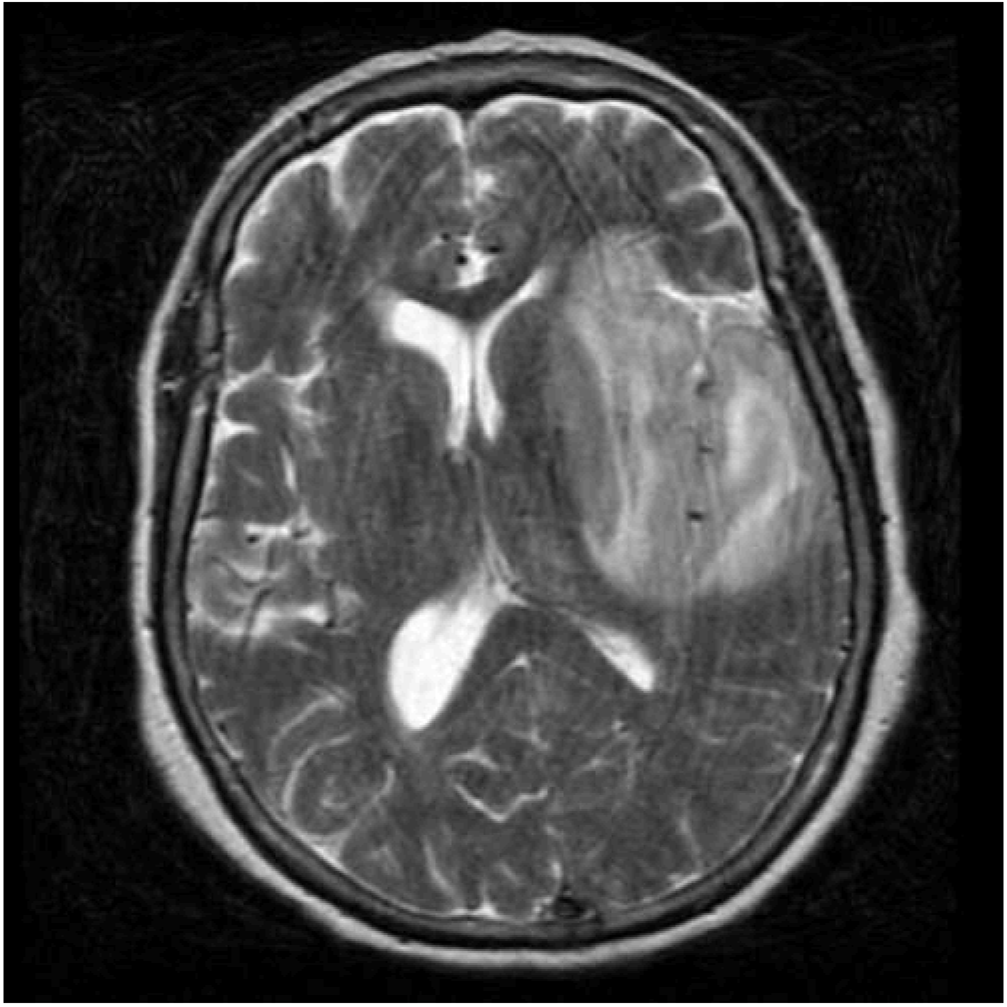
Features
- fever, headache, psychiatric symptoms, seizures, vomiting
- focal features e.g. aphasia
- peripheral lesions (e.g. cold sores) have no relation to the presence of HSV encephalitis

Pathophysiology
- HSV-1 is responsible for 95% of cases in adults
- typically affects temporal and inferior frontal lobes

Investigation
- CSF: lymphocytosis, elevated protein
- PCR for HSV
- CT: medial temporal and inferior frontal changes (e.g. petechial haemorrhages) - normal in one-third of patients
- MRI is better
- EEG pattern: lateralised periodic discharges at 2 Hz

Treatment
- intravenous aciclovir

The prognosis is dependent on whether aciclovir is commenced early. If treatment is started promptly the mortality is 10-20%. Left untreated the mortality approaches 80%



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MRI of a patient with HSV encephalitis. There is hyperintensity of the affected white matter and cortex in the medial temporal lobes and insular cortex.

123

Next question

Textbooks

High-yield textbook

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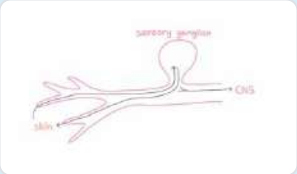
Media


sensory ganglion
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Herpes simplex encephalitis

macrophage - YouTube 👍 2 👎 0

Report broken media

Score: **20.9%**

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Question 87 of 299

A 22-year-old man attends with a 3-month history of low mood. He also states that he has reduced energy levels and does not get enjoyment from things that he usually would. On mental state examination, he has no evidence of delusions or thoughts of self-harm. He is started on sertraline.

When should the patient be reviewed?

- ○ 1 week
- ○ 2 weeks
- ○ 3 months
- ○ 4 weeks
- ○ 6 months

Submit answer

Reference ranges

Score: **20.3%**

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A 22-year-old man attends with a 3-month history of low mood. He also states that he has reduced energy levels and does not get enjoyment from things that he usually would. On mental state examination, he has no evidence of delusions or thoughts of self-harm. He is started on sertraline.

When should the patient be reviewed?

1 week	43%
2 weeks	29%
3 months	5%
4 weeks	21%
6 months	3%

Patients ≤ 25 years who have been started on an SSRI should be reviewed after 1 week

Important for me Less important

1 week is correct. Patients under the age of 25 years or who are at increased risk of suicide (e.g. psychosis, previous self-harm) should be reviewed after 1-week following the initiation of antidepressant therapy. This is because some evidence suggests that selective serotonin reuptake inhibitors (SSRIs) may cause worsening of suicidal ideas in vulnerable patients, particularly in the very early stages of treatment.

Discuss (2) Improve

Next question

Selective serotonin reuptake inhibitors

Selective serotonin reuptake inhibitors (SSRIs) are considered first-line treatment for the majority of patients with depression.

- citalopram (although see below re: QT interval) and fluoxetine are currently the preferred SSRIs
- sertraline is useful post myocardial infarction as there is more evidence for its safe use in this situation than other antidepressants
- SSRIs should be used with caution in children and adolescents. Fluoxetine is the drug of choice when an antidepressant is indicated

Adverse effects

- gastrointestinal symptoms are the most common side-effect
- there is an increased risk of gastrointestinal bleeding in patients taking SSRIs. A proton pump inhibitor should be prescribed if a patient is also taking a NSAID
- patients should be counselled to be vigilant for increased anxiety and agitation after starting a SSRI
- fluoxetine and paroxetine have a higher propensity for drug interactions

Citalopram and the QT interval

- the Medicines and Healthcare products Regulatory Agency (MHRA) released a warning on the use of citalopram in 2011
- it advised that citalopram and escitalopram are associated with dose-dependent QT interval prolongation and should not be used in those with: congenital long QT syndrome; known pre-existing QT interval prolongation; or in combination with other medicines that prolong the QT interval
- the maximum daily dose is now 40 mg for adults; 20 mg for patients older than 65 years; and 20 mg for those with hepatic impairment

Interactions

- NSAIDs: NICE guidelines advise 'do not normally offer SSRIs', but if given co-prescribe a proton pump inhibitor
- warfarin / heparin: NICE guidelines recommend avoiding SSRIs and considering mirtazapine
- aspirin
- triptans - increased risk of serotonin syndrome
- monoamine oxidase inhibitors (MAOIs) - increased risk of serotonin syndrome

Following the initiation of antidepressant therapy patients should normally be reviewed by a doctor after 2 weeks. For patients under the age of 25 years or at increased risk of suicide should be reviewed after 1 week. If a patient makes a good response to antidepressant therapy they should continue on treatment for at least 6 months after remission as this reduces the risk of relapse.

When stopping a SSRI the dose should be gradually reduced over a 4 week period (this is not necessary with fluoxetine). Paroxetine has a higher incidence of discontinuation symptoms.

Discontinuation symptoms

- increased mood change
- restlessness
- difficulty sleeping
- unsteadiness
- sweating
- gastrointestinal symptoms: pain, cramping, diarrhoea, vomiting
- paraesthesia

SSRIs and pregnancy
- BNF says to weigh up benefits and risk when deciding whether to use in pregnancy.
- Use during the first trimester gives a small increased risk of congenital heart defects
- Use during the third trimester can result in persistent pulmonary hypertension of the newborn
- Paroxetine has an increased risk of congenital malformations, particularly in the first trimester

123

Next question

Textbooks

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Links

NICE 29 43

2022 Depression in adults: treatment and management

Suggest link Report broken link

Media

Antidepressants

Speed Pharmacology - YouTube 👍 6 👎 0

ANTIDEPRESSANTS
PHARMACOLOGY

Antidepressants Pharmacology

Soton Brain Hub - YouTube 👍 0 👎 0

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Score: **20.9%**

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A 54-year-old woman presents to the Emergency Department with a five hour history of headache and visual disturbance. She states the headache came on quickly and she has vomited ten times since this pain started. She denies any past medical history and states she wears glasses for reading.

On examination there is a mid-dilated pupil in the left eye. The right eye direct pupillary reaction was satisfactory. There is no tenderness over the left temporal artery and there is no other neurological deficit.

Her observations revealed a blood pressure of 141/90 mmHg, heart rate 90 bpm, temperature 36.4ºC, oxygen saturation 96% on room air and respiratory rate 12/min.

Her blood results are as follows:

Hb	140 g/L	Male: (135-180) Female: (115 - 160)
Platelets	450 * 10^9/L	(150 - 400)
WBC	11.0 * 10^9/L	(4.0 - 11.0)
Na^+	135 mmol/L	(135 - 145)
K^+	4.6 mmol/L	(3.5 - 5.0)
Urea	5 mmol/L	(2.0 - 7.0)
Creatinine	80 µmol/L	(55 - 120)
CRP	8 mg/L	(< 5)

Topical pilocarpine was the applied to the eye.

What other medication should be given urgently to help manage this condition?

○ Intravenous acetazolamide ×

○ Topical cyclopentolate ×

○ Oral nimodipine ×

○ Topical tropicamide ×

○ Oral beta blocker ×

Submit answer

Reference ranges ⌄

Score: **20.3%**

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Topical pilocarpine was the applied to the eye.

What other medication should be given urgently to help manage this condition?

Intravenous acetazolamide	82%
Topical cyclopentolate	5%
Oral nimodipine	4%

Topical tropicamide 6%

Oral beta blocker 3%

Alongside eye drops, IV acetazolamide is used in the initial emergency medical management of acute angle-closure glaucoma

Important for me Less important

The answer here is acetazolamide. This patient presents with acute angle-closure glaucoma. The acute onset headache, mid-dilated pupil with no other neurological deficit makes acute angle-closure glaucoma the most likely pathology. After administering eye drops such as timolol and pilocarpine the optimal intravenous treatment is acetazolamide; a carbonic anhydrase inhibitor which decreases the level of aqueous humour in the eye.

Cyclopentolate is and acetylcholine receptor antagonist antagonist which allows mydriasis, this would cause further closure of the iridocorneal angle and therefore worsening this patients symptoms.

Nimodipine is a calcium channel blocker used in the treatment of subarachnoid haemorrhage to combat the effects of cerebral vasospasm.

Tropicamide is a dilating eye drop which is used may reduced the iridocorneal angle and therefore drainage of aqueous humour, potentially worsening the acute angle closure glaucoma.

Pilocarpine and timolol are used in the treatment of acute angle-closure glaucoma but are used as eye drops rather than intravenously. Pilocarpine causes miosis, thereby increasing the rate at which aqueous humour leaves the eye and decreasing intraocular pressure. The exact mechanism of timolol is not known but it is thought that through inhibition of beta receptors, the production of aqueous humour is reduced. Timolol is a beta blocker but is used topically, there is no known role for oral beta blockers in acute angle closure glaucoma.

👍 👎 Discuss Improve

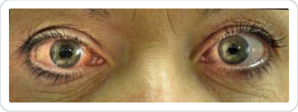
Next question ›

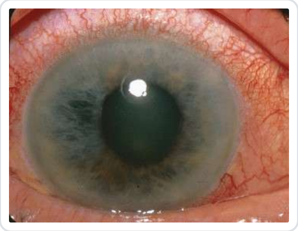
Acute angle-closure glaucoma ★

Glaucoma is a group of disorders characterised by optic neuropathy due, in the majority of patients, to raised intraocular pressure (IOP). It is now recognised that a minority of patients with raised IOP do not have glaucoma and vice versa.

In acute angle-closure glaucoma (AACG) there is a rise in IOP secondary to an impairment of aqueous outflow. Factors predisposing to AACG include:
- hypermetropia (long-sightedness)
- pupillary dilatation
- lens growth associated with age

Features
- severe pain: may be ocular or headache
- decreased visual acuity
- symptoms worse with mydriasis (e.g. watching TV in a dark room)
- hard, red-eye
- haloes around lights
- semi-dilated non-reacting pupil
- corneal oedema results in dull or hazy cornea
- systemic upset may be seen, such as nausea and vomiting and even abdominal pain





Investigations
- tonometry to assess for elevated IOP
- gonioscopy (literally looking, *oscopy*, at the angle, *gonio*): a special lens for the slit lamp that allows visualisation of the angle

Management

The management of AACG is an emergency and should prompt urgent referral to an ophthalmologist. Emergency medical treatment is required to lower the IOP with more definitive surgical treatment given once the acute attack has settled.

There are no guidelines for the initial medical treatment emergency treatment. An example regime would be:
- combination of eye drops, for example:
    - a direct parasympathomimetic (e.g. pilocarpine, causes contraction of the ciliary muscle → opening the trabecular meshwork → increased outflow of the aqueous humour)
    - a beta-blocker (e.g. timolol, decreases aqueous humour production)

    - an alpha-2 agonist (e.g. apraclonidine, dual mechanism, decreasing aqueous humour production and increasing uveoscleral outflow)
- intravenous acetazolamide
    - reduces aqueous secretions
- some guidelines also recommend the use of topical steroids to reduce inflammation

Definitive management

- laser peripheral iridotomy
    - creates a tiny hole in the peripheral iris → aqueous humour flowing to the angle

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

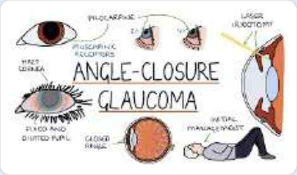
Links

Patient.info 👍 26 👎 9

Acute angle closure glaucoma review

Suggest link Report broken link

Media



Acute angle closure glaucoma

Zero To Finals - YouTube 👍 4 👎 0

GLAUCOMA
Osmosis.org

Glaucoma (open-angle, closed-angle, and normal-tension)

Osmosis - YouTube 👍 6 👎 3

Report broken media

Score: **20.9%**

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Back to top

A 27-year-old lady was admitted to the Emergency Department due to acute shortness of breath. She had been completely normal that morning. Her sister mentioned that this was not the first time she had suffered such an attack and she improved every time after staying in hospital for a couple of hours and taking some nebulisers. The patient is not known to have any medical condition. Her father is on long term prednisone tablets and nebulisers for chronic obstructive pulmonary disease.

On examination: blood pressure 123/86 mmHg, pulse rate 110/min, respiratory rate 35/min. Oxygen saturation 98%. The chest was clear with good air entry.

Blood investigations showed:

Hb	128 g/l
Platelets	$320 * 10^9$/l
WBC	$8 * 10^9$/l
Na^+	137 mmol/l
K^+	4.2 mmol/l
Urea	6 mmol/l
Creatinine	100 µmol/l

A 3-month-old pulmonary function test was normal.

What is the next step in management?

○ Discharge home

○ Refer to repeat pulmonary function tests

○ Nijmegen questionnaire

○ Serial ECG

○ Psychiatric review

Submit answer

Reference ranges

Score: **20.3%**

1 ✗
2 ✗
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A 3-month-old pulmonary function test was normal.

What is the next step in management?

Discharge home	5%
Refer to repeat pulmonary function tests	6%
Nijmegen questionnaire	43%
Serial ECG	2%
Psychiatric review	43%

Normal patient observations and examination ruled out any significant pathology. The history of repeated admissions without a diagnosis and rapid recovery are all pointers towards hyperventilation syndrome (HVS). If a doctor encounters such presentation, the Nijmegen questionnaire can be used to test whether or not the patient has HVS. This questionnaire involves asking about 16 different symptoms such as chest pain and tingling fingers. Each one of these symptoms should be assigned a number from 0 to 4 according to how often it is felt. A score of more than 23 out of 64 is diagnostic of HVS.

Discuss (8) Improve

Next question >

Generalised anxiety disorder and panic disorder

Anxiety is a common disorder that can present in multiple ways. NICE define the central feature as an 'excessive worry about a number of different events associated with heightened tension.'

Always look for a potential physical cause when considering a psychiatric diagnosis. In anxiety disorders, important alternative causes include hyperthyroidism, cardiac disease and medication-induced anxiety (NICE). Medications that may trigger anxiety include salbutamol, theophylline, corticosteroids, antidepressants and caffeine

Management of generalised anxiety disorder (GAD)

NICE suggest a step-wise approach:

- step 1: education about GAD + active monitoring
- step 2: low-intensity psychological interventions (individual non-facilitated self-help or individual guided self-help or psychoeducational groups)
- step 3: high-intensity psychological interventions (cognitive behavioural therapy or applied relaxation) or drug treatment. See drug treatment below for more information
- step 4: highly specialist input e.g. Multi agency teams

Drug treatment

- NICE suggest sertraline should be considered the first-line SSRI
- if sertraline is ineffective, offer an alternative SSRI or a serotonin-noradrenaline reuptake inhibitor (SNRI)
  - examples of SNRIs include duloxetine and venlafaxine
- If the person cannot tolerate SSRIs or SNRIs, consider offering pregabalin
- interestingly for patients under the age of 30 years NICE recommend you warn patients of the increased risk of suicidal thinking and self-harm. Weekly follow-up is recommended for the first month

Management of panic disorder

Again a stepwise approach:

- step 1: recognition and diagnosis
- step 2: treatment in primary care - see below
- step 3: review and consideration of alternative treatments
- step 4: review and referral to specialist mental health services
- step 5: care in specialist mental health services

Treatment in primary care

- NICE recommend either cognitive behavioural therapy or drug treatment
- SSRIs are first-line. If contraindicated or no response after 12 weeks then imipramine or clomipramine should be offered

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

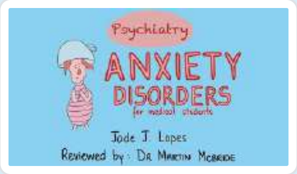
NICE 👍 9 👎 12

2011 Generalised anxiety disorder and panic disorder guidelines

Suggest link

Report broken link

Media


Psychiatry
ANXIETY
DISORDERS
for medical students
Jode J. Lopes
Reviewed by: Dr Martin McBride

Anxiety Disorders for Medical Students

MedFlix - YouTube 👍 2 👎 0

PANIC
DISORDER

Panic disorder

Osmosis - YouTube 👍 1 👎 0

IMPENDING DOOM
ARACHNOPHOBIA
COMPULSION
ANXIETY
PANIC ATTACK
WORRY
PALPITATIONS

Understanding Anxiety (Anxiety Disorders Explained)

Rhesus Medicine - YouTube 👍 0 👎 0

GENERALIZED
ANXIETY DISORDER

Generalized anxiety disorder

Osmosis - YouTube 👍 1 👎 1

Report broken media

Score: **20.9%**

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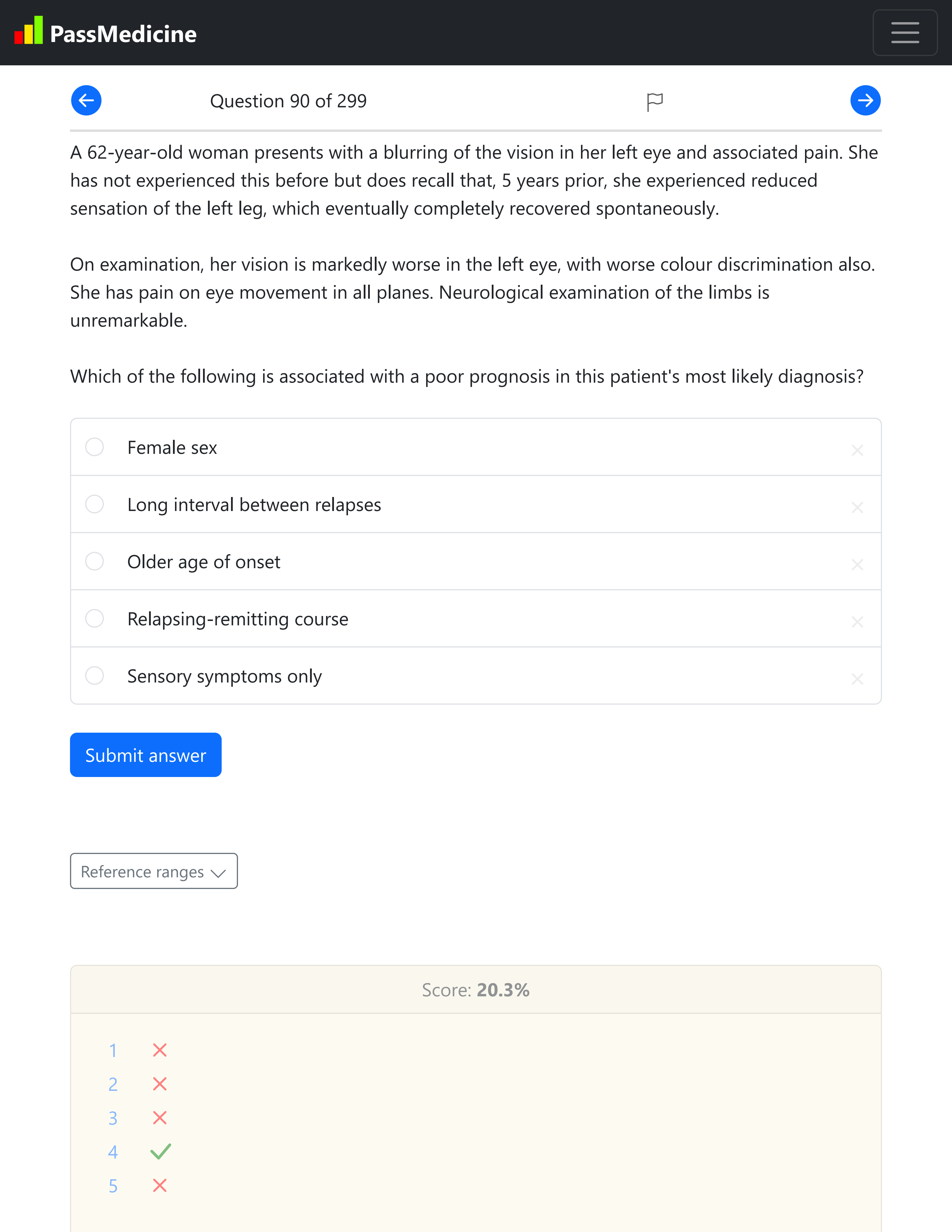

A 62-year-old woman presents with a blurring of the vision in her left eye and associated pain. She has not experienced this before but does recall that, 5 years prior, she experienced reduced sensation of the left leg, which eventually completely recovered spontaneously.

On examination, her vision is markedly worse in the left eye, with worse colour discrimination also. She has pain on eye movement in all planes. Neurological examination of the limbs is unremarkable.

Which of the following is associated with a poor prognosis in this patient's most likely diagnosis?

- ○ Female sex
- ○ Long interval between relapses
- ○ Older age of onset
- ○ Relapsing-remitting course
- ○ Sensory symptoms only

Score: **20.3%**

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On examination, her vision is markedly worse in the left eye, with worse colour discrimination also. She has pain on eye movement in all planes. Neurological examination of the limbs is unremarkable.

Which of the following is associated with a poor prognosis in this patient's most likely diagnosis?

Female sex	13%
Long interval between relapses	2%
Older age of onset	73%
Relapsing-remitting course	9%
Sensory symptoms only	3%

Older age of onset is associated with a poor prognosis in multiple sclerosis

Important for me Less important

The diagnosis here is likely to be multiple sclerosis, given the two distinct episodes over time. The first episode presented with weakness of the left leg some years ago, recovered fully, and the recent episode is indicative of optic neuritis. It is worth remembering that the 'typical' patient carries a good prognosis - of the feature of this patient her age on diagnosis is atypical; older age is associated with a poorer prognosis. Usually, patients would present in their 20s or 30s.

Female sex is associated with a good prognosis in multiple sclerosis.

A 5-year interval between episodes, as seen here, is a significant amount of time between the first two episodes and is considered a marker of good prognosis, not poor.

This patient also demonstrates a relapsing-remitting course - complete recovery between episodes. This is also indicative of a good prognosis, not poor.

The presence of only symptoms, as opposed to motor symptoms too, is also considered to be a sign of good prognosis, not poor.

Discuss Improve

Next question

Multiple sclerosis: prognostic features

Good prognosis features

- female sex
- age: young age of onset (i.e. 20s or 30s)
- relapsing-remitting disease
- sensory symptoms only
- long interval between first two relapses
- complete recovery between relapses

Ways of remembering prognostic features

- the typical patient carries a better prognosis than an atypical presentation

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **20.9%**

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110 ✗

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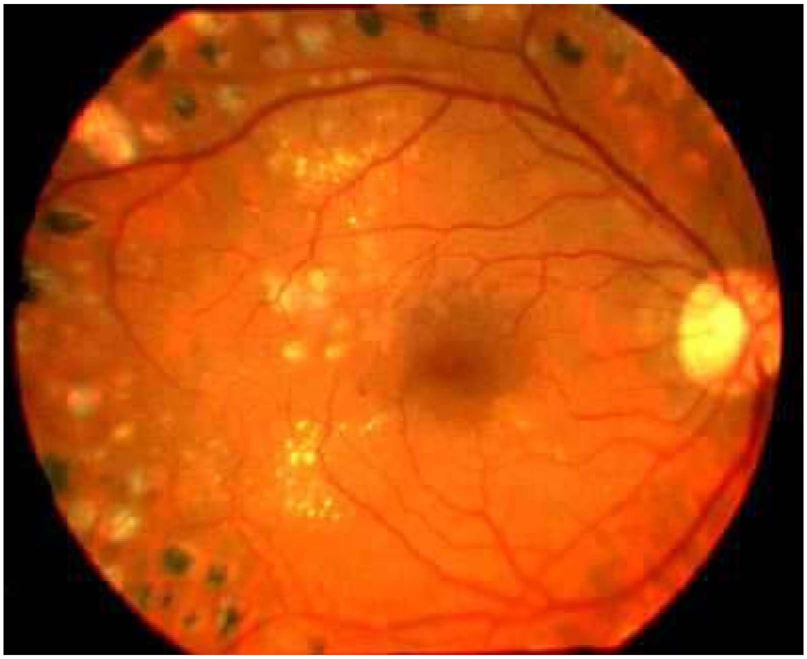
Back to top

A 64-year-old man presents with visual loss. He is on holiday in the United Kingdom; his English is limited and obtaining a history is difficult. He appears generally well in himself and does not appear to be in any obvious pain. Retinal photography is undertaken and shown below:



Which of the following is the most likely diagnosis?

○ Age-related macular degeneration

○ Central retinal artery occlusion

○ Central retinal vein occlusion

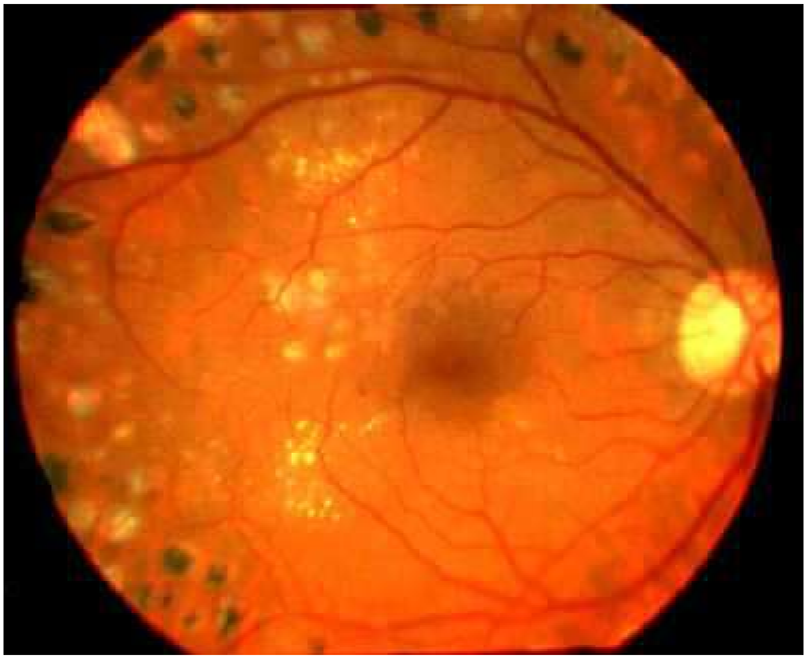
○ Diabetic retinopathy

○ Anterior ischaemic optic neuropathy

Submit answer

Reference ranges

A 64-year-old man presents with visual loss. He is on holiday in the United Kingdom; his English is limited and obtaining a history is difficult. He appears generally well in himself and does not appear to be in any obvious pain. Retinal photography is undertaken and shown below:



Which of the following is the most likely diagnosis?

Age-related macular degeneration	31%
Central retinal artery occlusion	9%
Central retinal vein occlusion	12%
Diabetic retinopathy	39%
Anterior ischaemic optic neuropathy	8%

The correct answer is diabetic retinopathy. This photo is classic of the condition. Microaneurysms are visible, as well as some central macular oedema. A number of exudative areas - 'cotton-wool spots' - are also visible. As the condition progresses, more neovascularization is expected.

Age-related macular degeneration is incorrect. The 'dry' form would characteristically appear with deposits of drusen around the macula, rather than the vascular changes seen here. The 'wet' form will have some vascular changes, but this will appear as choroidal neovascularisation rather than the various microaneurysms seen here.

Central retinal artery occlusion is not consistent with the photo above - it would typically show a cherry red spot, where the centre of the macula is red, but surrounding retina is pale due to lack of blood flow. The retina in general may appear pale.

Central retinal vein occlusion would present with widespread flame haemorrhages rather than the smaller changes seen here, as well as dilated and tortuous veins, which are not really seen in this photo.

Ischaemic optic neuropathy would also look different to the photo above, presenting with a pale, oedematous optic disc. The vascular changes and exudates seen here would not be present.

👍 👎 Discuss (6) Improve

Next question

Diabetic retinopathy

Diabetic retinopathy is the most common cause of blindness in adults aged 35-65 years-old.

Pathophysiology

Hyperglycaemia is thought to cause increased retinal blood flow and abnormal metabolism in the retinal vessel walls. This precipitates damage to endothelial cells and pericytes

Endothelial dysfunction leads to increased vascular permeability which causes the characteristic exudates seen on fundoscopy. Pericyte dysfunction predisposes to the formation of microaneurysms. Neovasculization is thought to be caused by the production of growth factors in response to retinal ischaemia

Classification

Patients are typically classified into those with non-proliferative diabetic retinopathy (NPDR), proliferative retinopathy (PDR) and maculopathy.

Non-proliferative diabetic retinopathy

Mild NPDR

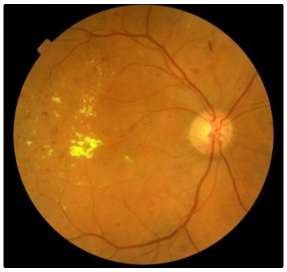
- 1 or more microaneurysm

Moderate NPDR

- microaneurysms
- blot haemorrhages
- hard exudates
- cotton wool spots ('soft exudates' - represent areas of retinal infarction), venous beading/looping and intraretinal microvascular abnormalities (IRMA) less severe than in severe NPDR

Severe NPDR

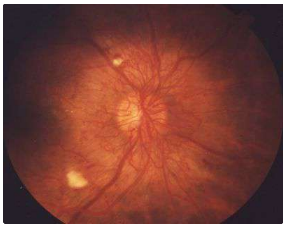
- blot haemorrhages and microaneurysms in 4 quadrants
- venous beading in at least 2 quadrants
- IRMA in at least 1 quadrant



Proliferative diabetic retinopathy

Key features

- retinal neovascularisation - may lead to vitrous haemorrhage
- fibrous tissue forming anterior to retinal disc
- more common in Type I DM, 50% blind in 5 years



Maculopathy

Key features

- based on location rather than severity, anything is potentially serious
- hard exudates and other 'background' changes on macula

- check visual acuity
- more common in Type II DM

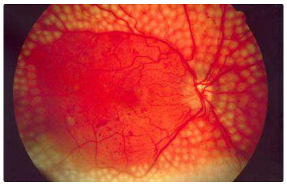
Management

All patients
- optimise glycaemic control, blood pressure and hyperlipidemia
- regular review by ophthalmology

Maculopathy
- if there is a change in visual acuity then intravitreal vascular endothelial growth factor (VEGF) inhibitors

Non-proliferative retinopathy
- regular observation
- if severe/very severe consider panretinal laser photocoagulation

Proliferative retinopathy
- panretinal laser photocoagulation
    - following treatment around 50% of patients develop a noticeable reduction in their visual fields due to the scarring of peripheral retinal tissue
    - other complications include a decrease in night vision (rods are predominantly responsible for vision in low light conditions, the majority of rod cells are located in the peripheral retina), a generalised decrease in visual acuity and macular oedema
- intravitreal VEGF inhibitors
    - often now used in combination with panretinal laser photocoagulation
    - examples include ranibizumab
    - strong evidence base suggests they both slow progression of proliferative diabetic retinopathy and improve visual acuity
- if severe or vitreous haemorrhage: vitreoretinal surgery



Next question ›

Textbooks

High-yield textbook

Extended textbook

Score: **20.9%**

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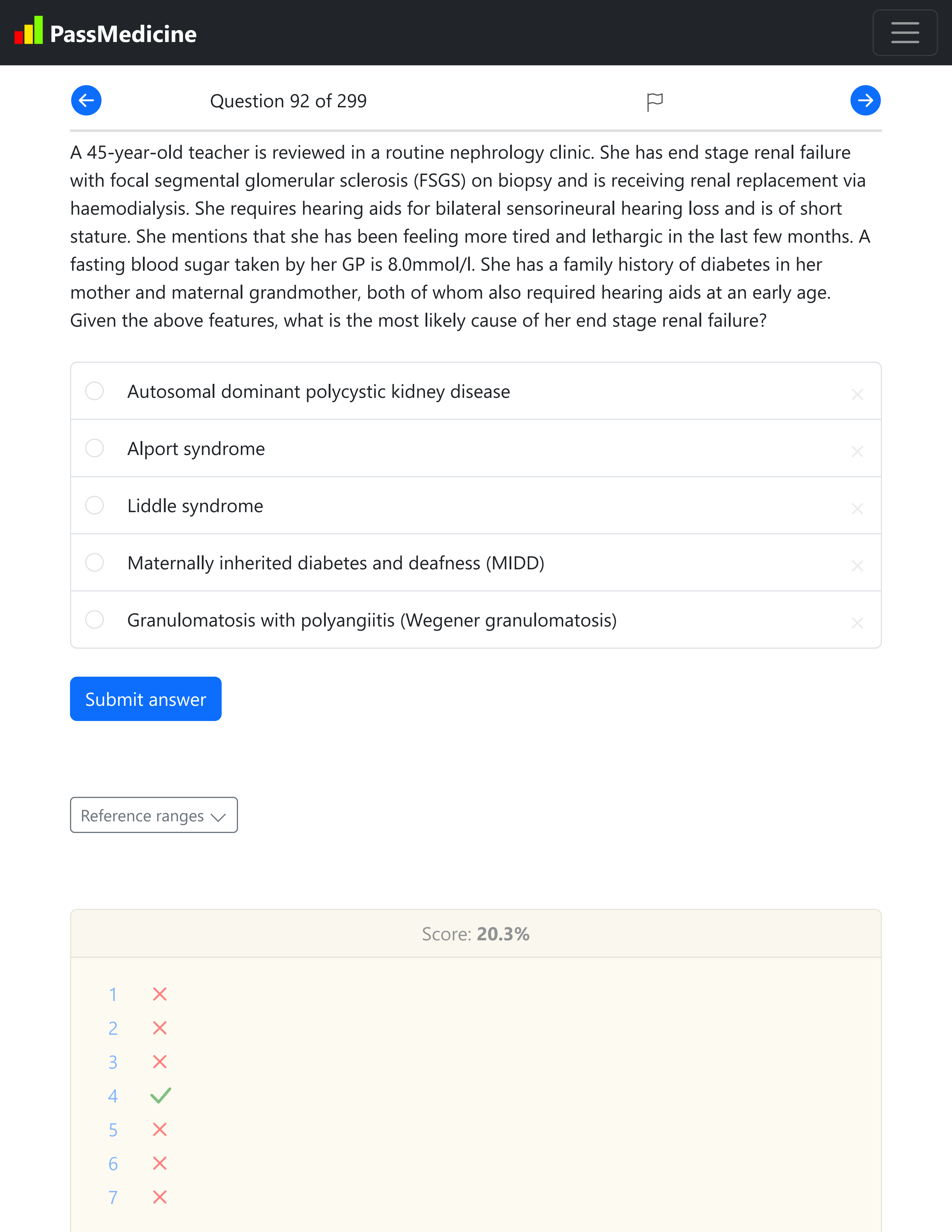

Question 92 of 299

A 45-year-old teacher is reviewed in a routine nephrology clinic. She has end stage renal failure with focal segmental glomerular sclerosis (FSGS) on biopsy and is receiving renal replacement via haemodialysis. She requires hearing aids for bilateral sensorineural hearing loss and is of short stature. She mentions that she has been feeling more tired and lethargic in the last few months. A fasting blood sugar taken by her GP is 8.0mmol/l. She has a family history of diabetes in her mother and maternal grandmother, both of whom also required hearing aids at an early age. Given the above features, what is the most likely cause of her end stage renal failure?

- ○ Autosomal dominant polycystic kidney disease ×
- ○ Alport syndrome ×
- ○ Liddle syndrome ×
- ○ Maternally inherited diabetes and deafness (MIDD) ×
- ○ Granulomatosis with polyangiitis (Wegener granulomatosis) ×

Score: **20.3%**

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A 45-year-old teacher is reviewed in a routine nephrology clinic. She has end stage renal failure with focal segmental glomerular sclerosis (FSGS) on biopsy and is receiving renal replacement via haemodialysis. She requires hearing aids for bilateral sensorineural hearing loss and is of short stature. She mentions that she has been feeling more tired and lethargic in the last few months. A fasting blood sugar taken by her GP is 8.0mmol/l. She has a family history of diabetes in her mother and maternal grandmother, both of whom also required hearing aids at an early age. Given the above features, what is the most likely cause of her end stage renal failure?

Autosomal dominant polycystic kidney disease	1%
Alport syndrome	63%
Liddle syndrome	1%
Maternally inherited diabetes and deafness (MIDD)	35%
Granulomatosis with polyangiitis (Wegener granulomatosis)	1%

Alport syndrome and GPA could also cause end stage renal failure and deafness. However, diabetes is not a feature of Alport syndrome and GPA is not a directly heritable disease.

ADPKD causes end stage renal failure with an autosomal dominant pattern of inheritance but would not explain the other features.

Liddle syndrome is a genetic cause of hypertension due to mutations in the epithelial sodium channel in the distal convoluted tubule.

Discuss (10) Improve

Next question

Maternally inherited diabetes and deafness (MIDD)

MIDD is a mitochondrial disease most commonly caused by the m.3243A>G mutation. The classical features are of sensorineural hearing loss and diabetes but a number of other systems can also be involved. It affects up to 1% of all patients with diabetes.

A hallmark of any mitochondrial disease is the maternal inheritance of the condition.

The same m.3243A>G mutation causes the MELAS syndrome (mitochondrial encephalomyopathy, lactic acidosis and stroke-like symptoms) highlighting the variable presentation of different patients with the same mutation.

Clinical features include:
- Diabetes
- Sensorineural hearing loss
- Stroke-like symptoms
- Retinal dystrophy
- Proximal myopathy
- Cardiomyopathy, arrhythmias
- End stage renal failure - most often FSGS pattern on biopsy
- Short stature, low BMI

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20.9%**

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A 55-year-old female is referred to the neurology clinic with fatigue and muscle weakness, particularly affecting the lower limbs causing difficulty walking. The patient has had several similar episodes in the past with one such episode associated with painful, reduced visual acuity. Previous episodes would typically last a few weeks before spontaneously resolving.

On examination today the patient has spasticity weakness of the lower limbs with reduced sensation distally. Cranial nerve and spinal examination are both normal.

An MRI brain is performed showing evidence of disseminated brain lesions and further tests confirm unpaired oligoclonal bands in the CSF, but not in the serum.

Which of the following is associated with a poor prognosis in the patient's most likely diagnosis?

- ○ Female sex
- ○ Long interval between first two relapses
- ○ Older age of onset
- ○ Presence of oligoclonal bands in the CSF at time of diagnosis
- ○ Relapsing-remitting disease

Submit answer

Reference ranges

Score: **20.3%**

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On examination today the patient has spasticity weakness of the lower limbs with reduced sensation distally. Cranial nerve and spinal examination are both normal.

An MRI brain is performed showing evidence of disseminated brain lesions and further tests confirm unpaired oligoclonal bands in the CSF, but not in the serum.

Which of the following is associated with a poor prognosis in the patient's most likely diagnosis?

Female sex	7%
Long interval between first two relapses	2%
Older age of onset	75%
Presence of oligoclonal bands in the CSF at time of diagnosis	10%
Relapsing-remitting disease	6%

Older age of onset is associated with a poor prognosis in multiple sclerosis

Important for me   Less important

This patient meets the definition for multiple sclerosis (MS) with the dissemination of lesions of the CNS, brain and/or spinal cord (motor and sensory disruption, optic neuritis, etc.) in time and space with characteristic findings on MRI and of unpaired oligoclonal bands in the CSF. Older age onset is associated with a poorer prognosis in MS when compared to patients who present at a younger age.

Females generally have a better prognosis in MS, this patient's likely diagnosis, when compared to male patients.

Longer intervals between a patient's first two relapses are associated with an improved prognosis in MS.

The presence of CSF oligoclonal bands at the time of MS diagnosis has not been linked to the condition's progression or prognosis.

Of the four MS subtypes/patterns, relapsing-remitting is associated with the best prognosis (other subtypes: secondary progressive disease, primary progressive disease, progressive-relapsing disease).

👍 👎 Discuss (1) Improve

Next question >

Multiple sclerosis: prognostic features

Good prognosis features
- female sex
- age: young age of onset (i.e. 20s or 30s)
- relapsing-remitting disease
- sensory symptoms only
- long interval between first two relapses
- complete recovery between relapses

Ways of remembering prognostic features
- the typical patient carries a better prognosis than an atypical presentation

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20.9%**

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Question 94 of 299

An eighty-year-old lady is reviewed. She has a complex past medical history of chronic kidney disease, hypertension, type II diabetes, osteoporosis, Paget's disease and epilepsy, taking ramipril, atorvastatin, sodium bicarbonate, linagliptin, zoledronic acid and phenytoin. On examination, she has asymptomatic cervical lymphadenopathy, with no other abnormal findings. Which medication could be causing this?

- ○ Phenytoin ×
- ○ Sodium bicarbonate ×
- ○ Atorvastatin ×
- ○ Linagliptin ×
- ○ Zoledronic acid ×

Submit answer

Reference ranges

Score: **20.3%**

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Question 94 of 299

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Phenytoin	89%
Sodium bicarbonate	2%
Atorvastatin	1%
Linagliptin	1%
Zoledronic acid	7%

Phenytoin can cause lymphadenopathy

Important for me Less important

Phenytoin is known to be an iatrogenic cause of lymphadenopathy, as well as carbamazepine, allopurinol and sulphonamides. Be aware, that a more systemic drug reaction is more often noted with medications, with lymphadenopathy as an associated, but not isolated, sign e.g. DRESS and serum sickness.

Sodium bicarbonate, atorvastatin, linagliptin and zoledronic acid are not commonly associated with lymphadenopathy.

Discuss (2) Improve

Next question

Phenytoin

Phenytoin is used in the management of seizures.

Mechanism of action

- binds to sodium channels increasing their refractory period

Adverse effects

Phenytoin is associated with a large number of adverse effects. These may be divided into acute, chronic, idiosyncratic and teratogenic. Phenytoin is also an inducer of the P450 system.

Acute

- initially: dizziness, diplopia, nystagmus, slurred speech, ataxia
- later: confusion, seizures

Chronic

- common: gingival hyperplasia (secondary to increased expression of platelet derived growth factor, PDGF), hirsutism, coarsening of facial features, drowsiness
- megaloblastic anaemia (secondary to altered folate metabolism)
- peripheral neuropathy
- enhanced vitamin D metabolism causing osteomalacia
- lymphadenopathy
- dyskinesia

Idiosyncratic

- fever
- rashes, including severe reactions such as toxic epidermal necrolysis
- hepatitis
- Dupuytren's contracture*
- aplastic anaemia
- drug-induced lupus

Teratogenic

- associated with cleft palate and congenital heart disease

Monitoring

Phenytoin levels do not need to be monitored routinely but **trough levels, immediately before dose** should be checked if:

- adjustment of phenytoin dose

- suspected toxicity
- detection of non-adherence to the prescribed medication

*although not listed in the BNF

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

BNF 👍 18 👎 8

Phenytoin

Suggest link Report broken link

Score: **20.9%**

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A 62-year-old man is reviewed following a thyroidectomy for follicular thyroid cancer without metastatic spread. He started developing first twitching and then cramps following the procedure, primarily affecting his legs. He finds this to be quite painful and has never experienced anything like this previously. He reports that the surgical registrar told him that the procedure went well without complication. He also has a past medical history of hypertension, vitamin D deficiency, depression and gout. He was taking ramipril, cholecalciferol with calcium and allopurinol. All his regular medication have been held for one day due to surgery. What is the most likely cause of his cramps?

○ Hypocalcaemia

○ Hypercalcaemia

○ Hypophosphataemia

○ Hyperphosphataemia

○ Hypokalaemia

Submit answer

Reference ranges

Score: **20.3%**

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A 62-year-old man is reviewed following a thyroidectomy for follicular thyroid cancer without metastatic spread. He started developing first twitching and then cramps following the procedure, primarily affecting his legs. He finds this to be quite painful and has never experienced anything like this previously. He reports that the surgical registrar told him that the procedure went well without complication. He also has a past medical history of hypertension, vitamin D deficiency, depression and gout. He was taking ramipril, cholecalciferol with calcium and allopurinol. All his regular medication have been held for one day due to surgery. What is the most likely cause of his cramps?

Hypocalcaemia	88%
Hypercalcaemia	4%
Hypophosphataemia	4%
Hyperphosphataemia	1%
Hypokalaemia	2%

Following thyroid removal, checking calcium is important to assess for hypoparathyroidism

Important for me Less important

The patient has undergone an thyroidectomy and developed symptoms consistent with hypocalcaemia soon after. This is most likely due to iatrogenic primary hypoparathyroidism as the parathyroid glands can easily be removed or have vascular damage from thyroid surgery. The patient would likely have low parathyroid levels, as well as low calcium and high phosphate. The low calcium would need to be replaced and then symptoms would resolve.

Discuss (1) Improve

Next question

Subtotal thyroidectomy

Surgery complications

- hypothyroidism: most common
- immediate haematoma (airway compression)
- hypoparathyroidism → hypocalcaemia (paraesthesia, spasm)
- recurrent laryngeal nerve palsy (hoarseness, bovine cough)
- superior laryngeal nerve injury (loss of pitch)
- thyroid crisis (fast AF, pulmonary oedema)

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20.9%**

1 ✗
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An 82-year-old female is brought into your falls clinic by her daughter after her third fall this year. No fractures were sustained and she appears to have no significant head injuries. The fall appears mechanical in nature. She currently lives with her daughter, who reports the patient's mobility to be progressively deteriorating, from full independence and no exercise limitations 1 year ago to restrictions at 50-70 yards now, limited by knee pain secondary to osteoarthritis. Her other past medical history includes hypertension, type 2 diabetes mellitus, chronic kidney disease and previous gallstones.

You note she is withdrawn and makes little eye contact. Her voice is quiet. When you ask her whether she is low in mood, she does not respond. She reports no suicidal ideations but has little hope for the future. She asks you to 'not worry about it', as she 'has been the same way for several months now'. However, the patient does seem amenable to some kind of treatment.

On the Beck depression scale, she scores 11/63 (0-13 = no or minimal depression), on the geriatric depression scale, she scores 11/15 (greater than 10 = indicative of depression) and mini-mental state examination, she scores 19/30 (20-26 = mild cognitive impairment, 10-19 = moderate cognitive impairment). Routine investigations including B12, folate, thyroid function, liver function tests and bone profile are unremarkable.

What is the most appropriate treatment pathway?

○ Citalopram

○ Amitriptyline

○ Mirtazapine

○ Donepezil

○ No treatment required

Submit answer

Reference ranges

Score: **20.3%**

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What is the most appropriate treatment pathway?

Citalopram	37%
Amitriptyline	4%
Mirtazapine	28%
Donepezil	25%
No treatment required	6%

Depression in elderly can depress cognitive function, hence cognition may be inaccurately depressed on measurement scales. Donepezil is thus not indicated on this measure alone. In elderly patients, GDS is more appropriate than Becks depression scale, as the latter focuses heavily on somatic symptoms that frequently under-score depression in elderly patients. This patient is describing depression without suicidal ideations. Tricyclic antidepressants and SSRIs are similarly efficacious in elderly patients but the first line of treatment for depression in the elderly is a SSRI such as citalopram, mainly due to the lack of interactions with primarily P450 enzymes[1]. TCAs produce significantly greater anticholinergic side effects. MIrtazapine and sertraline failed to demonstrate a significant benefit compared to placebo in Alzheimers patients with depression.

1. Rodda J, Walker Z, Carter J. Depression in older adults. BMJ 2011; 34:d5219

Discuss (9) | Improve

Next question >

Depression in older people ★

Older patients are less likely to complain of depressed mood

Features

- physical complaints (e.g. hypochondriasis)
- agitation
- insomnia

Management

- SSRIs are first line (adverse side-effect profile of TCAs more of an issue in the elderly)

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20.9%**

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A 46-year-old white Caucasian female presents to the Emergency Department after waking up unable to walk and complains about problems with vision in her right eye. The patient says she is normally extremely healthy and the only time she has ever visited her GP was when she had an episode of diarrhoea and vomiting about a week ago. She is understandably distressed as she has not experienced any symptoms before and has not been previously diagnosed with any medical conditions. She has no drug or family history. Nursing staff report to you that she has been given a pad on her bed as she is incontinent of urine, which she is tearfully embarrassed by.

On examination of her cranial nerves, a right relative afferent papillary defect is noted. Visual acuity and colour vision are 6/6 with 17/17 Ishihara plates on the left, 6/60 with 0/17 Ishihara plates on the right. She reports no diplopia with a full range of eye movements and normal facial sensation. Facial movements are normal. Fundoscopy was unremarkable. Examination of the patient's upper and lower limb power demonstrated normal tone with 2-/5 power on the left arm and leg in all movements; and 4-/5 all movements in right arm and leg. There is a patchy loss of sensation to cotton wool on right lateral wrist and anterior aspect left lateral shin. Both plantars are upgoing. Reflexes are brisk at knee and ankles bilaterally. Anal tone and saddle sensation are intact. The patient's cognitive state is normal on superficial examination.

An urgent MRI head and whole spine demonstrates abnormal high signal in the cervical cord from C3 to C7. Her blood tests were mislabelled and hence unavailable. A lumbar puncture was performed with the following results:

WCC	12 mm^3
RBC	<1 mm^3
Protein	0.9 g/l
Glucose	5.2 mmol/l (10.2 mmol/l serum)
Oligoclonal bands	awaited
Viral PCR	awaited

What is the most likely diagnosis?

○ Idiopathic intracranial hypertension

○ Transverse myelitis

○ Miller-Fisher syndrome ×

○ Multiple sclerosis ×

○ Devic's disease ×

Submit answer

Reference ranges ⌄

Score: **20.3%**

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WCC	12 mm/³
RBC	<1 mm/³
Protein	0.9 g/l
Glucose	5.2 mmol/l (10.2 mmol/l serum)
Oligoclonal bands	awaited
Viral PCR	awaited

What is the most likely diagnosis?

Idiopathic intracranial hypertension 1%

Transverse myelitis 18%

Miller-Fisher syndrome	21%
Multiple sclerosis	20%
Devic's disease	40%

A first episode of an optic neuritis and myelitis occurring together is suggestive of a central neuroinflammatory cause. A full range of eye movements makes Miller-Fisher syndrome unlikely (and any ataxia demonstrated is likely secondary to limb weakness). Although there is the temptation of diagnosing multiple sclerosis in a patient with features disseminated in place, the keys to the answer are the sites of the lesion (optic nerve and cervical cord) and the long length of cord involvement beyond 3 vertebral levels, much more suggestive of neuromyelitis optic or Devic's disease. The diagnosis would be clinched by a positive aquaporin 4 antibody.

👍 👎 Discuss (12) Improve

Next question >

Neuromyelitis optica ★

Neuromyelitis optica (NMO) is a monophasic or relapsing-remitting demyelinating CNS disorder Although previously thought to be a variant of multiple sclerosis, it is now recognised to be a distinct disease, particularly prevalent in Asian populations. It typically involves the optic nerves and cervical spine, with imaging of the brain frequently normal. Vomiting is also a common presenting complaint.

Diagnosis requires bilateral optic neuritis, myelitis and 2 of the following 3 criteria:
- 1. Spinal cord lesion involving 3 or more spinal levels
- 2. Initially normal MRI brain
- 3. Aquaporin 4 positive serum antibody

Management
- immunosuppressant e.g. with anti-CD20 agent rituximab)

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20.9%**

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Question 98 of 299

A 42-year-old woman presents with 6 days of drowsiness and a gradual onset headache, initially starting in the occipital region and radiating to the apex. She has recently returned from Australia in holiday one week ago but has complained of poor appetite after a cough and cold since landing. She has no past medical history, takes no regular medications except the oral contraceptive pill. She is a non-smoker and drinks minimally. On examination, the patient has a full range of neck movements and no photophobia. Examination of her limbs is unremarkable. You request a CT head without contrast, which demonstrates two small areas of subarachnoid blood in right convexity. She denies any recent head trauma. Which investigation will most likely provide the conclusive diagnosis?

- ○ CT head with contrast
- ○ MRI head
- ○ MR venogram
- ○ Lumbar puncture with xanthochromia
- ○ CT angiography

Submit answer

Reference ranges

Score: **20.3%**

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2 ✗
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CT head with contrast	1%
MRI head	3%
MR venogram	35%
Lumbar puncture with xanthochromia	14%
CT angiography	47%

MR Venogram is the gold standard test for diagnosing venous sinus thrombosis

Important for me Less important

The patient presents with a non-specific headache on a background of a number of different risk factors for venous thromboembolic disease: recent long-haul flight, likely dehydration from poor oral intake, oral contraceptive pill. A generalised headache is present in almost 90% of all patients with cerebral venous thrombosis (CVT)[1]. The headache is caused by impaired absorption of cerebrospinal fluid due to the sinus thrombus, resulting in increased intracranial pressure. CVTs can also present with seizures, encephalopathy and focal symptoms, but be aware that the presenting clinical features can be extremely variable.

A plain CT head can sometimes demonstrate signs of CVT in one-third of patients. A dense triangle sign is often seen in the posterior superior sagittal sinus. In addition, the empty or negative delta sign[2], is seen on a contrasted CT head where a central area is lacking contrast flow due to thrombus obstruction. However, MR venogram (CT venogram in the hands of highly skilled radiologists) is the most sensitive imaging modality.

This scenario is a useful demonstration that not all subarachnoid blood equates to a spontaneously ruptured aneurysm. Cerebral venous thrombosis is a classic cause of intraparenchymal and subarachnoid haemorrhages, particularly if around the convexity[3]. In addition, the description of the headache is atypical for an aneurysmal subarachnoid haemorrhage: the headache is of gradual onset, persists for days, with no meningism. Although it may seem illogical, treatment with anticoagulation will treat the sinus thrombus, resulting in improved CSF flow, while the subarachnoid blood will slowly resolve.

1. Ferro JM, CanhÃ£o P, Stam J et al. Prognosis of cerebral vein and dural sinus thrombosis: results of the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT). Stroke. 2004;35(3):664
2. Lee EJ. The empty delta sign. Radiology. 2002;224(3):788
3. Sztajzel R, Coeytaux A, Dehdashti AR et al. Subarachnoid hemorrhage: a rare presentation of cerebral venous thrombosis. Headache. 2001;41(9):889

👍 👎 Discuss (12) Improve

Next question >

Intracranial venous thrombosis

Overview

- can cause cerebral infarction, much lesson common than arterial causes
- 50% of patients have isolated sagittal sinus thromboses - the remainder have coexistent lateral sinus thromboses and cavernous sinus thromboses

General features

Common features

- headache (may be sudden onset)
- nausea & vomiting
- reduced consciousness

Investigation

- MRI venography is the gold standard

    - CT venography is an alternative
- non-contrast CT head is normal in around 70% of patients
- D-dimer levels may be elevated

Management

- anticoagulation
    - typically with low molecular weight heparin acutely
    - warfarin is still generally used for longer term anticoagulation

Specific syndromes

Sagittal sinus thrombosis

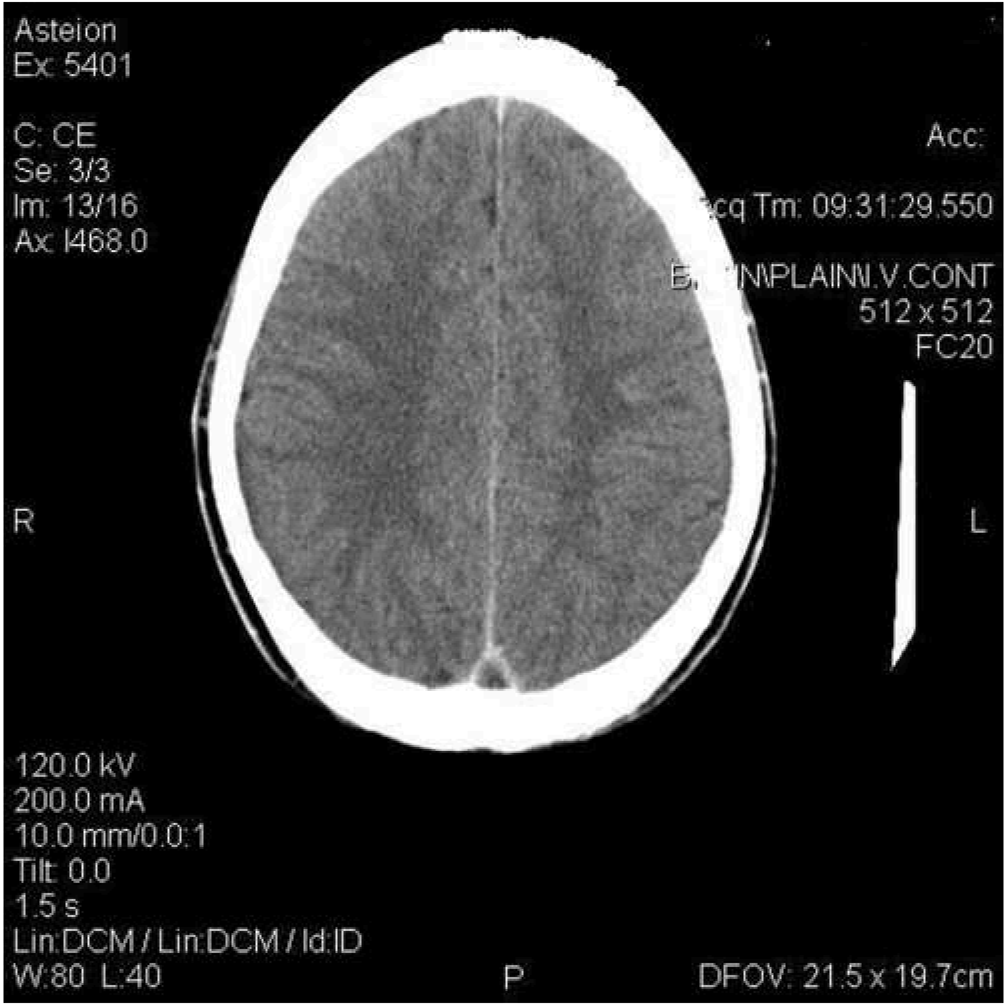
- may present with seizures and hemiplegia
- parasagittal biparietal or bifrontal haemorrhagic infarctions are sometimes seen
- 'empty delta sign' seen on venography

Cavernous sinus thrombosis

- other causes of cavernous sinus syndrome: local infection (e.g. sinusitis), neoplasia, trauma
- periorbital erythema and oedema
- ophthalmoplegia: 6th nerve damage typically occurs before 3rd & 4th
- trigeminal nerve involvement may lead to hyperaesthesia of upper face and eye pain
- central retinal vein thrombosis

Lateral sinus thrombosis

- 6th and 7th cranial nerve palsies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© Image used on license from Radiopaedia

CT with contrast demonstrating a **superior sagittal sinus thrombosis** showing the typical empty delta sign. Look at the 'bottom' of the scan for the triangular shaped dural sinus. This should normally be white due to it being filled with contrast. The empty delta sign occurs when the thrombus fails to enhance within the dural sinus and is outlined by enhanced collateral channels in the falx. This sign is seen in only about 25%-30% of cases but is highly diagnostic for sagittal sinus thrombosis

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **20.9%**

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3 ✗
4 ✓
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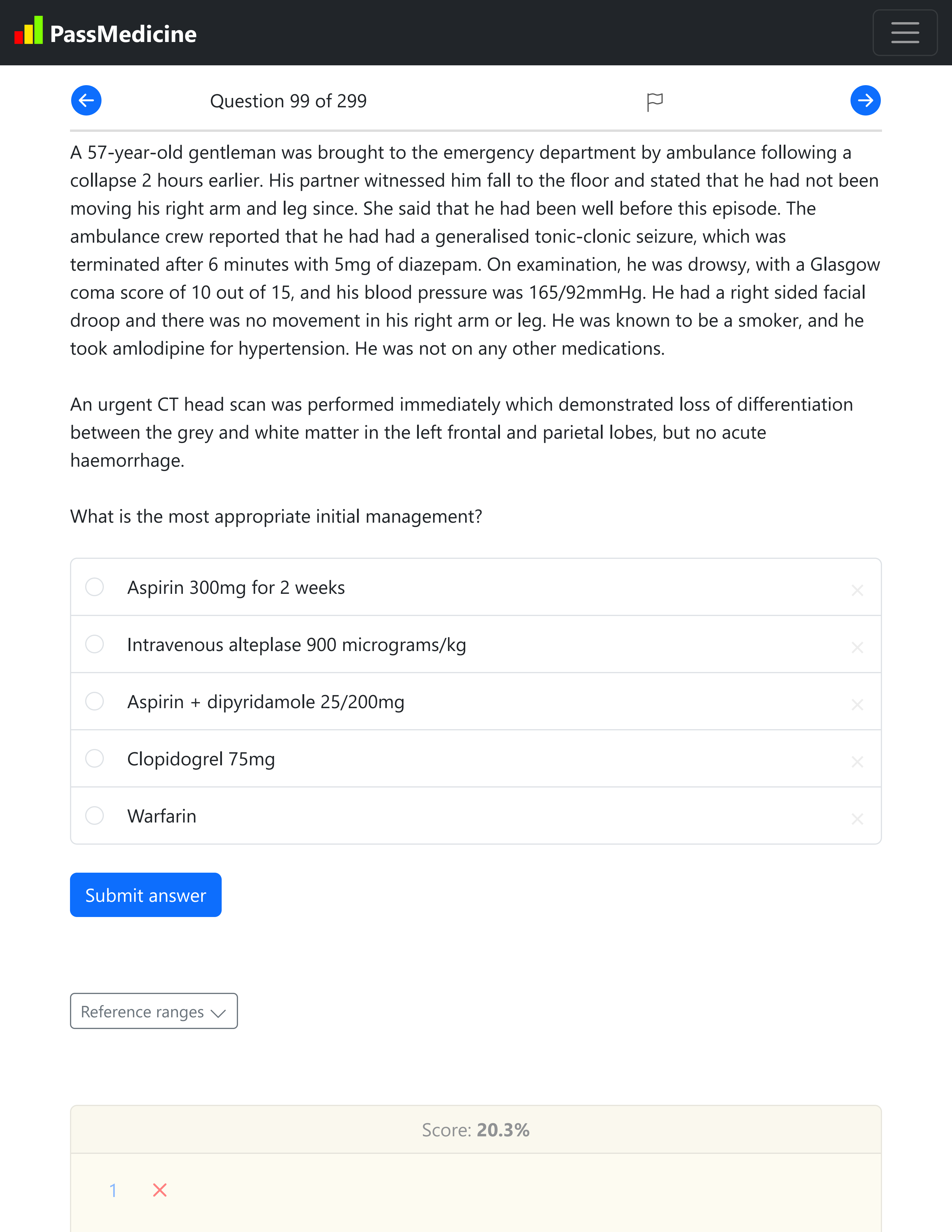

A 57-year-old gentleman was brought to the emergency department by ambulance following a collapse 2 hours earlier. His partner witnessed him fall to the floor and stated that he had not been moving his right arm and leg since. She said that he had been well before this episode. The ambulance crew reported that he had had a generalised tonic-clonic seizure, which was terminated after 6 minutes with 5mg of diazepam. On examination, he was drowsy, with a Glasgow coma score of 10 out of 15, and his blood pressure was 165/92mmHg. He had a right sided facial droop and there was no movement in his right arm or leg. He was known to be a smoker, and he took amlodipine for hypertension. He was not on any other medications.

An urgent CT head scan was performed immediately which demonstrated loss of differentiation between the grey and white matter in the left frontal and parietal lobes, but no acute haemorrhage.

What is the most appropriate initial management?

○ Aspirin 300mg for 2 weeks ×

○ Intravenous alteplase 900 micrograms/kg ×

○ Aspirin + dipyridamole 25/200mg ×

○ Clopidogrel 75mg ×

○ Warfarin ×

Score: **20.3%**

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An urgent CT head scan was performed immediately which demonstrated loss of differentiation between the grey and white matter in the left frontal and parietal lobes, but no acute haemorrhage.

What is the most appropriate initial management?

Aspirin 300mg for 2 weeks	36%
Intravenous alteplase 900 micrograms/kg	57%
Aspirin + dipyridamole 25/200mg	3%
Clopidogrel 75mg	3%
Warfarin	1%

This patient presents with a sudden onset of neurological deficit, with lateralising signs. This history is strongly suggestive of a stroke, and the CT head findings suggest an ischaemic cause.

Acute management of ischaemic stroke involves keeping the patient nil by mouth until swallow assessment is performed, nasogastric tube insertion if dysphagia is problematic, and admission to a high dependency stroke unit for close monitoring.

If the presentation is within 4.5 hours of onset thrombolysis with a recombinant tissue plasminogen-activator (such as alteplase) can be considered, which has been shown to improve neurological outcome. This patient presents at 2 hours, but his seizure at the onset of stroke precludes the use of thrombolytic therapy (see table).

If the presentation is beyond 4.5 hours, or thrombolysis is contraindicated, aspirin should be commenced at a dose of 300mg for 2 weeks as soon as possible. Lifelong anti-platelet therapy should be continued thereafter. Aspirin in combination with dipyridamole is only recommended if clopidogrel is contraindicated. Warfarin is not indicated in the immediate management of stroke, though may be started if the patient is shown to have atrial fibrillation. This is usually delayed for 2 weeks, due to the risk of haemorrhagic transformation if given before this.

Discuss (12) Improve

Next question >

Stroke: management of acute ischaemic stroke

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
    - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
    - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:

- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.

- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
    - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
    - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%
- it should be performed as soon as possible within 7 days

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 11 👎 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 16

Stroke and TIA guidelines

Age and Ageing 👍 21 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

2023 National Clinical Guideline for Stroke

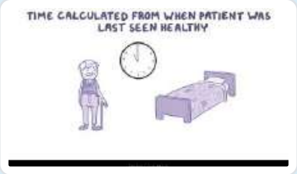
NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link

Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia
Acute Ischaemic Stroke

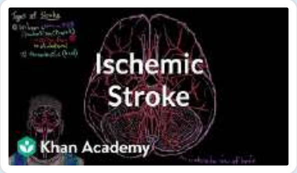
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

Khan Academy - YouTube

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Report broken media

Score: **20.9%**

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Question 100 of 299

A 45-year-old woman has been brought to the Emergency Department by ambulance. Her 20-year-old son found her collapsed on the floor with a note stating that she wished to take her own life. The paramedics have brought all the medication packages that they could find. This included sertraline, atorvastatin and diazepam. Previous medical history includes depression with suicidal ideation and a paracetamol overdose 3 years ago that required admission.

Her 50mg sertraline package shows five empty slots. Her 10mg diazepam package shows 30 empty slots.

On assessment of the patient:

- A - Airway is supported by the anaesthetist with a bag valve mask.
- B - Respiratory rate is being supported up to 12/min. Saturations are maintained at 98% however, the patient desaturates and hyperventilates without assistance.
- C - BP 102/74 mmHg, pulse 94 bpm.
- D - GCS 9, (E1, M5, V3), pupils 4mm bilaterally and reactive.

What is the next step in the management of this patient?

○ Activated charcoal

○ Flumazenil

○ High dose insulin therapy

○ Intubation and ventilation

○ Naloxone infusion

Submit answer

Reference ranges

Score: **20.3%**

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- D - GCS 9, (E1, M5, V3), pupils 4mm bilaterally and reactive.

What is the next step in the management of this patient?

Activated charcoal	3%
Flumazenil	32%
High dose insulin therapy	1%
Intubation and ventilation	61%
Naloxone infusion	3%

Benzodiazepine overdose is best managed supportively and with airway protection and ventilation if needed. Flumazenil should be avoided unless for reversal of anaesthesia

Important for me Less important

Intubation and ventilation is correct. The patient is hypoventilating and unable to protect their airway. The cause is likely an overdose of benzodiazepines which requires organ support whilst the drug is metabolised. Further investigations such as CT head, ECG, paracetamol and salicylate levels will also be important to ensure there is no alternative cause for the sudden drop in GCS.

Activated charcoal is incorrect because the patient has a low GCS. Although activated charcoal can be used in overdose if the patient presents within 60 minutes of ingestion, it should not be administered to a patient with a decreased GCS. Charcoal is an emetic and increases the risk of aspiration in a patient with a low GCS. In this case, the time of consumption of medication is also unknown.

Flumazenil is incorrect because it is not known if this is an isolated benzodiazepine overdose and the patient may have a dependence on benzodiazepines. Flumazenil is rarely used outside of the operating theatre. It is the antidote to benzodiazepines but must not be used in mixed overdoses. If the patient has any risk of seizures then administration of flumazenil will prevent benzodiazepines from providing seizure control. In this case, the patient has likely ingested a considerable dose of benzodiazepines but may have taken a mixed overdose.

High dose insulin therapy is incorrect because this is used in beta-blocker and calcium channel blocker overdose. High dose insulin therapy is the administration of insulin at 1unit/kg. This therapy may overcome myocardial starvation and increase myocardial function without an increase in oxygen demand.

Naloxone infusion is incorrect because there is no suspicion of an opioid overdose. Naloxone is the antidote to opioid toxicity. Although it is entirely appropriate to trial naloxone in suspected mixed overdose and suspicion of opioids, a naloxone infusion should only be started if the patient has shown a response to an initial naloxone bolus. The initial dose is 400mcg, however experienced clinicians may titrate this to affect in chronic opioid users as this can lead to sudden and severe withdrawal symptoms.

Discuss (3) Improve

Next question

Status epilepticus

Status epilepticus is defined as:

- a single seizure lasting >5 minutes, or
- >= 2 seizures within a 5-minute period without the person returning to normal between them

This is a medical emergency. The priority is the termination of seizure activity, which if prolonged will lead to irreversible brain damage.

Management

- ABC
    - airway adjunct
    - oxygen
    - check blood glucose
- First-line drugs are benzodiazepines
    - in the prehospital setting PR diazepam or buccal midazolam may be given
    - in hospital IV lorazepam is generally used. This may be repeated once after 5-10minutes
- If ongoing (or 'established') status it is appropriate to start a second-line agent such as levetiracetam, phenytoin or sodium valproate
    - NICE state 'Take into account that levetiracetam may be quicker to administer and have fewer adverse effects than the alternative options.'
- If no response ('refractory status') within 45 minutes from onset, then the best way to achieve rapid control of seizure activity is induction of general anaesthesia or phenobarbital

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 14 👎 9

2022 Epilepsy guidelines

Suggest link

Report broken link

Score: **20.9%**

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Question 101 of 299

A 72-year-old man is referred to the neurology outpatient department with an insidious onset of tremor by his general practitioner. He has a past medical history of hypertension and recurrent falls. He is on regular amlodipine. He does not smoke or drink alcohol.

On examination, there is bradykinesia and rigidity and a pill-rolling tremor of the right hand. He is unable to make vertical saccades with his eyes when tested. He appears to lung forward when walking.

What is the likely diagnosis?

○ Corticobasal degeneration

○ Dementia with Lewy Bodies

○ Multiple system atrophy

○ Parkinson's disease

○ Progressive supranuclear palsy

Submit answer

Reference ranges

Score: **20.3%**

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A 72-year-old man is referred to the neurology outpatient department with an insidious onset of tremor by his general practitioner. He has a past medical history of hypertension and recurrent falls. He is on regular amlodipine. He does not smoke or drink alcohol.

On examination, there is bradykinesia and rigidity and a pill-rolling tremor of the right hand. He is unable to make vertical saccades with his eyes when tested. He appears to lung forward when walking.

What is the likely diagnosis?

Corticobasal degeneration	1%
Dementia with Lewy Bodies	1%
Multiple system atrophy	4%
Parkinson's disease	16%
Progressive supranuclear palsy	78%

Progressive supranuclear palsy: postural instability, impairment of vertical gaze, parkinsonism, frontal lobe dysfunction

Important for me Less important

Progressive supranuclear palsy is the correct answer. This patient presents with parkinsonism, postural instability and impairment of vertical gaze, which is characteristic of this condition.

Dementia with Lewy Bodies is incorrect. While this can cause parkinsonism, it does not cause gaze palsies and dementia is a prominent feature.

Multiple system atrophy is incorrect. This is another 'parkinson-plus' syndrome but is characterized by autonomic dysfunction and postural instability rather than gaze palsies.

Corticobasal degeneration is incorrect. This is characterized by parkinsonism, apraxia, aphasia and classically 'alien hand syndrome', which results in the failure of the individual to control the movements of their limb resulting from the sensation that the limb is 'foreign'.

👍 👎 Discuss (2) Improve

Next question >

Progressive supranuclear palsy ★

Overview

- aka Steele-Richardson-Olszewski syndrome
- a 'Parkinson Plus' syndrome

Features

- postural instability and falls
    - patients tend to have a stiff, broad-based gait
- impairment of vertical gaze (down gaze worse than up gaze - patients may complain of difficultly reading or descending stairs)
- parkinsonism
    - bradykinesia is prominent
- cognitive impairment
    - primarily frontal lobe dysfunction

Management

- poor response to L-dopa

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20.9%**

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Question 102 of 299

A 65-year-old gentleman presents with left sided arm pain that originates in the shoulder and spreads into the ring and little fingers. This has worsened over the past three weeks to the point that he cannot sleep at night. His GP has trialled co-codamol without any benefit.

He is a previous smoker of 40 pack years, has hypertension and a myocardial infarction 5 years previously. He is currently well but on further questioning reveals that he has lost 2 stone in weight over the last 3 months despite increasing his food intake. He has a productive cough which has occasional streaks of blood in.

On examination, he is able to move his left shoulder, elbow and wrist with a full range of motion and no tenderness but reports that the pain is present whilst not moving. He has some weakness of his left biceps with a diminished reflex and sensation is reduced to the fourth and fifth in response to light touch. The limb has normal pulses and capillary refill time with no swelling. There are crepitations on the left upper zone and he has clubbing present.

Hb	103 g/l	Na$^+$	137 mmol/l
Platelets	456 * 10^9/l	K$^+$	4.2 mmol/l
WBC	13.1 * 10^9/l	Urea	5.6 mmol/l
Neuts	9.8 * 10^9/l	Creatinine	98 µmol/l
Lymphs	0.89 * 10^9/l	CRP	89 mg/l
Eosin	0.25 * 10^9/l		

Chest x-ray	consolidation seen in the left upper zone

What is the likely diagnosis?

○ Bronchial carcinoma

○ Pneumonia

○ Cervical rib

○ Multiple sclerosis

○ Tuberculosis

Submit answer

Reference ranges ⌵

Score: **20.3%**

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101 -

102 -

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Lymphs	0.89 * 10^9/l	CRP	89 mg/l
Eosin	0.25 * 10^9/l		

Chest x-ray	consolidation seen in the left upper zone

What is the likely diagnosis?

- Bronchial carcinoma 83%
- Pneumonia 1%
- Cervical rib 9%
- Multiple sclerosis 0%
- Tuberculosis 5%

Lung carcinoma can present with a brachial neuritis

Important for me Less important

This gentleman presents with a neuralgia pain in the left arm and altered neurology in the dermatome of C8. Of these, pneumonia is unlikely to ever cause this. He does have respiratory symptoms and an extensive smoking history and this excludes multiple sclerosis as there are no other areas of altered neurology. The diminished reflexes suggest a lower motor neuron lesion and C8 is classically affected in a Pancoast tumour. Tuberculosis can rarely infiltrate the brachial plexus but bronchial carcinoma is the most likely. A cervical rib is unlikely to present at this age.

Discuss (3) Improve

Next question >

Brachial neuritis ★

Brachial neuritis is characterized by acute onset of unilateral (occasionally bilateral) severe pain, followed by shoulder and scapular weakness several days later. Sensory changes are usually minimal. There may be subsequent rapid wasting of the arm muscles in accordance to which nerve is involved. Precipitating factors include recent trauma, infection, surgery, or even vaccination. Rarely it may be hereditary. The prognosis is usually good except when the phrenic nerve is involved since this can result in significant breathlessness.

AI + Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Score: **20.9%**

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A 46-year-old lady is admitted to emergency department with a 2 hour history of dysphasia, visual disturbance and weakness. Examination reveals left homonymous hemianopia, left sided hemiparesis with sensory loss and dysphasia. Observations show she is apyrexial with a heart rate of 102/min and blood pressure of 145/82 mmHg.

An urgent CT head and angiography are booked indicating a large infarct in the territory of the middle cerebral artery in the right cerebrum, while the CT angiography is indicative of proximal occlusion.

There is no significant past medical history and the blood investigations for this lady are normal. Her modified Rankin score is 1 and her national institutes of health stroke scale (NIHSS) is 12.

What is the next most appropriate course of action in her management given that we are now four hours from presentation?

- ○ Offer intravenous thrombolysis alone
- ○ Reduce blood pressure via the administration of nifedipine
- ○ Offer thrombectomy with intravenous thrombolysis cover
- ○ Load with clopidogrel
- ○ Treat conservatively with aspirin and secondary prevention

Submit answer

Reference ranges

Score: **20.3%**

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There is no significant past medical history and the blood investigations for this lady are normal. Her modified Rankin score is 1 and her national institutes of health stroke scale (NIHSS) is 12.

What is the next most appropriate course of action in her management given that we are now four hours from presentation?

Offer intravenous thrombolysis alone	14%
Reduce blood pressure via the administration of nifedipine	1%
Offer thrombectomy with intravenous thrombolysis cover	81%
Load with clopidogrel	1%
Treat conservatively with aspirin and secondary prevention	3%

A combination of thrombolysis AND thrombectomy is recommend for patients with an acute ischaemic stroke who present within 4.5 hours

Important for me Less important

This question highlights the importance of understanding the timing scales applicable to stroke management. The time limit for offering thrombolysis is 4.5 hours currently, while the thrombectomy window (provided that certain anatomical and functional criteria are met) is that of 6 hours.

This young lady fulfils the criteria for thrombectomy and thrombolysis and it is important to note that thrombolysis is not contra-indicated but actually encouraged en-route to thrombectomy.

There is no indication in reducing this lady's blood pressure as it is well below 180/110 mmHg.

There is no place for clopidogrel loading in the acute management of stroke, with the exception of aspirin allergy.

As this lady fulfils all the criteria, has a reasonable pre-stroke score (modified Rankin score <3) and a high stroke score indicating the impact of the occlusion to her functioning (NIHSS >5), thrombectomy with thrombolysis cover is the most appropriate course of action.

👍 👎 Discuss (8) Improve

Next question >

Stroke: management of acute ischaemic stroke

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
    - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
    - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:

- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.

- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
    - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
    - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 11 👎 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 16

Stroke and TIA guidelines

Age and Ageing 👍 21 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

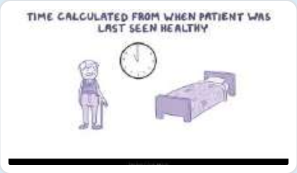
2023 National Clinical Guideline for Stroke

NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia
Acute Ischaemic Stroke

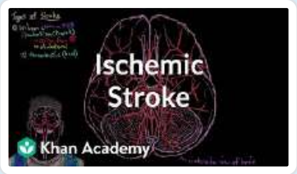
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

Khan Academy - YouTube 👍 1 👎 1

Report broken media

Score: **20.9%**

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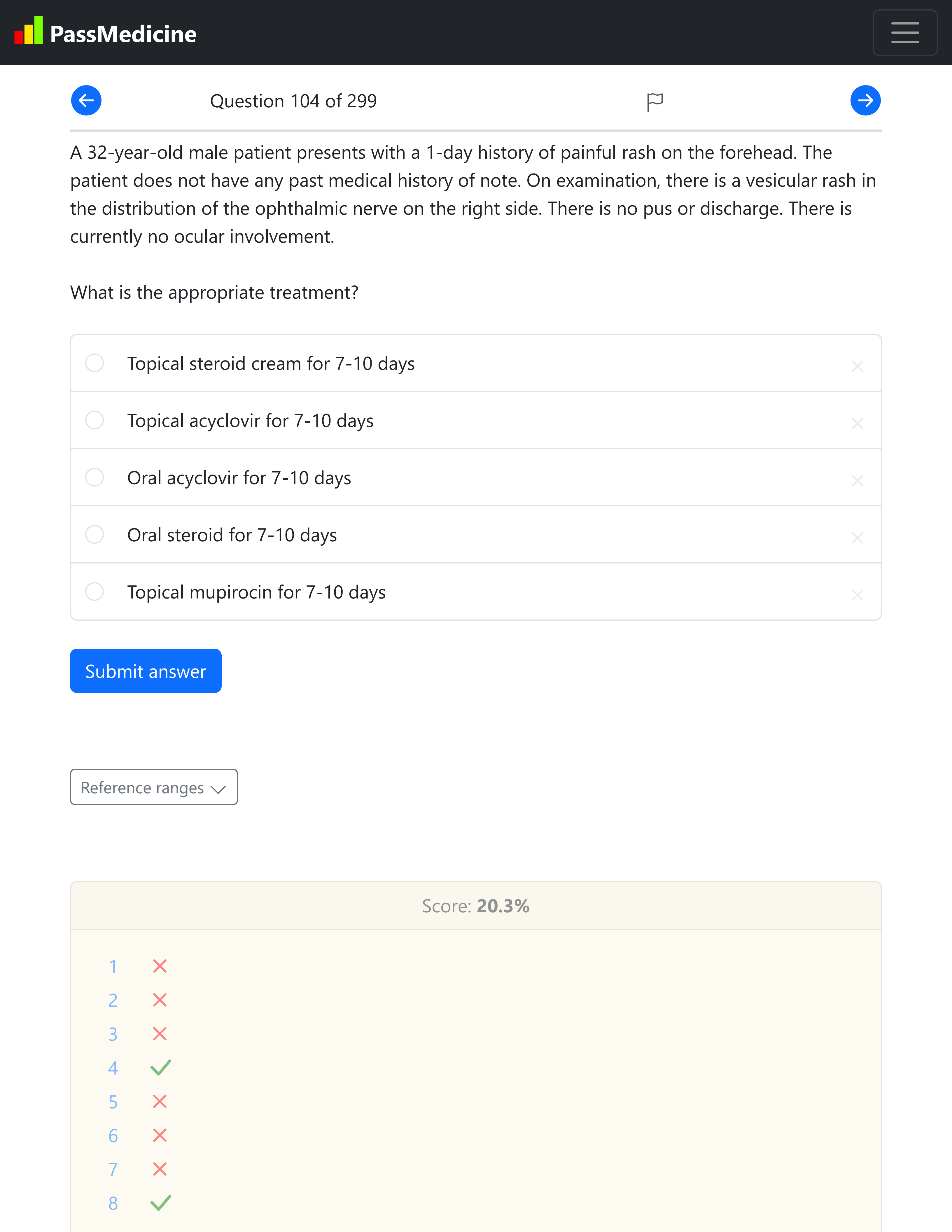

A 32-year-old male patient presents with a 1-day history of painful rash on the forehead. The patient does not have any past medical history of note. On examination, there is a vesicular rash in the distribution of the ophthalmic nerve on the right side. There is no pus or discharge. There is currently no ocular involvement.

What is the appropriate treatment?

- ○ Topical steroid cream for 7-10 days ×
- ○ Topical acyclovir for 7-10 days ×
- ○ Oral acyclovir for 7-10 days ×
- ○ Oral steroid for 7-10 days ×
- ○ Topical mupirocin for 7-10 days ×

Score: **20.3%**

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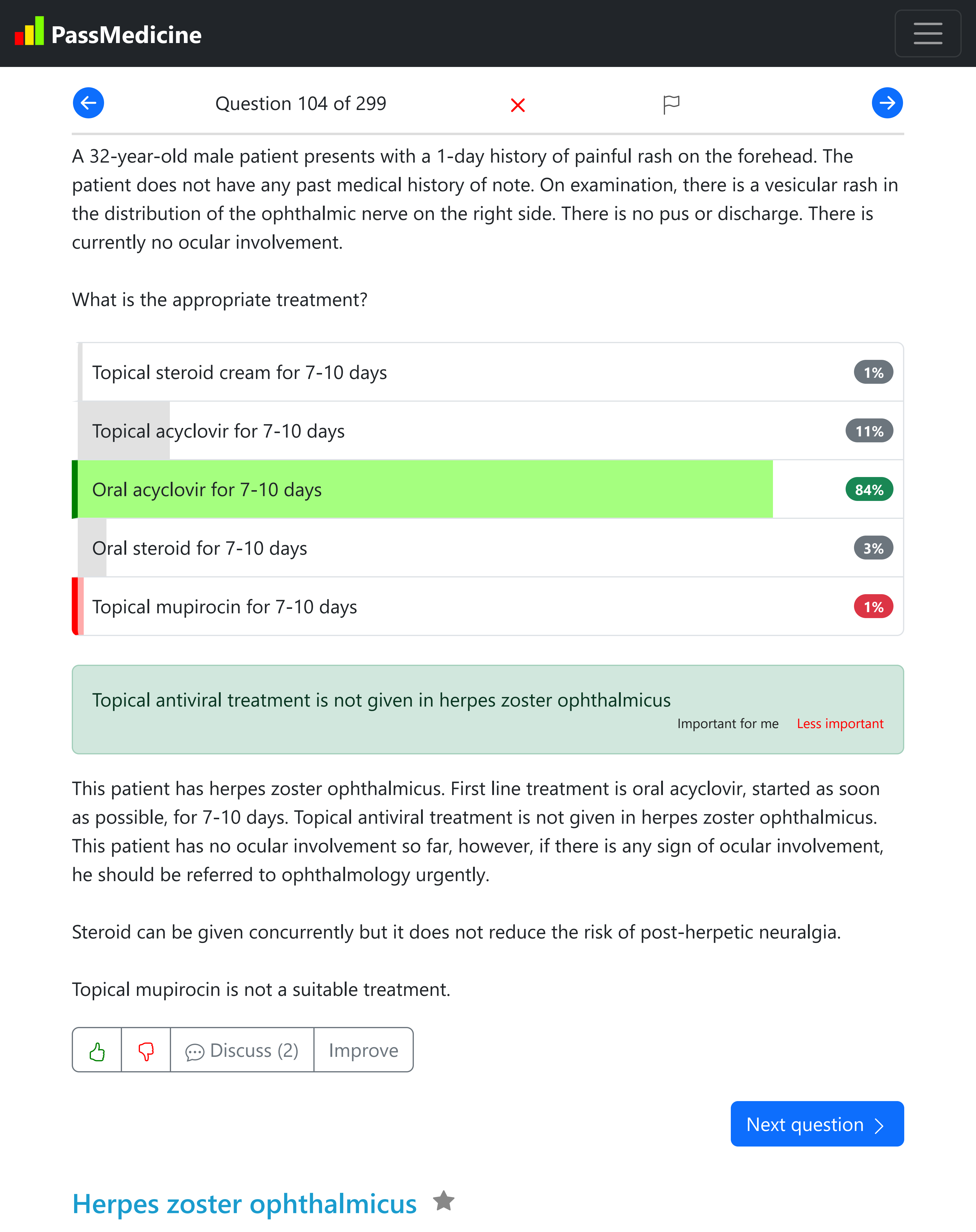

A 32-year-old male patient presents with a 1-day history of painful rash on the forehead. The patient does not have any past medical history of note. On examination, there is a vesicular rash in the distribution of the ophthalmic nerve on the right side. There is no pus or discharge. There is currently no ocular involvement.

What is the appropriate treatment?

Topical steroid cream for 7-10 days	1%
Topical acyclovir for 7-10 days	11%
Oral acyclovir for 7-10 days	84%
Oral steroid for 7-10 days	3%
Topical mupirocin for 7-10 days	1%

Topical antiviral treatment is not given in herpes zoster ophthalmicus

This patient has herpes zoster ophthalmicus. First line treatment is oral acyclovir, started as soon as possible, for 7-10 days. Topical antiviral treatment is not given in herpes zoster ophthalmicus. This patient has no ocular involvement so far, however, if there is any sign of ocular involvement, he should be referred to ophthalmology urgently.

Steroid can be given concurrently but it does not reduce the risk of post-herpetic neuralgia.

Topical mupirocin is not a suitable treatment.

Herpes zoster ophthalmicus

Herpes zoster ophthalmicus (HZO) describes the reactivation of the varicella-zoster virus in the area supplied by the ophthalmic division of the trigeminal nerve. It accounts for around 10% of case of shingles.

Features
- vesicular rash around the eye, which may or may not involve the actual eye itself
- Hutchinson's sign: rash on the tip or side of the nose. Indicates nasociliary involvement and is a strong risk factor for ocular involvement

Management
- oral antiviral treatment for 7-10 days
  - ideally started within 72 hours
  - intravenous antivirals may be given for very severe infection or if the patient is immunocompromised
  - topical antiviral treatment is not given in HZO
- topical corticosteroids may be used to treat any secondary inflammation of the eye
- ocular involvement requires urgent ophthalmology review

Complications
- ocular: conjunctivitis, keratitis, episcleritis, anterior uveitis
- ptosis
- post-herpetic neuralgia

123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

PRIMAI
VIRUS LEAVES LUNGS
ORAL or SKIN LESIONS
LYMPH NODE

Varicella zoster virus

Osmosis - YouTube 👍 0 👎 0

Report broken media

Score: **20.9%**

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Back to top

A 28-year-old man is admitted to the respiratory ward with sudden onset chest pain and shortness of breath. He has no past medical history of note. On examination, there is reduced air entry in the left lung that is hyper-resonant to percussion. The likely diagnosis is confirmed on a chest x-ray and he is treated with a pleural aspiration and observed on the ward for 24 hours.

During this period of observation, he remains well with normal observations and resolution of his chest pain and shortness of breath. A repeat chest x-ray is taken that appears normal and he is medically fit for discharge.

The patient mentions that he is due to fly to Brazil for a holiday in 2 days and wants to know if this will be safe.

What is the most appropriate advice to give this patient?

- ○ Able to fly following pleurodesis ×
- ○ Able to fly immediately ×
- ○ Able to fly in 1 week ×
- ○ Able to fly in 6 weeks ×
- ○ Unable to fly for life ×

Submit answer

Reference ranges

Score: **20.3%**

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Back to top

A 28-year-old man is admitted to the respiratory ward with sudden onset chest pain and shortness of breath. He has no past medical history of note. On examination, there is reduced air entry in the left lung that is hyper-resonant to percussion. The likely diagnosis is confirmed on a chest x-ray and he is treated with a pleural aspiration and observed on the ward for 24 hours.

During this period of observation, he remains well with normal observations and resolution of his chest pain and shortness of breath. A repeat chest x-ray is taken that appears normal and he is medically fit for discharge.

The patient mentions that he is due to fly to Brazil for a holiday in 2 days and wants to know if this will be safe.

What is the most appropriate advice to give this patient?

- Able to fly following pleurodesis 6%
- Able to fly immediately 10%
- Able to fly in 1 week 63%
- Able to fly in 6 weeks 20%
- Unable to fly for life 1%

Pneumothorax -> can fly 1 week after treatment if chest x-ray shows resolution

Important for me Less important

This patient's clinical history, examination and investigation findings are in keeping with a pneumothorax. Given that this patient is young with no other past medical history, it is likely that he has had a spontaneous, primary pneumothorax which, if large enough, should be treated with needle aspiration. Current guidance from the British thoracic society states that following complete resolution of the pneumothorax, patients are **able to fly 1 week after treatment**. If a pneumothorax arises following a traumatic injury, then flying 2 weeks after chest x-ray confirmation of resolution is advised.

A **pleurodesis** is usually required for recurrent pneumothoraces and can be either chemical or surgical. Given that this patient's chest x-ray shows resolution of the pneumothorax, pleurodesis is not necessary and would not be an indication for this patient to travel by air.

It is too soon for the patient **to fly immediately** as guidelines state that patients not air travel until 1 week from the complete resolution of pneumothorax on chest x-ray.

Previous guidelines advised that air travel would be safe following **6 weeks** from the resolution of pneumothorax. However, this has since been changed to 1 week.

Unable to fly for life is incorrect. Following a pneumothorax, guidance is given to avoid deep-sea diving for life. However, air travel is still possible following pneumothorax resolution.

👍 👎 💬 Discuss (1) Improve

Next question ›

Fitness to fly ★

The Civil Aviation Authority (CAA) has issued guidelines on air travel for people with medical conditions; please see the link provided.

Cardiovascular disease

- unstable angina, uncontrolled hypertension, uncontrolled cardiac arrhythmia, decompensated heart failure, severe symptomatic valvular disease: should not fly
- uncomplicated myocardial infarction: may fly after 7-10 days
- complicated myocardial infarction: after 4-6 weeks
- coronary artery bypass graft: after 10-14 days
- percutaneous coronary intervention: after 3 days
- stroke: patients are advised to wait 10 days following an event, although if stable may be carried within 3 days of the event

Respiratory disease

- pneumonia: should be 'clinically improved with no residual infection'
- pneumothorax: absolute contraindication, the CAA suggest patients may travel 2 weeks after successful drainage if there is no residual air. The British Thoracic Society used to recommend not travelling by air for a period of 6 weeks but this has now been changed to 1 week post check x-ray

Pregnancy

- most airlines do not allow travel after 36 weeks for a single pregnancy and after 32 weeks for a multiple pregnancy

- most airlines require a certificate after 28 weeks confirming that the pregnancy is progressing normally

Surgery

- travel should be avoided for 10 days following abdominal surgery
- laparoscopic surgery: after 24 hours
- colonoscopy: after 24 hours
- following the application of a plaster cast, the majority of airlines restrict flying for 24 hours on flights of less than 2 hours or 48 hours for longer flights

Haematological disorders

- patients with a haemoglobin of greater than 8 g/dl may travel without problems (assuming there is no coexisting condition such as cardiovascular or respiratory disease)

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Civil Aviation Authority 👍 18 👎 7

Fitness to fly guidelines

Suggest link

Report broken link

Score: **20.9%**

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Question 106 of 299

A 68-year-old man presents to the acute medical unit with an episode of loss of consciousness. He states that he suffered a headache earlier that day. He describes it as a sudden onset headache at the back of his head which reached 10/10 severity within a few seconds.

You organise the following investigations:

CT head	Blood in the sulci, fissures, basal cisterns and ventricles
Cerebral CT angiogram	Evidence of ruptured posterior cerebral artery aneurysm

What is the most appropriate initial management?

- ○ Triple H therapy (hypertension, hypervolaemia, and haemodilution) ×
- ○ Labetalol ×
- ○ Magnesium sulphate ×
- ○ Amlodipine ×
- ○ Nimodipine ×

Submit answer

Reference ranges

Score: **20.3%**

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Cerebral CT angiogram	Evidence of ruptured posterior cerebral artery aneurysm

What is the most appropriate initial management?

Triple H therapy (hypertension, hypervolaemia, and haemodilution)	15%
Labetalol	11%
Magnesium sulphate	1%
Amlodipine	1%
Nimodipine	72%

Nimodipine is used to prevent vasospasm in aneurysmal subarachnoid haemorrhages

Important for me Less important

The patient has suffered from a subarachnoid haemorrhage (SAH) as confirmed on CT brain. A CT angiography has confirmed that it is aneurysmal. He is now at significant risk of re-bleeding and vasospasm.

To prevent re-bleeding he needs urgent endovascular clipping or coiling.

The gold standard for preventing vasospasm is nimodipine. Alternative calcium channel blockers can be used but the strongest evidence favours nimodipine.

The combination of induced hypertension, hypervolaemia, and haemodilution (triple H therapy) is often used to prevent and treat cerebral vasospasm after aneurysmal SAH, however the evidence for this approach is inconclusive.

👍 👎 Discuss (7) Improve

Next question >

Subarachnoid haemorrhage ★

A subarachnoid haemorrhage (SAH) is an intracranial haemorrhage that is defined as the presence of blood within the subarachnoid space, i.e. deep to the subarachnoid layer of the meninges.

The most common cause of SAH is head injury and this is called *traumatic SAH* . In the absence of trauma, SAH is termed *spontaneous SAH* . The rest of this note focuses on spontaneous SAH.

Causes of spontaneous SAH include:

- intracranial aneurysm (saccular 'berry' aneurysms)
    - accounts for around 85% of cases
    - conditions associated with berry aneurysms include hypertension,adult polycystic kidney disease, Ehlers-Danlos syndrome and coarctation of the aorta
- arteriovenous malformation
- pituitary apoplexy
- mycotic (infective) aneurysms

Classical presenting features include:

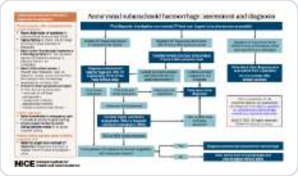
- headache
    - usually of sudden-onset ('thunderclap' or 'hit with a baseball bat')
    - severe ('worst of my life')
    - occipital
    - typically peaking in intensity within 1 to 5 minutes
    - there may be a history of a less-severe 'sentinel' headache in the weeks prior to presentation
- nausea and vomiting
- meningism (photophobia, neck stiffness)
- coma
- seizures
- ECG changes including ST elevation may be seen
    - this may be secondary to either autonomic neural stimulation from the hypothalamus or elevated levels of circulating catecholamines

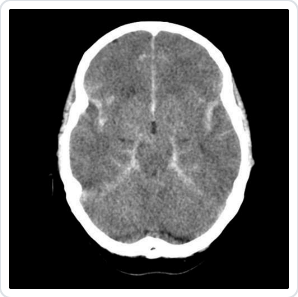
Investigation

- non-contrast CT head is the first-line investigation of choice
    - acute blood (hyperdense/bright on CT) is typically distributed in the basal cisterns, sulci and in severe cases the ventricular system.
- if CT head is done within 6 hours of symptom onset and is normal
    - new guidelines suggest **not** doing a lumbar puncture
    - consider an alternative diagnosis
- if CT head is done more than 6 hours after symptom onset and is normal
    - do a lumber puncture (LP)
    - timing wise the LP should be performed at least 12 hours following the onset of symptoms to allow the development of xanthochromia (the result of red blood cell breakdown).
    - xanthochromia helps to distinguish true SAH from a 'traumatic tap' (blood introduced by the LP procedure).
    - as well as xanthochromia, CSF findings consistent with subarachnoid haemorrhage include a normal or raised opening pressure
- if the CT shows evidence of a SAH
    - referral to neurosurgery to be made as soon as SAH is confirmed

After spontaneous SAH is confirmed, the aim of investigation is to identify a causative pathology that needs urgent treatment:

- CT intracranial angiogram (to identify a vascular lesion e.g. aneurysm or AVM)
- +/- digital subtraction angiogram (catheter angiogram)


Aneurysmal subarachnoid haemorrhage: assessment and diagnosis
NICE



Management of a confirmed aneurysmal subarachnoid haemorrhage

- supportive
    - bed rest
    - analgesia
    - venous thromboembolism prophylaxis
    - discontinuation of antithrombotics (reversal of anticoagulation if present)
- vasospasm is prevented using a course of oral nimodipine
- intracranial aneurysms are at risk of rebleeding and therefore require prompt intervention, preferably within 24 hours

    - most intracranial aneurysms are now treated with a coil by interventional neuroradiologists, but a minority require a craniotomy and clipping by a neurosurgeon

Complications of aneurysmal SAH:

- re-bleeding
    - happens in around 10% of cases and most common in the first 12 hours
    - if rebleeding is suspected (e.g. sudden worsening of neurological symptoms) then a repeat CT should be arranged
    - associated with a high mortality (up to 70%)
- hydrocephalus
    - hydrocephalus is temporarily treated with an external ventricular drain (CSF diverted into a bag at the bedside) or, if required, a long-term ventriculoperitoneal shunt
- vasospasm (also termed delayed cerebral ischaemia), typically 7-14 days after onset
    - ensure euvolaemia (normal blood volume)
    - consider treatment with a vasopressor if symptoms persist
- hyponatraemia (most typically due to syndrome inappropriate anti-diuretic hormone (SIADH))
- seizures

Important predictive factors in SAH:

- conscious level on admission
- age
- amount of blood visible on CT head

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia 👍 15 👎 15

Subarachnoid haemorrhage

Life in the Fast Lane 👍 19 👎 10

Example ECG changes in subarachnoid haemorrhage

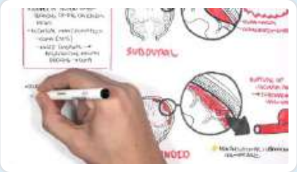
NICE 👍 9 👎 4

2022 Subarachnoid haemorrhage caused by a ruptured aneurysm: diagnosis and management

Suggest link

Report broken link

Media



Intracranial Haemorrhage Types

Armando Hasudungan - YouTube 👍 2 👎 0

Arteriovenous malformation

Osmosis - YouTube 👍 0 👎 0

NEUROLOGY
NEUROSURGERY
SUBARACHNOID
HAEMORRHAGE

Subarachnoid Haemorrhage / pathophysiology, complications and management

Armando Hasudungan - YouTube 👍 0 👎 0

Report broken media

Score: **20.9%**

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The emergency department registrar asks for your advice regarding a 67 year-old female patient who presented with an acutely painful right eye. On examination the right pupil is fixed in mid-dilation and not responsive to light. The conjunctiva is injected. The patient is clearly in considerable pain and has vomited twice in the department. The emergency department physicians have given intravenous analgesia and anti-emetics. You refer urgently to ophthalmology.

What is appropriate initial treatment?

- ○ Topical pilocarpine
- ○ Urgent iridotomy
- ○ IV acetazolamide
- ○ Topical latanoprost
- ○ Topical brimonidine

Submit answer

Reference ranges

Score: **20.3%**

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What is appropriate initial treatment?

Topical pilocarpine	26%
Urgent iridotomy	10%
IV acetazolamide	56%
Topical latanoprost	5%
Topical brimonidine	3%

The diagnosis here is acute angle closure glaucoma, an ophthalmological emergency. The correct urgent management is as follows:

- IV analgesia and antiemetics, lie patient supine.
- Topical beta blockers and steroids.
- IV acetazolamide.
- After initial treatment, pilocarpine will induce meiosis and open the angle. This is initially ineffective due to pressure induced ischaemic paralysis of the iris.
- Iridotomy 24-48 hours after intra-ocular pressure is controlled to prevent recurrence.

Discuss (9) Improve

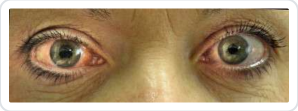
Next question

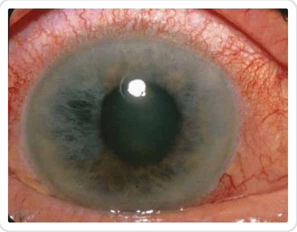
Acute angle-closure glaucoma

Glaucoma is a group of disorders characterised by optic neuropathy due, in the majority of patients, to raised intraocular pressure (IOP). It is now recognised that a minority of patients with raised IOP do not have glaucoma and vice versa.

In acute angle-closure glaucoma (AACG) there is a rise in IOP secondary to an impairment of aqueous outflow. Factors predisposing to AACG include:
- hypermetropia (long-sightedness)
- pupillary dilatation
- lens growth associated with age

Features
- severe pain: may be ocular or headache
- decreased visual acuity
- symptoms worse with mydriasis (e.g. watching TV in a dark room)
- hard, red-eye
- haloes around lights
- semi-dilated non-reacting pupil
- corneal oedema results in dull or hazy cornea
- systemic upset may be seen, such as nausea and vomiting and even abdominal pain





Investigations
- tonometry to assess for elevated IOP
- gonioscopy (literally looking, *oscopy*, at the angle, *gonio*): a special lens for the slit lamp that allows visualisation of the angle

Management

The management of AACG is an emergency and should prompt urgent referral to an ophthalmologist. Emergency medical treatment is required to lower the IOP with more definitive surgical treatment given once the acute attack has settled.

There are no guidelines for the initial medical treatment emergency treatment. An example regime would be:
- combination of eye drops, for example:

    - a direct parasympathomimetic (e.g. pilocarpine, causes contraction of the ciliary muscle → opening the trabecular meshwork → increased outflow of the aqueous humour)
    - a beta-blocker (e.g. timolol, decreases aqueous humour production)
    - an alpha-2 agonist (e.g. apraclonidine, dual mechanism, decreasing aqueous humour production and increasing uveoscleral outflow)
- intravenous acetazolamide
    - reduces aqueous secretions
- some guidelines also recommend the use of topical steroids to reduce inflammation

Definitive management
- laser peripheral iridotomy
    - creates a tiny hole in the peripheral iris → aqueous humour flowing to the angle

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

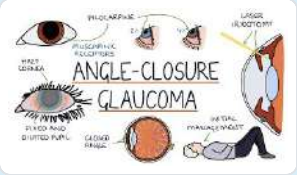
Links

Patient.info 👍 26 👎 9

Acute angle closure glaucoma review

Suggest link Report broken link

Media


ANGLE-CLOSURE
GLAUCOMA

Acute angle closure glaucoma

Zero To Finals - YouTube 👍 4 👎 0

GLAUCOMA
Osmosis.org

Glaucoma (open-angle, closed-angle, and normal-tension)

Osmosis - YouTube 👍 6 👎 3

Report broken media

Score: **20.9%**

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Question 108 of 299

A 20-year-old female with multiple sclerosis (MS) attends the emergency department following a seizure. She was diagnosed with MS at age 16 and has received different immunomodulatory regimes in the past, including azathioprine. She commenced natalizumab 2 years ago. This is her first seizure however once recovered, she tells you that she has noticed she has been increasingly clumsy over the last 6 months, she drops things easily. In addition, she has had several episodes where her speech has been slurred and people have commented that she sounds tired.
On examination, you find that her tone is normal throughout, power in all muscles groups in both upper and lower limbs is normal. On cranial nerve examination, you identify a right homonymous superior quadrantanopia however she does have a full range of eye movements and no ptosis. She has an ataxic gait. Her speech is normal throughout the history.

She is concerned, what is the diagnosis?

- ○ Progressive multifocal leukoencephalopathy ×
- ○ Transient ischaemic attacks ×
- ○ Progression of multiple sclerosis ×
- ○ Newly diagnosed epilepsy ×
- ○ myasthenia gravis ×

Submit answer

Reference ranges

Score: **20.3%**

1 ✗
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On examination, you find that her tone is normal throughout, power in all muscles groups in both upper and lower limbs is normal. On cranial nerve examination, you identify a right homonymous superior quadrantanopia however she does have a full range of eye movements and no ptosis. She has an ataxic gait. Her speech is normal throughout the history.

She is concerned, what is the diagnosis?

Progressive multifocal leukoencephalopathy	79%
Transient ischaemic attacks	2%
Progression of multiple sclerosis	17%
Newly diagnosed epilepsy	1%
myasthenia gravis	1%

Natalizumab is a humanised monoclonal antibody against the cell adhesion molecule 4-integrin and is licensed as a monotherapy for the treatment of multiple sclerosis in Europe.

Progressive multifocal leukoencephalopathy (PML) is an opportunistic infection caused by the JC virus that only occurs in patients who are immunocompromised and has a risk of 2.1 in every 1,000 using natalizumab. Patients who have taken immunosuppressants before the initiation of natalizumab therapy have the highest risk of developing PML

Bloomgren, G et al. Risk of Natalizumab-Associated Progressive Multifocal Leukoencephalopathy. 2012 New England Journal of Medicine 366 (20): 18701880.

Kappos, L; Bates, D; Edan, et al. Natalizumab treatment for multiple sclerosis: updated recommendations for patient selection and monitoring. 2010 Lancet neurology 10 (8): 74558.

Discuss (9) Improve

Next question

Multiple sclerosis: management

Treatment in multiple sclerosis is focused on reducing the frequency and duration of relapses. There is no cure.

Acute relapse

High-dose steroids (e.g. oral or IV methylprednisolone) may be given for 5 days to shorten the length of an acute relapse. It should be noted that steroids shorten the duration of a relapse and do not alter the degree of recovery (i.e. whether a patient returns to baseline function)

Disease modifying drugs

A number of drugs have been shown to reduce the risk of relapse in patients with MS. Typical indications for disease-modifying drugs include:

- relapsing-remitting disease + 2 relapses in past 2 years + able to walk 100m unaided
- secondary progressive disease + 2 relapses in past 2 years + able to walk 10m (aided or unaided)

Drug options for reducing the risk of relapse in MS

- natalizumab
    - a recombinant monoclonal antibody that antagonises alpha-4 beta-1-integrin found on the surface of leucocytes
    - inhibit migration of leucocytes across the endothelium across the blood-brain barrier
    - generally considered to have the strongest evidence base for preventing relapse of the disease-modifying and hence is often used first-line
    - given intravenously
- ocrelizumab
    - humanized anti-CD20 monoclonal antibody
    - like natalizumab, it is considered a high-efficacy drug that is often used first-line
    - given intravenously
- fingolimod
    - sphingosine 1-phosphate (S1P) receptor modulator
    - prevents lymphocytes from leaving lymph nodes
    - oral formulations are available

- beta-interferon
  - not considered to be as effective as alternative disease-modifying drugs
  - given subcutaneously/intramuscularly
- glatiramer acetate
  - immunomodulating drug - acts as an 'immune decoy'
  - given subcutaneously
  - along with beta-interferon considered an 'older drug' with less effectiveness compared to monoclonal antibodies and S1P) receptor modulators

Some specific problems

Fatigue

- once other problems (e.g. anaemia, thyroid or depression) have been excluded NICE recommend a trial of amantadine
- other options include mindfulness training and CBT

Spasticity

- baclofen and gabapentin are first-line. Other options include diazepam, dantrolene and tizanidine
- physiotherapy is important
- cannabis and botox are undergoing evaluation

Bladder dysfunction

- may take the form of urgency, incontinence, overflow etc
- guidelines stress the importance of getting an ultrasound first to assess bladder emptying - anticholinergics may worsen symptoms in some patients
- if significant residual volume → intermittent self-catheterisation
- if no significant residual volume → anticholinergics may improve urinary frequency

Oscillopsia (visual fields appear to oscillate)

- gabapentin is first-line

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Multiple Sclerosis Society 👍 6 👎 14

Primary care guidelines

NICE 👍 15 👎 7

2014 Multiple Sclerosis guidelines

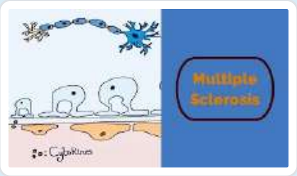
Suggest link

Report broken link

Media

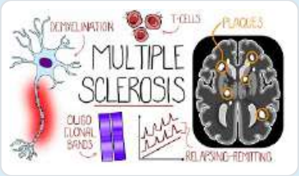
Multiple sclerosis

Podmedics - YouTube 👍 4 👎 0



Multiple sclerosis

Soton Brain Hub - YouTube 👍 3 👎 0


DEMYELINATION
T-CELLS
PLAQUES
MULTIPLE SCLEROSIS
OLIGO CLONAL BANDS
RELAPSING-REMITTING

Multiple sclerosis

Rhesus Medicine - YouTube 👍 1 👎 0

MULTIPLE SCLEROSIS

Multiple sclerosis - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 7 👎 1

Report broken media

Score: **20.9%**

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Question 109 of 299

A 22-year-old, recently married woman presents to the first seizure clinic and is diagnosed with idiopathic generalised epilepsy. She tells you that she is keen to start a family soon. Which first line anti epileptic drug should be avoided?

- ○ Levetiracetam ×
- ○ Topiramate ×
- ○ Lamotrigine ×
- ○ Carbamazepine ×
- ○ Valproate ×

Submit answer

Reference ranges

Score: **20.3%**

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Question 109 of 299 ✓

A 22-year-old, recently married woman presents to the first seizure clinic and is diagnosed with idiopathic generalised epilepsy. She tells you that she is keen to start a family soon. Which first line anti epileptic drug should be avoided?

Levetiracetam	7%
Topiramate	6%
Lamotrigine	9%
Carbamazepine	5%
Valproate	73%

All antiepileptic drugs are associated with a risk of major congenital malformations in the offspring. However, the potential for the mother to develop tonic clonic seizures, which can result in significant adverse health outcomes for the fetus, means that discontinuing antiepileptic drug therapy during pregnancy is still discouraged by most practitioners.The largest randomised trials of newer generation versus older generation antiepileptic drugs (SANAD trials) found that valproate is superior to both lamotrigine and topiramate for the treatment of idiopathic generalised epilepsy. However, valproate is associated with teratogenicity and a risk of low IQ and should therefore be avoided in women of childbearing potential. The risk of teratogenecitiy for the newer generation antiepileptics drugs remains largely unknown, due to small sample sizes and polytherapy exposures. The data for lamotrigine is limited but is believed to be less teratogenic than valproate.

Carbamazepine is associated with a higher risk of developing major congenital malformations. For Levetiracetam, although pregnancy exposure data is limited, the UK epilepsy and pregnancy registry have not identified any major congenital malformations in association with its use.

Discuss (3) Improve

Next question ›

Epilepsy: pregnancy and breast feeding

The risks of uncontrolled epilepsy during pregnancy generally outweigh the risks of medication to the fetus. All women thinking about becoming pregnant should be advised to take folic acid 5mg per day well before pregnancy to minimise the risk of neural tube defects. Around 1-2% of newborns born to non-epileptic mothers have congenital defects. This rises to 3-4% if the mother takes antiepileptic medication.

Other points
- aim for monotherapy
- there is no indication to monitor antiepileptic drug levels
- sodium valproate: associated with neural tube defects
- carbamazepine: often considered the least teratogenic of the older antiepileptics
- phenytoin: associated with cleft palate
- lamotrigine: studies to date suggest the rate of congenital malformations may be low. The dose of lamotrigine may need to be increased in pregnancy

Breast feeding is generally considered safe for mothers taking antiepileptics with the possible exception of the barbiturates

It is advised that pregnant women taking phenytoin are given vitamin K in the last month of pregnancy to prevent clotting disorders in the newborn

Sodium valproate

The November 2013 issue of the Drug Safety Update also carried a warning about new evidence showing a significant risk of neurodevelopmental delay in children following maternal use of sodium valproate.

The update concludes that sodium valproate should not be used during pregnancy and in women of childbearing age unless clearly necessary. Women of childbearing age should not start treatment without specialist neurological or psychiatric advice.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

SIGN 👍 8 👎 7

2018 Diagnosis and management of epilepsy in adults

NICE 👍 9 👎 4

2022 Epilepsy guidelines

Royal College of Obstetricians and Gynaecologists 👍 8 👎 2

2016 Epilepsy in Pregnancy

Suggest link

Report broken link

Score: **20.9%**

1 ✗
2 ✗
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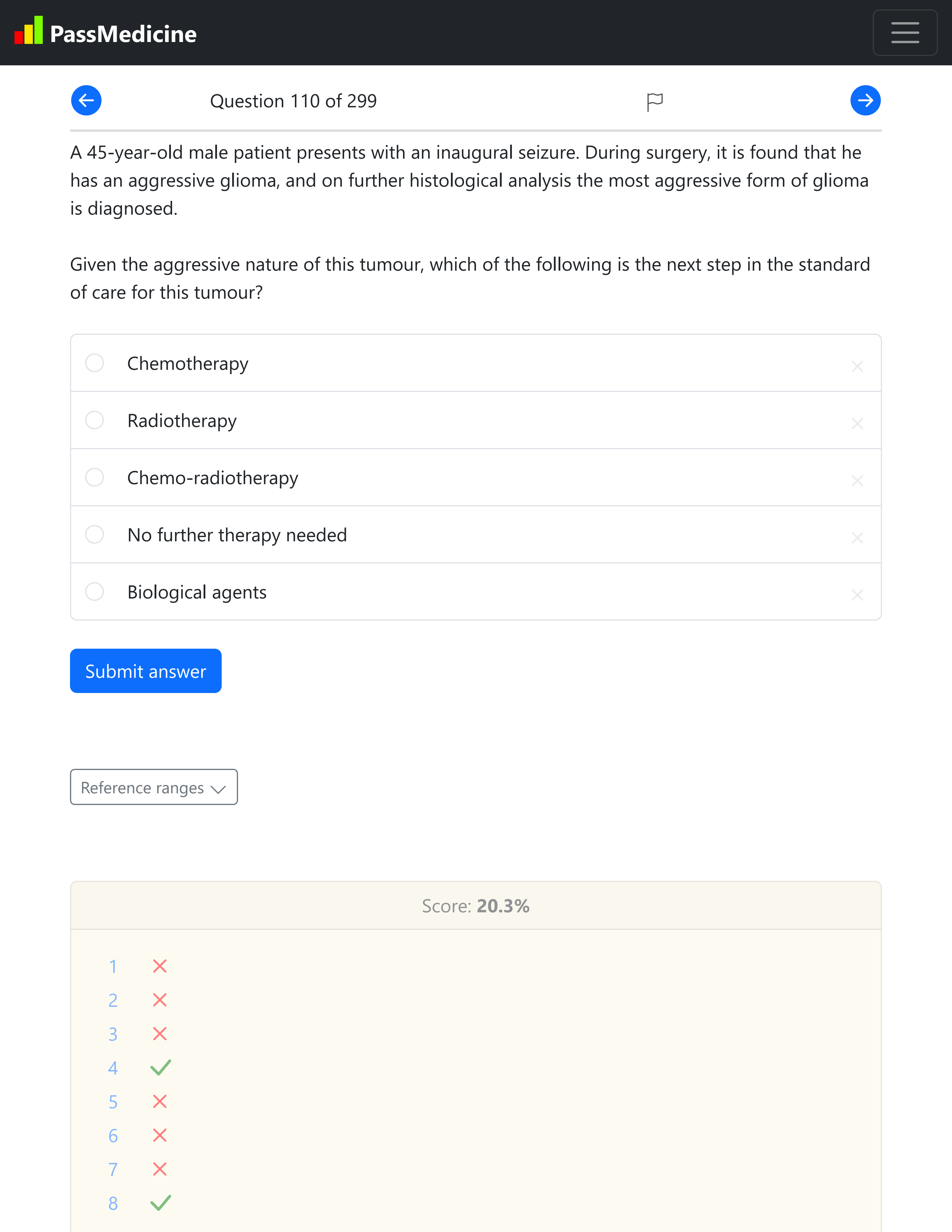

PassMedicine

Question 110 of 299

A 45-year-old male patient presents with an inaugural seizure. During surgery, it is found that he has an aggressive glioma, and on further histological analysis the most aggressive form of glioma is diagnosed.

Given the aggressive nature of this tumour, which of the following is the next step in the standard of care for this tumour?

- ○ Chemotherapy
- ○ Radiotherapy
- ○ Chemo-radiotherapy
- ○ No further therapy needed
- ○ Biological agents

Score: **20.3%**

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A 45-year-old male patient presents with an inaugural seizure. During surgery, it is found that he has an aggressive glioma, and on further histological analysis the most aggressive form of glioma is diagnosed.

Given the aggressive nature of this tumour, which of the following is the next step in the standard of care for this tumour?

Chemotherapy	6%
Radiotherapy	22%
Chemo-radiotherapy ✓	63%
No further therapy needed	7%
Biological agents ✗	2%

Glioblastoma multiforme is by far the most common and most malignant/aggressive of the glial tumours. It kills virtually every patient that is affected by it, and until a trial in 2007 was published, the standard of care was surgery and radiotherapy.

Currently, the standard of care is surgery followed by chemo-radiotherapy with temozolomide, an alkylating chemotherapy agent. The key is that the temozolomide and the radiotherapy have a synergistic effect on killing tumour cells.

Discuss (5) Improve

Next question ›

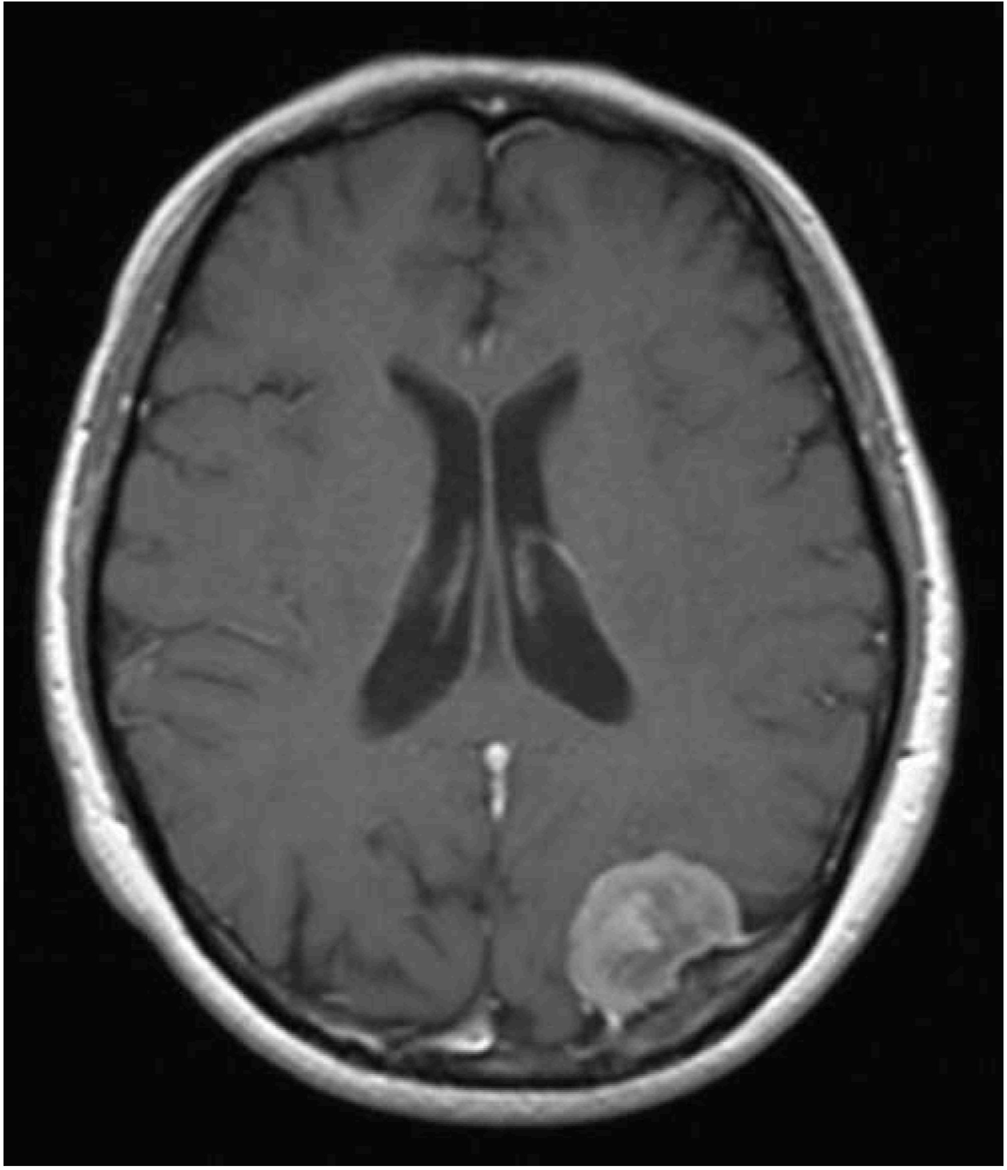
Brain tumours ★

The majority of adult tumours are supratentorial, where as the majority of childhood tumours are infratentorial.

Type of tumour	Features
Metastases	Metastatic brain cancer is the most common form of brain tumours. They are often multiple and not treatable with surgical intervention. Tumours that most commonly spread to the brain include: • lung (most common) • breast • bowel • skin (namely melanoma) • kidney
Glioblastoma multiforme	• Glioblastoma is the most common primary tumour in adults and is associated with a poor prognosis (~ 1yr). • On imaging they are solid tumours with central necrosis and a rim that enhances with contrast. Disruption of the blood-brain barrier and therefore are associated with vasogenic oedema. • Histology: Pleomorphic tumour cells border necrotic areas • Treatment is surgical with postoperative chemotherapy and/or radiotherapy. Dexamethasone is used to treat the oedema.
Meningioma	• The second most common primary brain tumour in adults • Meningiomas are typically benign, extrinsic tumours of the central nervous system. They arise from the arachnoid cap cells of the meninges and are typically located next to the dura and cause symptoms by compression rather than invasion. • They typically are located at the falx cerebri, superior sagittal sinus, convexity or skull base. • Histology: Spindle cells in concentric whorls and calcified psammoma bodies • Investigation is with CT (will show contrast enhancement) and MRI, and treatment will involve either observation, radiotherapy or surgical resection.
Vestibular schwannoma	• A vestibular schwannoma (previously termed acoustic neuroma) is a benign tumour arising from the eighth cranial nerve (vestibulocochlear nerve). Often seen in the cerebellopontine angle. It presents with

Type of tumour	Features
	hearing loss, facial nerve palsy (due to compression of the nearby facial nerve) and tinnitus. • Neurofibromatosis type 2 is associated with bilateral vestibular schwannomas. • Histology: Antoni A or B patterns are seen. Verocay bodies (acellular areas surrounded by nuclear palisades) • Treatment may involve observation, radiotherapy or surgery.
Pilocytic astrocytoma	• The most common primary brain tumour in children • Histology: Rosenthal fibres (corkscrew eosinophilic bundle)
Medulloblastoma	• A medulloblastoma is an aggressive paediatric brain tumour that arises within the infratentorial compartment. It spreads through the CSF system. Treatment is surgical resection and chemotherapy. • Histology: Small, blue cells. Rosette pattern of cells with many mitotic figures
Ependymoma	• Commonly seen in the 4th ventricle • May cause hydrocephalus • Histology: perivascular pseudorosettes
Oligodendroma	• Benign, slow-growing tumour common in the frontal lobes • Histology: Calcifications with 'fried-egg' appearance
Haemangioblastoma	• Vascular tumour of the cerebellum • Associated with von Hippel-Lindau syndrome • Histology: foam cells and high vascularity

Type of tumour	Features
Pituitary adenoma	• Pituitary adenomas are benign tumours of the pituitary gland. They are either secretory (producing a hormone in excess) or non-secretory. They may be divided into microadenomas (smaller than 1cm) or macroadenoma (larger than 1cm). • Patients will present with the consequences of hormone excess (e.g. Cushing's due to ACTH, or acromegaly due to GH) or depletion. Compression of the optic chiasm will cause a bitemporal hemianopia due to the crossing nasal fibers. • Investigation requires a pituitary blood profile and MRI. Treatment can either be hormonal or surgical (e.g. transphenoidal resection).
Craniopharyngioma	• Most common paediatric supratentorial tumour • A craniopharyngioma is a solid/cystic tumour of the sellar region that is derived from the remnants of Rathke's pouch. It is common in children, but can present in adults also. It may present with hormonal disturbance, symptoms of hydrocephalus or bitemporal hemianopia. • Histology: Derived from remnants of Rathke pouch • Investigation requires pituitary blood profile and MRI. Treatment is typically surgical with or without postoperative radiotherapy.



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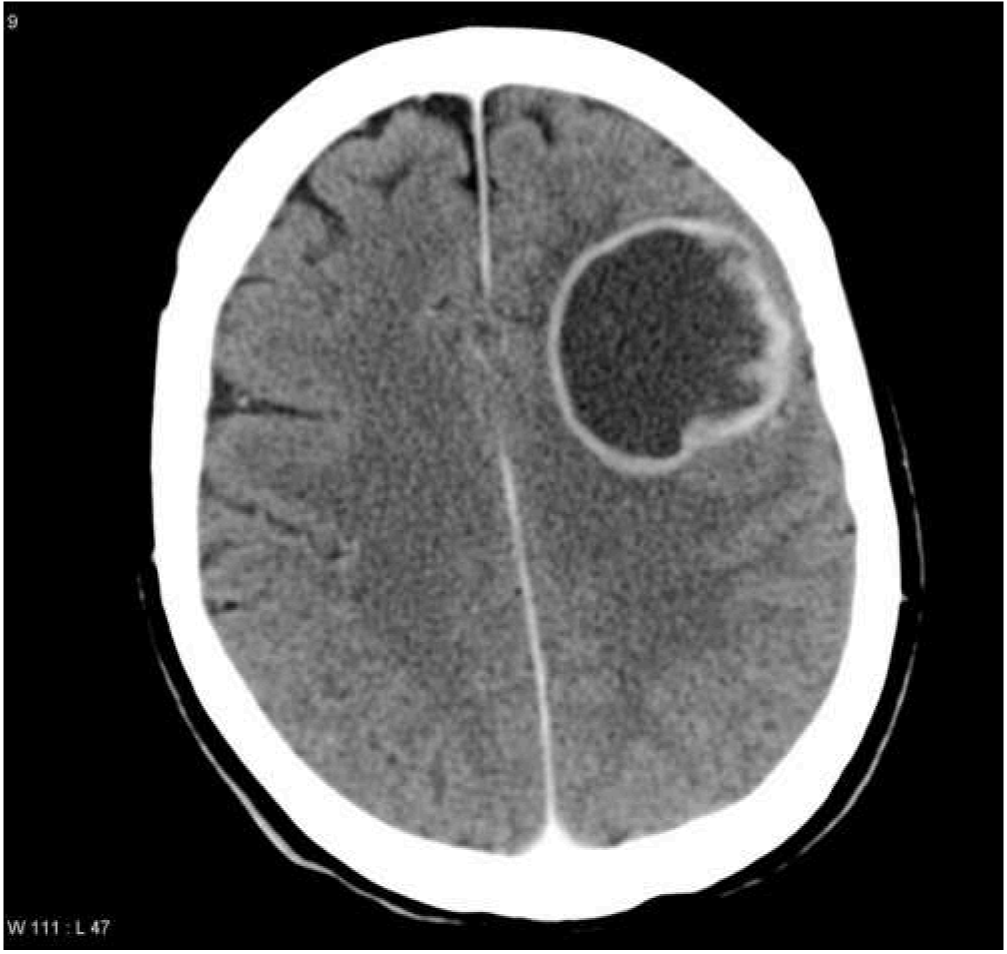
Meningioma - MRI showing the typical well-circumscribed appearance. A dural tail can be where the tumour 'connects' to the dura. It is seen in around 65% of meningiomas.



© Image used on license from Radiopaedia

Glioblastoma multiforme - CT showing a peripherally enhancing lesion within the left frontal lobe. Note the contrast to the more homogenous meningioma above.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

ADULT
BRAIN TUMORS
Osmosis.org

[Adult brain tumours]

Osmosis - YouTube 👍 4 👎 0

GLIOBLASTOMA
OSMOSIS
KNOWLEDGE SHOT

[Glioblastoma]

Osmosis - YouTube 👍 2 👎 1

PEDIATRIC
BRAIN TUMORS
Osmosis.org

[Paediatric brain tumours]

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **20.9%**

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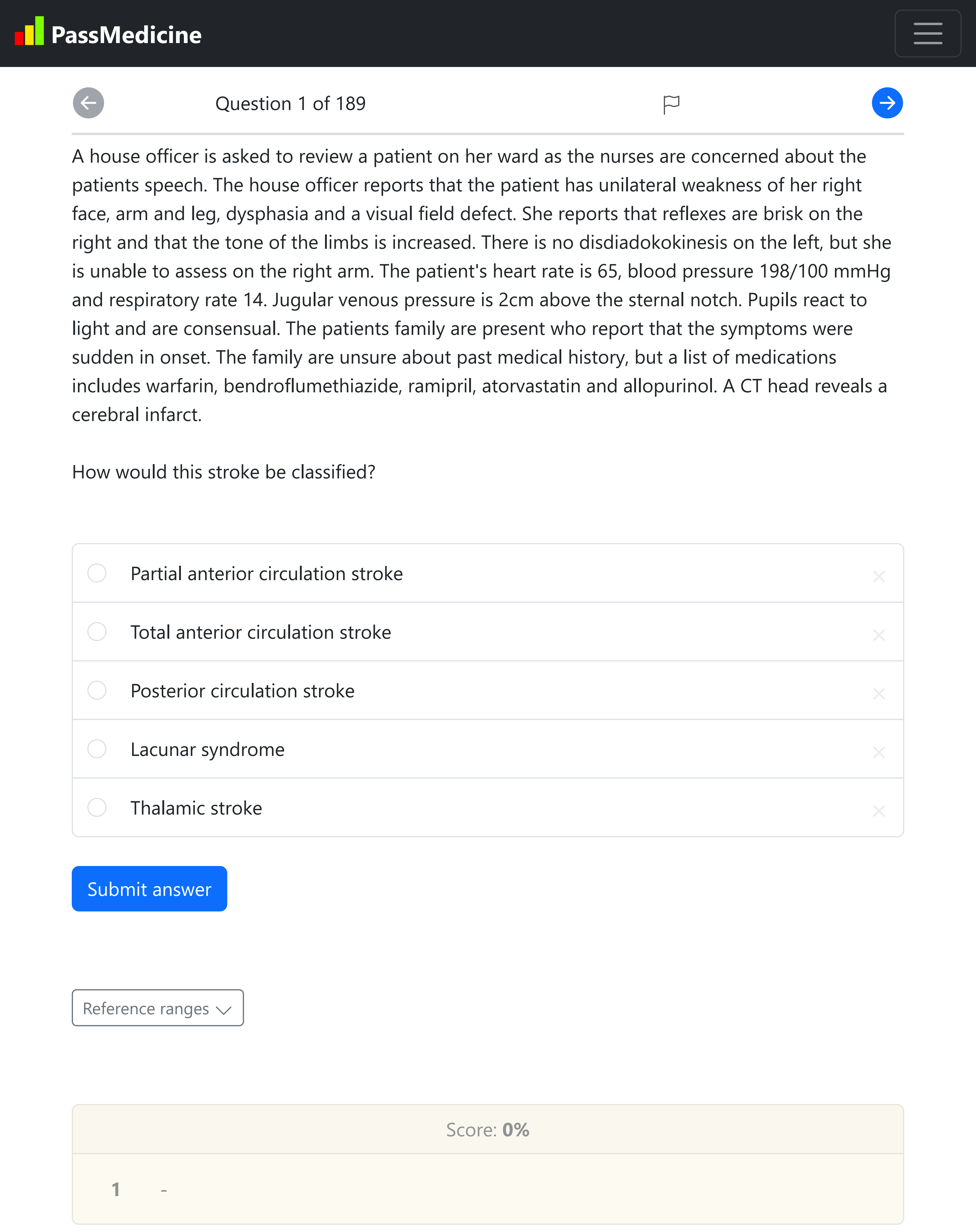

A house officer is asked to review a patient on her ward as the nurses are concerned about the patients speech. The house officer reports that the patient has unilateral weakness of her right face, arm and leg, dysphasia and a visual field defect. She reports that reflexes are brisk on the right and that the tone of the limbs is increased. There is no disdiadokokinesis on the left, but she is unable to assess on the right arm. The patient's heart rate is 65, blood pressure 198/100 mmHg and respiratory rate 14. Jugular venous pressure is 2cm above the sternal notch. Pupils react to light and are consensual. The patients family are present who report that the symptoms were sudden in onset. The family are unsure about past medical history, but a list of medications includes warfarin, bendroflumethiazide, ramipril, atorvastatin and allopurinol. A CT head reveals a cerebral infarct.

How would this stroke be classified?

- ○ Partial anterior circulation stroke
- ○ Total anterior circulation stroke
- ○ Posterior circulation stroke
- ○ Lacunar syndrome
- ○ Thalamic stroke

Score: **0%**

1 -

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A house officer is asked to review a patient on her ward as the nurses are concerned about the patients speech. The house officer reports that the patient has unilateral weakness of her right face, arm and leg, dysphasia and a visual field defect. She reports that reflexes are brisk on the right and that the tone of the limbs is increased. There is no disdiadokokinesis on the left, but she is unable to assess on the right arm. The patient's heart rate is 65, blood pressure 198/100 mmHg and respiratory rate 14. Jugular venous pressure is 2cm above the sternal notch. Pupils react to light and are consensual. The patients family are present who report that the symptoms were sudden in onset. The family are unsure about past medical history, but a list of medications includes warfarin, bendroflumethiazide, ramipril, atorvastatin and allopurinol. A CT head reveals a cerebral infarct.

How would this stroke be classified?

Partial anterior circulation stroke	14%
Total anterior circulation stroke	68%
Posterior circulation stroke	8%
Lacunar syndrome	7%
Thalamic stroke	3%

The correct answer is **Total anterior circulation stroke**. A total anterior circulation stroke (TACS) is characterised by the presence of all three features: motor or sensory deficit affecting at least two out of three areas (face, arm and leg), homonymous hemianopia, and higher cerebral dysfunction (e.g. dysphasia). In this case, the patient presents with unilateral weakness of her right face, arm and leg, dysphasia and a visual field defect which are indicative of a TACS.

A **Partial anterior circulation stroke** is incorrect because it does not involve all three areas mentioned above. It typically involves at least two out of the three areas but not all. The patient's presentation indicates involvement of all these areas hence suggesting a total rather than partial anterior circulation stroke.

Posterior circulation stroke is also incorrect as it usually results in symptoms such as vertigo, nausea/vomiting, nystagmus and cranial nerve palsy which are not reported in this case. The posterior circulation supplies the brainstem, cerebellum and occipital lobes so deficits would be expected in these areas rather than the unilateral motor/sensory loss described.

Lacunar syndrome is another incorrect option. Lacunar strokes result from the occlusion of small penetrating arteries that provide blood to the brain's deep structures. They typically present with pure motor or sensory symptoms and do not include cortical signs such as dysphasia or visual field defects.

Lastly, **Thalamic stroke** is also an incorrect choice. Thalamic strokes can present with various symptoms including sensory loss, weakness, speech difficulties and behavioural changes depending on which part of the thalamus was affected. However, they do not generally cause widespread motor deficits as seen in this patient's presentation.

👍 | 👎 | 💬 Discuss (4) | Improve

Next question ›

Stroke: types ★

The **Oxford Stroke Classification** (also known as the Bamford Classification) classifies strokes based on the initial symptoms. A summary is as follows:

The following criteria should be assessed:

- 1. unilateral hemiparesis and/or hemisensory loss of the face, arm & leg
- 2. homonymous hemianopia
- 3. higher cognitive dysfunction e.g. dysphasia

Total anterior circulation infarcts (TACI, c. 15%)

- involves middle and anterior cerebral arteries
- all 3 of the above criteria are present

Partial anterior circulation infarcts (PACI, c. 25%)

- involves smaller arteries of anterior circulation e.g. upper or lower division of middle cerebral artery
- 2 of the above criteria are present

Lacunar infarcts (LACI, c. 25%)

- involves perforating arteries around the internal capsule, thalamus and basal ganglia

- presents with 1 of the following:
- 1. unilateral weakness (and/or sensory deficit) of face and arm, arm and leg or all three.
- 2. pure sensory stroke.
- 3. ataxic hemiparesis

Posterior circulation infarcts (POCI, c. 25%)

- involves vertebrobasilar arteries
- presents with 1 of the following:
- 1. cerebellar or brainstem syndromes
- 2. loss of consciousness
- 3. isolated homonymous hemianopia

Other recognised patterns of stroke:

Lateral medullary syndrome (posterior inferior cerebellar artery)

- aka Wallenberg's syndrome
- ipsilateral: ataxia, nystagmus, dysphagia, facial numbness, cranial nerve palsy e.g. Horner's
- contralateral: limb sensory loss

Weber's syndrome

- ipsilateral III palsy
- contralateral weakness

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

Strokes & The Rule of 4s

Dirty Medicine - YouTube 👍 19 👎 0

Normal HIT (worrisome)

The HINTS exam in vertigo

Individual - YouTube 👍 5 👎 0

Brainstem Stroke

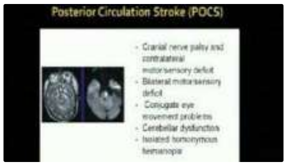
Brainstem stroke syndromes

Soton Brain Hub - YouTube 👍 3 👎 0

TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 2 👎 0



Posterior Circulation Stroke (POCS)

- Cranial nerve palsy and contralateral motor/sensory deficit
- Bilateral motor/sensory deficit
- Conjugate eye movement problems
- Cerebellar dysfunction
- Isolated homonymous hemianopia

Bamford classification

Individual - YouTube 👍 1 👎 0

[Stroke](#)

Podmedics - YouTube 👍 0 👎 0

NEUROLOGY
MIDBRAIN
SYNDROMES

Midbrain Syndromes - Weberâ€™s Syndrome, Benediktâ€™s Syndrome and Parinaud Syndrome

Armando Hasudungan - YouTube 👍 0 👎 0

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Score: **48%**

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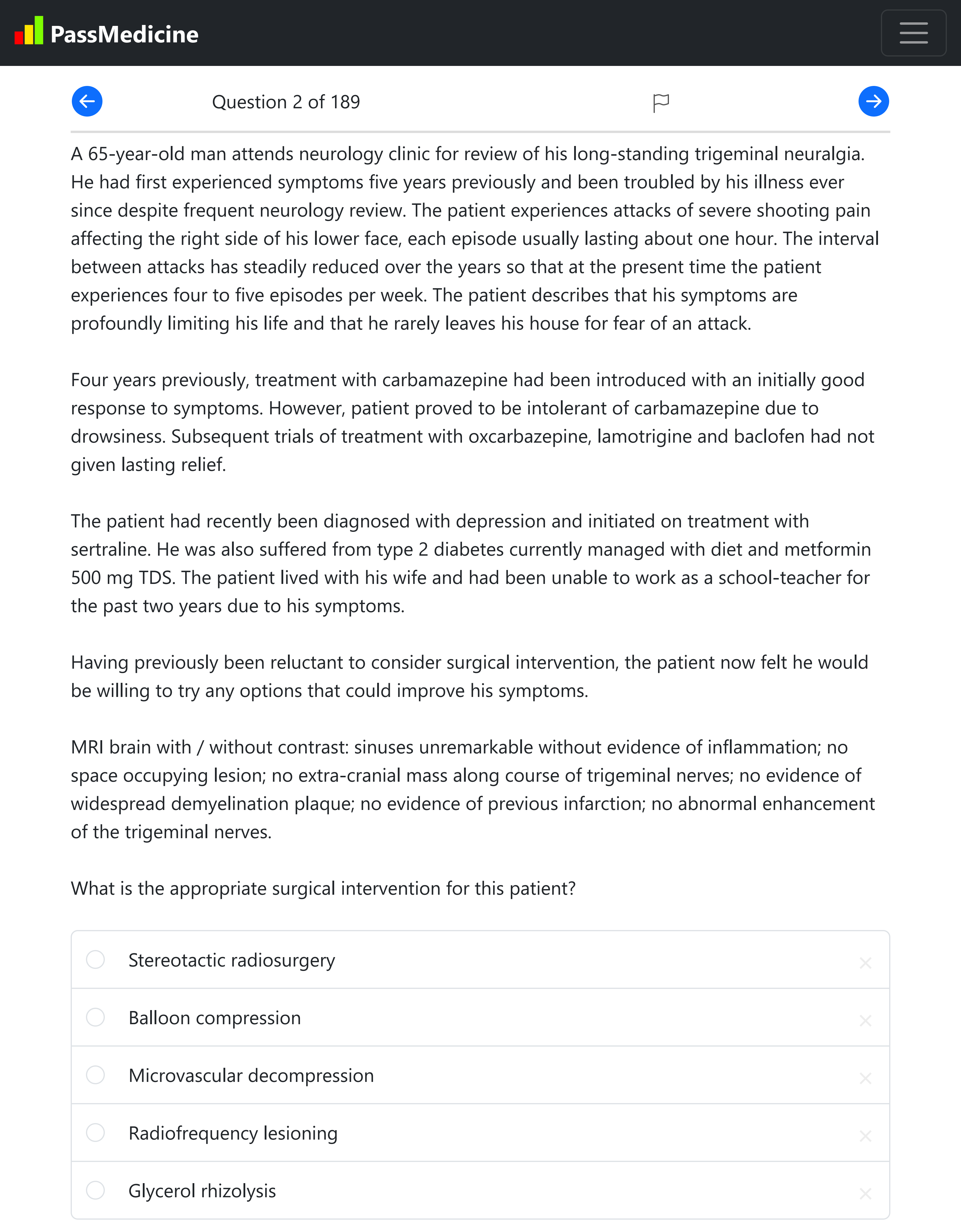

A 65-year-old man attends neurology clinic for review of his long-standing trigeminal neuralgia. He had first experienced symptoms five years previously and been troubled by his illness ever since despite frequent neurology review. The patient experiences attacks of severe shooting pain affecting the right side of his lower face, each episode usually lasting about one hour. The interval between attacks has steadily reduced over the years so that at the present time the patient experiences four to five episodes per week. The patient describes that his symptoms are profoundly limiting his life and that he rarely leaves his house for fear of an attack.

Four years previously, treatment with carbamazepine had been introduced with an initially good response to symptoms. However, patient proved to be intolerant of carbamazepine due to drowsiness. Subsequent trials of treatment with oxcarbazepine, lamotrigine and baclofen had not given lasting relief.

The patient had recently been diagnosed with depression and initiated on treatment with sertraline. He was also suffered from type 2 diabetes currently managed with diet and metformin 500 mg TDS. The patient lived with his wife and had been unable to work as a school-teacher for the past two years due to his symptoms.

Having previously been reluctant to consider surgical intervention, the patient now felt he would be willing to try any options that could improve his symptoms.

MRI brain with / without contrast: sinuses unremarkable without evidence of inflammation; no space occupying lesion; no extra-cranial mass along course of trigeminal nerves; no evidence of widespread demyelination plaque; no evidence of previous infarction; no abnormal enhancement of the trigeminal nerves.

What is the appropriate surgical intervention for this patient?

- ○ Stereotactic radiosurgery
- ○ Balloon compression
- ○ Microvascular decompression
- ○ Radiofrequency lesioning
- ○ Glycerol rhizolysis

Submit answer

Reference ranges

Score: **0%**

1	-
2	-

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What is the appropriate surgical intervention for this patient?

Stereotactic radiosurgery	20%
Balloon compression	3%
🟩 Microvascular decompression	55%
Radiofrequency lesioning	15%
🟥 Glycerol rhizolysis	8%

The patient has long-standing and severe trigeminal neuralgia with life-limiting symptoms despite appropriate medical treatment. Surgical intervention should therefore be considered.

In around 95 % of cases, trigeminal neuralgia is believed to be caused by compression of the trigeminal nerve by adjacent blood vessels, although the exact mechanism of the pathophysiology is incompletely understood. In a minority of cases, trigeminal neuralgia can be due to an alternative diagnosis such as sinusitis, extra-cranial or intra-cranial masses, multiple sclerosis or infarction of the thalamus or brainstem.

The MRI brain undergone by the patient does not provide evidence for any of the rare causes of trigeminal neuralgia with the patient's symptoms therefore very likely due to vascular compression. Using specialist techniques not included in the standard protocol, MRI can detect vascular contact with the trigeminal nerve. However, vascular contact with cranial nerves is also a normal variant in some individuals so these techniques are not routinely used.

Microvascular decompression is therefore the surgical technique of choice and is effective treatment in 95 % of such cases of trigeminal neuralgia. The other possible answers refer to methods of palliative destruction of the trigeminal nerve root, therefore carrying the risk of causing facial numbness. However, they are effective in the minority of trigeminal neuralgia cases not caused by vascular compression.

Zakrzewska J, Linskey M. Trigeminal neuralgia. BMJ 2014;348:g474.

👍 👎 Discuss (3) Improve

Next question ›

Trigeminal neuralgia ★

Trigeminal neuralgia is a pain syndrome characterised by severe unilateral pain. The vast majority of cases are idiopathic but compression of the trigeminal roots by tumours or vascular problems may occur.

The International Headache Society defines trigeminal neuralgia as:

- a unilateral disorder characterised by brief electric shock-like pains, abrupt in onset and termination, limited to one or more divisions of the trigeminal nerve
- the pain is commonly evoked by light touch, including washing, shaving, smoking, talking, and brushing the teeth (trigger factors), and frequently occurs spontaneously
- small areas in the nasolabial fold or chin may be particularly susceptible to the precipitation of pain (trigger areas)

- the pains usually remit for variable periods

The following as red flag symptoms and signs suggesting a serious underlying cause:

- Sensory changes
- Deafness or other ear problems
- History of skin or oral lesions that could spread perineurally
- Pain only in the ophthalmic division of the trigeminal nerve (eye socket, forehead, and nose), or bilaterally
- Optic neuritis
- A family history of multiple sclerosis
- Age of onset before 40 years

Management

- carbamazepine is first-line
- failure to respond to treatment or atypical features (e.g. < 50 years old) should prompt referral to neurology

+ Q 123

Next question >

B *I* A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 19 👎 3

Trigeminal Neuralgia guidelines

Suggest link Report broken link

Score: **48%**

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A 42-year-old woman attends neurology clinic for review of her treatment strategy for multiple sclerosis. The patient had first developed symptoms of multiple sclerosis five years previously. Her first episode had involved sensory loss and motor weakness affecting her left leg. An MRI brain scan conducted at this time had demonstrated multiple lesions suggestive of multiple sclerosis. At this point in time, the patient had decided against receiving disease modifying therapy.

One year after her first episode, the patient suffered an episode of optic neuritis. A repeat MRI scan had demonstrated a progression of the previously observed radiographic lesions. Subsequently, the patient had been initiated on treatment with interferon beta. This treatment was continued for two years during which time the patient suffered no further relapses of multiple sclerosis. However, the patient found the unwanted effects of interferon beta to be progressively harder to tolerate and she eventually decided against continuing with this treatment. A subsequent trial of glatiramer acetate was quickly halted due to the patient experiencing severe symptoms of flushing.

Following the cessation of disease-modifying therapy, the patient had experienced multiple further relapses, including one episode where she had required hospitalisation due to cranial nerve involvement. Further assessment during this period found that the patient met the local criteria for aggressive or highly active multiple sclerosis. Accordingly, later line therapies were discussed with the patient who was keen to proceed.

Aside from her history of multiple sclerosis the patient suffered from no other significant medical problems and took no regular medications. The patient was a science teacher that had been unable to work for the past year due to her multiple relapses.

The later line therapy recommended for the patient is natalizumab. What is the essential investigation that must be completed prior to initiation of treatment with natalizumab?

○ Transthoracic echocardiogram

○ JCV antibody status

○ Pulmonary function tests

○ Thyroid function tests

○ CMV antibody status

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Reference ranges

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2 -
3 -

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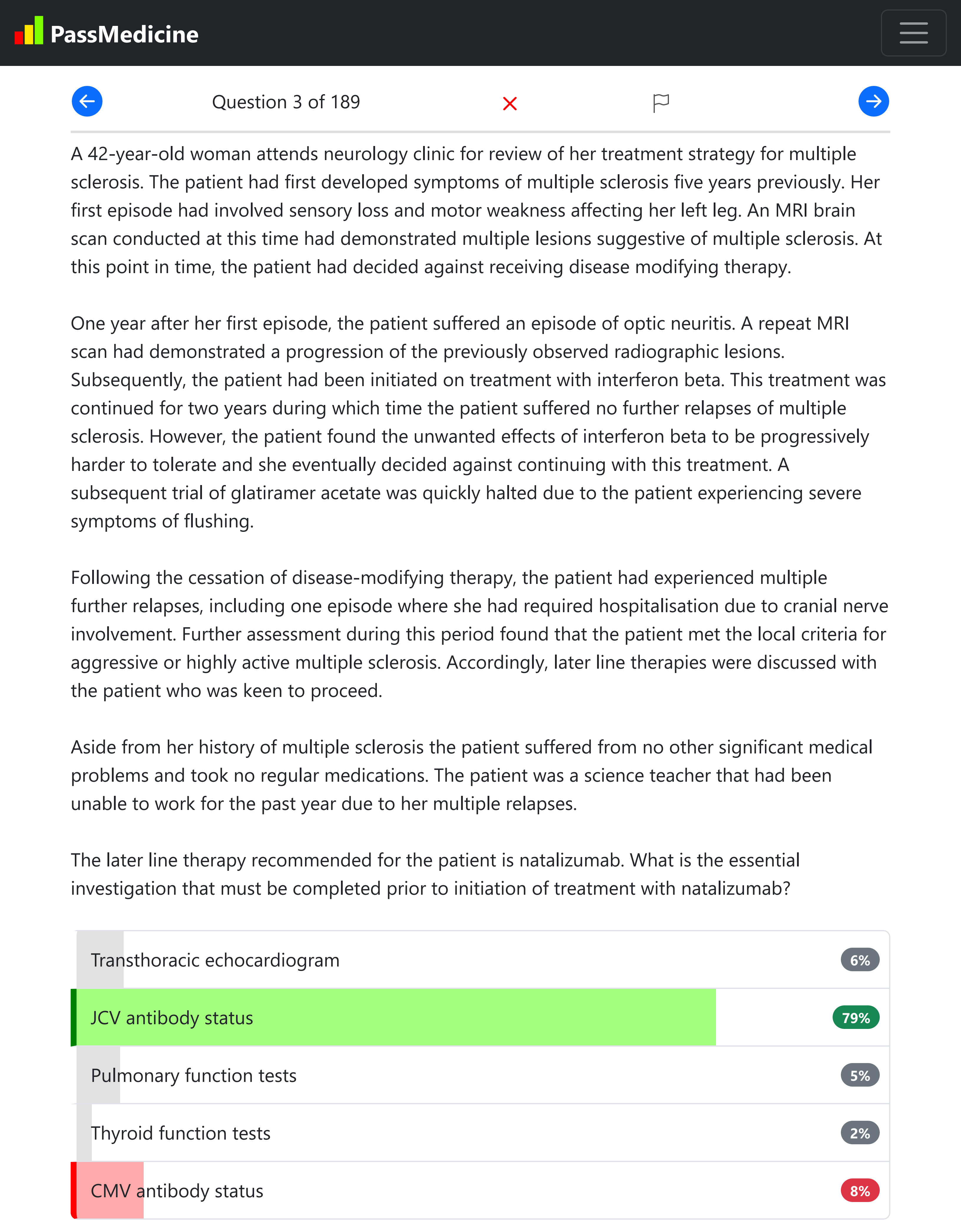

Question 3 of 189

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The later line therapy recommended for the patient is natalizumab. What is the essential investigation that must be completed prior to initiation of treatment with natalizumab?

Transthoracic echocardiogram	6%
JCV antibody status	79%
Pulmonary function tests	5%
Thyroid function tests	2%
CMV antibody status	8%

The patient has highly active multiple sclerosis causing significant morbidity. While she had obtained benefit from the standard initial therapies interferon beta and glatiramer acetate, these treatments were ultimately not tolerated. In this situation, consideration of escalation to second-line disease-modifying therapy is appropriate. Observational studies have reported that escalation from a first-line therapy to a second-line drug reduces relapse rate at one year.

Evidence suggests that alemtuzumab, natalizumab and fingolimod outperformed other disease-modifying therapies at preventing relapse at 2 years. In addition, natalizumab also reduced 2-year disability progression rates.

Natalizumab is strongly associated with progressive multifocal leukoencephalopathy, an infection of the central nervous system with the John Cunningham virus (JCV). Therefore, JCV antibody status is an essential part of assessing an individual for treatment with natalizumab. Patient's who are seropositive for JCV are not treated with natalizumab or switched to another drug.

Wingerchuk D, Weinshenker B. Disease-modifying therapies for relapsing multiple sclerosis. *BMJ* 2016;354:i3518.

👍 👎 💬 Discuss (4) Improve

Next question >

Multiple sclerosis: management ★

Treatment in multiple sclerosis is focused on reducing the frequency and duration of relapses. There is no cure.

Acute relapse

High-dose steroids (e.g. oral or IV methylprednisolone) may be given for 5 days to shorten the length of an acute relapse. It should be noted that steroids shorten the duration of a relapse and do not alter the degree of recovery (i.e. whether a patient returns to baseline function)

Disease modifying drugs

A number of drugs have been shown to reduce the risk of relapse in patients with MS. Typical indications for disease-modifying drugs include:

- relapsing-remitting disease + 2 relapses in past 2 years + able to walk 100m unaided

- secondary progressive disease + 2 relapses in past 2 years + able to walk 10m (aided or unaided)

Drug options for reducing the risk of relapse in MS

- natalizumab
    - a recombinant monoclonal antibody that antagonises alpha-4 beta-1-integrin found on the surface of leucocytes
    - inhibit migration of leucocytes across the endothelium across the blood-brain barrier
    - generally considered to have the strongest evidence base for preventing relapse of the disease-modifying and hence is often used first-line
    - given intravenously
- ocrelizumab
    - humanized anti-CD20 monoclonal antibody
    - like natalizumab, it is considered a high-efficacy drug that is often used first-line
    - given intravenously
- fingolimod
    - sphingosine 1-phosphate (S1P) receptor modulator
    - prevents lymphocytes from leaving lymph nodes
    - oral formulations are available
- beta-interferon
    - not considered to be as effective as alternative disease-modifying drugs
    - given subcutaneously/intramuscularly
- glatiramer acetate
    - immunomodulating drug - acts as an 'immune decoy'
    - given subcutaneously
    - along with beta-interferon considered an 'older drug' with less effectiveness compared to monoclonal antibodies and S1P) receptor modulators

Some specific problems

Fatigue

- once other problems (e.g. anaemia, thyroid or depression) have been excluded NICE recommend a trial of amantadine
- other options include mindfulness training and CBT

Spasticity

- baclofen and gabapentin are first-line. Other options include diazepam, dantrolene and tizanidine
- physiotherapy is important
- cannabis and botox are undergoing evaluation

Bladder dysfunction

- may take the form of urgency, incontinence, overflow etc
- guidelines stress the importance of getting an ultrasound first to assess bladder emptying - anticholinergics may worsen symptoms in some patients
- if significant residual volume → intermittent self-catheterisation
- if no significant residual volume → anticholinergics may improve urinary frequency

Oscillopsia (visual fields appear to oscillate)

- gabapentin is first-line

+ Q 123

Next question >

Textbooks

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Extended textbook

Links

Multiple Sclerosis Society 👍 6 👎 15

Primary care guidelines

NICE 👍 15 👎 7

2014 Multiple Sclerosis guidelines

Suggest link

Report broken link

Media

Multiple sclerosis

Podmedics - YouTube 👍 4 👎 0

Multiple Sclerosis

Multiple sclerosis

Soton Brain Hub - YouTube 👍 3 👎 0

DEMYELINATION
T-CELLS
PLAQUES
MULTIPLE SCLEROSIS
OLIGO CLONAL BANDS
RELAPSING-REMITTING

Multiple sclerosis

Rhesus Medicine - YouTube 👍 1 👎 0

MULTIPLE SCLEROSIS

Multiple sclerosis - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 7 👎 1

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Score: **48%**

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Question 4 of 189

A 45-year-old lady presented to the emergency department with prolonged fever and malaise, She had a new murmur and clinical signs clearly indicating subacute bacterial endocarditis. ECHO confirms vegetations on her aortic valve and blood culture is still pending. She is a known case of myasthenia gravis and has been warned against taking some groups of antibiotics.

Which of the following antibiotics can worsen the symptoms of myasthenia gravis?

- ○ Gentamicin ×
- ○ Augmentin ×
- ○ Meropenem ×
- ○ Benzylpenicillin ×
- ○ Trimethoprim sulphamethoxazole ×

Submit answer

Reference ranges

Score: **0%**

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Which of the following antibiotics can worsen the symptoms of myasthenia gravis?

Gentamicin	80%
Augmentin	2%
Meropenem	1%
Benzylpenicillin	4%
Trimethoprim sulphamethoxazole	13%

Answer: Gentamicin.

Aminoglycosides are known to worsen myasthenia gravis. Other medications that have this effect include: ciprofloxacin, verapamil, beta blockers like propranolol, lithium, D-penicillamine and muscle relaxants like Botox.

Update on myasthenia gravis B R Thanvi1, T C N Lo2 http://pmj.bmj.com/content/80/950/690.full

Discuss (4) Improve

Next question ›

Myasthenia gravis: exacerbating factors

The most common exacerbating factor is exertion resulting in fatigability, which is the hallmark feature of myasthenia gravis.. Symptoms become more marked during the day

The following drugs may exacerbate myasthenia:

- penicillamine
- quinidine, procainamide
- beta-blockers
- lithium
- phenytoin
- antibiotics: gentamicin, macrolides, quinolones, tetracyclines

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians (Edin) 👍 4 👎 2

Myasthenia gravis review

Myasthenia Gravis Association 👍 9 👎 3

Drugs which may aggravate myasthenia gravis

Suggest link

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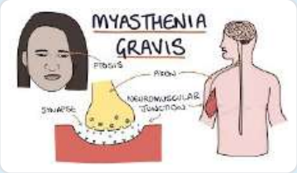
Media

MYASTHENIA GRAVIS

Myasthenia gravis - causes, symptoms, treatment, pathology

Osmosis - YouTube 👍 1 👎 0


MYASTHENIA GRAVIS
PTOSIS
AXON
NEUROMUSCULAR JUNCTION
SYNAPSE

Myasthenia gravis

Zero to Finals - YouTube 👍 2 👎 1

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Score: **48%**

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A 36-year-old female presents to clinic with transient visual loss. She reports three episodes over the last few months where her vision 'turns black' in both eyes despite being alert. This lasts for a few seconds and is then followed by a unilateral throbbing headache associated with nausea and phonophobia. It is worse on exertion and lasts for a couple of days. On examination her visual acuity is 20/20 bilaterally, her visual fields are normal and fundoscopy is unremarkable.

What is the most likely diagnosis?

- ○ Transient ischaemic attack ×
- ○ Amaurosis fugax ×
- ○ Anterior ischaemic optic neuropathy ×
- ○ Temporal arteritis ×
- ○ Migraine with aura ×

Submit answer

Reference ranges

Score: **0%**

1 -
2 -
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4 -
5 -

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Question 5 of 189 ✓

A 36-year-old female presents to clinic with transient visual loss. She reports three episodes over the last few months where her vision 'turns black' in both eyes despite being alert. This lasts for a few seconds and is then followed by a unilateral throbbing headache associated with nausea and phonophobia. It is worse on exertion and lasts for a couple of days. On examination her visual acuity is 20/20 bilaterally, her visual fields are normal and fundoscopy is unremarkable.

What is the most likely diagnosis?

Transient ischaemic attack	1%
Amaurosis fugax	16%
Anterior ischaemic optic neuropathy	4%
Temporal arteritis	3%
Migraine with aura ✓	75%

The headache in this patient is very typical of migraine which can be preceded by an aura lasting between 5 minutes to an hour usually. This patient's visual loss was relatively brief and in the context of unilateral vision loss would certainly raise the possibility of transient ischaemic attack (TIA) However, she reports her vision turned black in both eyes which would be very unlikely with a TIA as there would have needed to be simultaneous involvement of both anterior visual pathways. Since there is isolated bilateral transient visual loss if this was a TIA it would be of the occipital cortex from occlusion of the posterior cerebral artery although this is very unlikely given it does not appear to be a homonymous hemianopia she describes, and there is a characteristic history of migraine which is the most common cause of transient bilateral visual loss in young adults. Amaurosis fugax specifies a type of TIA, which typically causes a unilateral visual disturbance.

Temporal arteritis represents an arteritic form of anterior ischaemic optic neuropathy. Temporal arteritis tends to present later in life and may be associated with other symptoms like scalp tenderness, and jaw claudication. The non-arteritic form of anterior ischaemic optic neuropathy tends to be related to cardiovascular factors, and therefore again presents later in life and more commonly restricted to one eye.

Discuss (3) | Improve

Next question >

Migraine: diagnostic criteria

The International Headache Society has produced the following diagnostic criteria for migraine without aura:

Point	Criteria
A	At least 5 attacks fulfilling criteria B-D
B	Headache attacks lasting 4-72 hours* (untreated or unsuccessfully treated)
C	Headache has at least two of the following characteristics: • 1. unilateral location* • 2. pulsating quality (i.e., varying with the heartbeat) • 3. moderate or severe pain intensity • 4. aggravation by or causing avoidance of routine physical activity (e.g., walking or climbing stairs)
D	During headache at least one of the following: • 1. nausea and/or vomiting* • 2. photophobia and phonophobia
E	Not attributed to another disorder (history and examination do not suggest a secondary headache disorder or, if they do, it is ruled out by appropriate investigations or headache attacks do not occur for the first time in close temporal relation to the other disorder)

*In children, attacks may be shorter-lasting, headache is more commonly bilateral, and gastrointestinal disturbance is more prominent.

Migraine with aura (seen in around 25% of migraine patients) tends to be easier to diagnose with a typical aura being progressive in nature and may occur hours prior to the headache. Typical aura include a transient hemianopic disturbance or a spreading scintillating scotoma ('jagged crescent'). Sensory symptoms may also occur

If we compare these guidelines to the **NICE criteria** the following points are noted:

- NICE suggests migraines may be unilateral or bilateral
- NICE also give more detail about typical auras:

Auras may occur with or without headache and:

- are fully reversible
- develop over at least 5 minutes
- last 5-60 minutes

The following aura symptoms are atypical and may prompt further investigation/referral;

- motor weakness
- double vision
- visual symptoms affecting only one eye
- poor balance
- decreased level of consciousness.

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

International Headache Society 👍 7 👎 5

2019 The International Classification of Headache Disorders

Suggest link

Report broken link

Media

Management of Migraine
Podmedics

Migraine

Podmedics - YouTube 👍 4 👎 0

Migraine

Townsend Teaching - YouTube 👍 0 👎 0

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Score: **48%**

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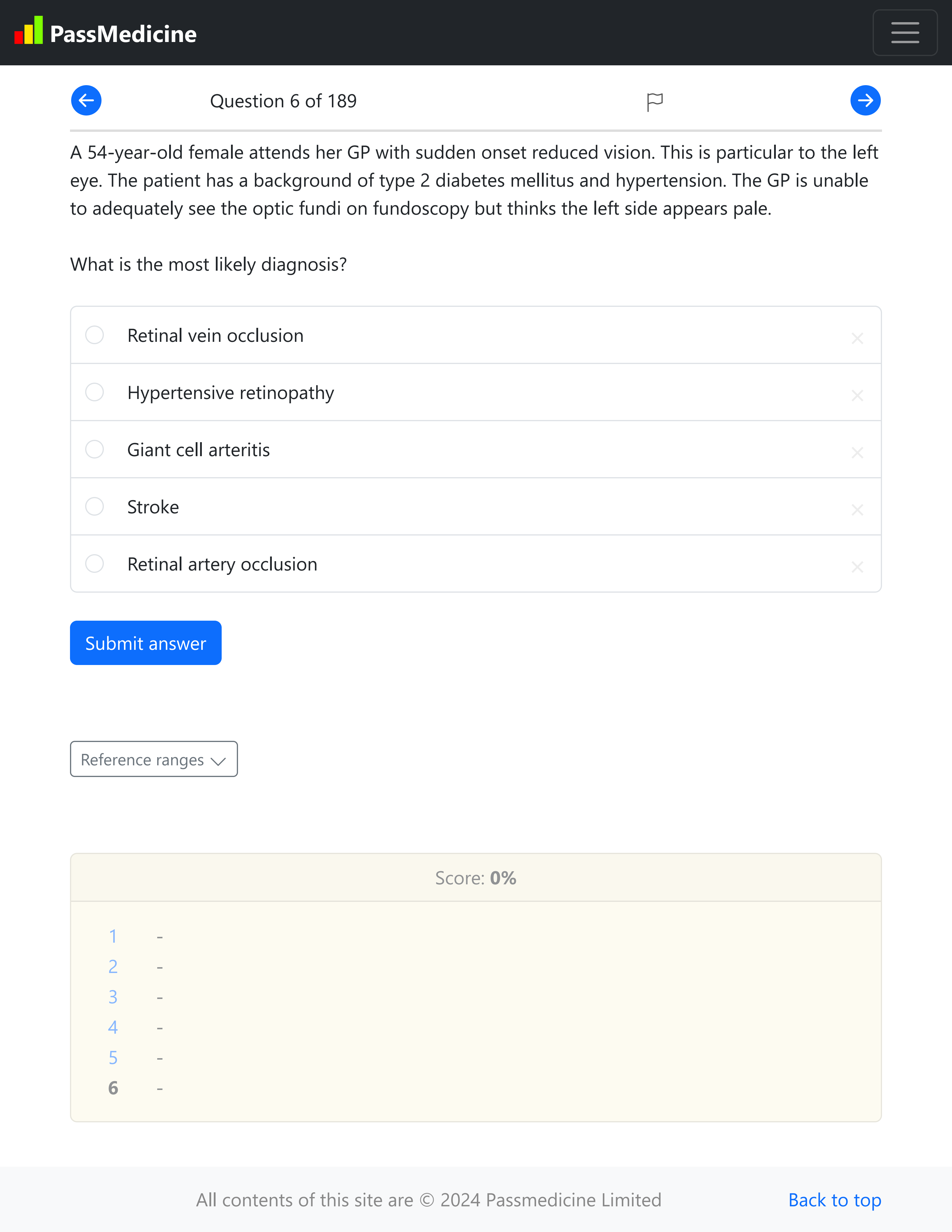

Question 6 of 189

A 54-year-old female attends her GP with sudden onset reduced vision. This is particular to the left eye. The patient has a background of type 2 diabetes mellitus and hypertension. The GP is unable to adequately see the optic fundi on fundoscopy but thinks the left side appears pale.

What is the most likely diagnosis?

- ○ Retinal vein occlusion
- ○ Hypertensive retinopathy
- ○ Giant cell arteritis
- ○ Stroke
- ○ Retinal artery occlusion

Score: **0%**

1 -
2 -
3 -
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Question 6 of 189 ✓

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What is the most likely diagnosis?

Retinal vein occlusion	13%
Hypertensive retinopathy	3%
Giant cell arteritis	4%
Stroke	1%
Retinal artery occlusion	79%

Retinal artery occlusion is associated with cardiovascular risk factors as in this case. It causes sudden onset painless visual loss. Fundoscopy may reveal a pale retina and the macula can appear as a 'cherry red spot'.

Retinal vein occlusion may present with distorted and blurred vision rather than visual loss. Additionally, fundoscopy may reveal vascular dilatation with associated haemorrhages.

Hypertensive retinopathy tends to be asymptomatic and is instead generally an indicator of end-organ damage due to long-term effects of hypertension.

Giant cell arteritis may be expected to have preceding symptoms of headache, jaw pain and scalp tenderness before abrupt visual loss.

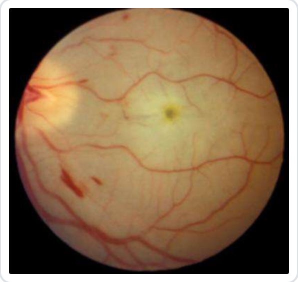
Discuss (6) | Improve

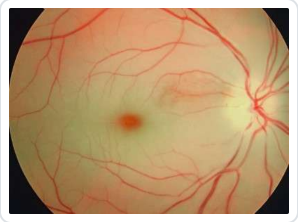
Next question

Central retinal artery occlusion

Central retinal artery occlusion is a relatively rare cause of sudden unilateral visual loss. It is due to thromboembolism (from atherosclerosis) or arteritis (e.g. temporal arteritis)

Features
- sudden, painless unilateral visual loss
- relative afferent pupillary defect
- 'cherry red' spot on a pale retina





Management is difficult and the prognosis is poor
- any underlying conditions should be identified and treated (e.g. intravenous steroids for temporal arteritis)
- if a patient presents acutely then Intraarterial thrombolysis may be attempted but currently, trials show mixed results.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

American Academy of Ophthalmology 👍 9 👎 4

Central Retinal Artery Occlusion

Suggest link Report broken link

Score: **48%**

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Question 7 of 189

A 25 year-old woman with ataxia telangiectasia undergoes open biopsy of an enlarged cervical lymph node. Microscopic examination reveals changes consistent with nodular sclerosing Hodgkins disease. Unfortunately, she develops spreading cellulitis around the biopsy site which fails to respond to intravenous antibiotics and ultimately requires surgical debridement.

What is the most common reason for the increased susceptibility to infections in ataxia telangiectasia?

- ○ Hypogammaglobulinaemia
- ○ Neutropaenia
- ○ Hyposplenism
- ○ Defect in phagocyte NADPH oxidase
- ○ Complement deficiency

Submit answer

Reference ranges

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What is the most common reason for the increased susceptibility to infections in ataxia telangiectasia?

Hypogammaglobulinaemia	60%
Neutropaenia	5%
Hyposplenism	8%
Defect in phagocyte NADPH oxidase	18%
Complement deficiency	10%

Ataxia telangiectasia is an autosomal recessive condition resulting in cerebellar ataxia with ocular telangiectasia. Individuals are often immunocompromised, typically due to hypogammaglobulinaemia. Lymphopaenia may also contribute.

There is also an increased risk of malignancy, typically lymphoma or leukaemia.

A defect in phagocyte NADPH oxidase is responsible for chronic granulomatous disease.

Discuss (2) Improve

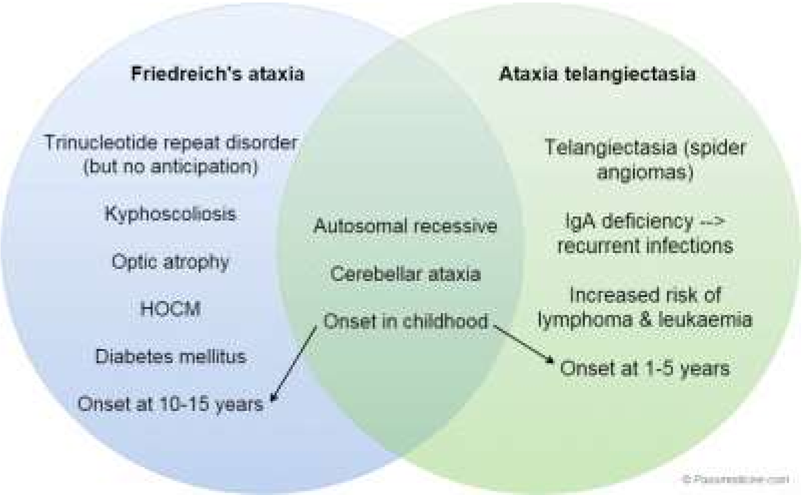
Next question

Ataxia telangiectasia

Ataxia telangiectasia is an autosomal recessive disorder caused by a defect in the ATM gene which encodes for DNA repair enzymes. It is one of the inherited combined immunodeficiency disorders. It typically presents in early childhood with abnormal movements.

Features

- cerebellar ataxia
- telangiectasia (spider angiomas)
- IgA deficiency resulting in recurrent chest infections
- 10% risk of developing malignancy, lymphoma or leukaemia, but also non-lymphoid tumours


Friedreich's ataxia
Trinucleotide repeat disorder (but no anticipation)
Kyphoscoliosis
Optic atrophy
HOCM
Diabetes mellitus
Onset at 10-15 years
Autosomal recessive
Cerebellar ataxia
Onset in childhood
Ataxia telangiectasia
Telangiectasia (spider angiomas)
IgA deficiency --> recurrent infections
Increased risk of lymphoma & leukaemia
Onset at 1-5 years
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Comparison of Friedreich's ataxia and ataxia telangiectasia. Note in particular how ataxia telangiectasia tends to present much earlier, often at the age of 1-2 years

123

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A 42 year-old man presents with an acute of onset of weakness in the left face, arm, and leg, which has not resolved. On examination there is upper motor neuron facial weakness on the left, with dense weakness of the left arm and leg. There is no evidence of sensory neglect, hemianopia, or dysphasia.

He past medical history includes migraine with visual aura, for which he takes propranolol and sumatriptan. His last migraine attack was a month ago. He has suffered two transient ischaemic attacks in the last year, and now also takes clopidogrel. His father also suffered from migraine with aura and died in his 50s after suffering a series of strokes.

Plain computed tomography shows multiple round lesions in the white matter, which appear the same density as cerebrospinal fluid. Magnetic resonance imaging shows scattered well-circumscribed lesions in the subcortical white matter which appear hypointense on T1 and hyperintense on T2-weighted sequences. On DWI (diffusion-weighted imaging) there is a hyperintense lesion in the right internal capsule, with a corresponding hypointense area on the ADC (apparent diffusion coefficient) map.

What is the most likely underlying diagnosis?

- ○ Patent foramen ovale
- ○ Progressive multifocal leukoencephalopathy
- ○ Migrainous infarction
- ○ Cerebral amyloid angiopathy
- ○ CADASIL

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Question 8 of 189 ✓

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What is the most likely underlying diagnosis?

Patent foramen ovale	4%
Progressive multifocal leukoencephalopathy	13%
Migrainous infarction	6%
Cerebral amyloid angiopathy	8%
CADASIL	69%

The clinical and radiological description fits with a lacunar infarction in the right internal capsule resulting in a pure motor hemiparesis. The area of acute infarction is not visible on the CT in the hyperacute phase, but is visible on the diffusion-weighted MR as described. The other white matter lesions are most likely old lacunar infarcts which may have been clinically silent.

The personal history of migraine with aura coupled with the family history make this most likely CADASIL (cerebral autosominal dominant arteriopathy with subcortical infarcts and leukoencephalopathy). The conditions results from mutations in the NOTCH3 gene on chromosome 19. Individuals typically develop migraine with aura around age 30, transient ischaemic attacks and ischaemic strokes in their 40s, and dementia around the 50s. Strokes are typically lacunar.

There is an association between patent foramen ovale, migraine with aura, and stroke. Stroke in this context is embolic, which characteristically produces wedge-shaped infarcts involving cortex and white matter, rather than the small lesions seen in lacunar stroke. Emboli may arise in the venous circulation and cross over into the arterial circulation via the shunt (paradoxical embolism).

Progressive multifocal leukoencephalopthy is a demyelinating disease of the central nervous system caused by reactivation of the JC virus, most typically seen in patients with AIDS and low CD4 counts.

In migranous infarction the neurological deficit develops during an attack of migraine.

Cerebral amyloid angiopathy is an important cause of primary lobar intracerebral haemhorrage in the elderly.

Discuss | Improve

Next question ›

CADASIL ★

Overview

- Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy (CADASIL)
- rare cause of multi-infarct dementia
- patients often present with migraine

AI + Q 123

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Media

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Rare Disease Report
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RDR

CADASIL Explained

Rare Disease Report - YouTube 👍 6 👎 2

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A 43-year-old man is apprehended by the police after a violent assault outside a pub. He is brought to the attention of the duty psychiatrist because he is extremely confused and agitated. He repeatedly says that his wife is having an affair with the Prime Minister. He believes this to be true because of a message encoded in the traffic lights. He also mentions that he has not slept in the last two days.

The psychiatric assessment is interrupted when he has a tonic-clonic seizure, which self-terminates in 3 minutes. He is taken to the local Accident and Emergency department and reviewed by the medical team.

The medical SHO manages to contact his wife, who reports that he is normally fit and well and she last saw him when he left for work five days ago. At that time he appeared normal but complained of a headache, and took some ibuprofen.

During the clerking, he is noted to have repetitive chewing movements of the mouth. Again, the assessment is interrupted by a tonic-clonic seizure which does not terminate after 10mg of intravenous diazepam. After 15 minutes he is loaded with phenytoin. He continues to fit so is intubated and transferred to the Intensive Care Unit. Termination of seizure activity is only achieved after induction of general anaesthesia with thiopentone.

What is the most likely diagnosis?

- ○ Schizophrenia
- ○ Fronto-temporal dementia
- ○ Acute intermittent porphyria
- ○ Post-ictal psychosis
- ○ Autoimmune limbic encephalitis

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What is the most likely diagnosis?

Schizophrenia	6%
Fronto-temporal dementia	8%
Acute intermittent porphyria	9%
Post-ictal psychosis	8%
Autoimmune limbic encephalitis	69%

Autoimmune limbic encephalitis presents with acute or sub-acute cognitive dysfunction, behavioural changes, and seizures. A number of specific antibody-related syndromes have been described. The description given here is suggestive of anti-NMDA receptor encephalitis, with a prodromal headache followed by prominent psychiatric features, with orofacial dyskinesias, insomnia, and progression to seizures.

Schizophrenia is a common cause of psychosis but the seizures should point away from a psychiatric disorder towards an organic brain pathology.

Fronto-temporal dementia is a slowly progressive condition.

Acute intermittent porphyria may cause a range of neurological manifestation including psychosis and seizures, but there are no features here that point specifically to this diagnosis.

Post-ictal psychosis is a rare manifestation of the post-ictal state, typically in patients who have had epilepsy for some time. The rapid development of psychosis and seizures in a previously well person should prompt the search for an alternative diagnosis.

👍 👎 Discuss (6) Improve

Next question >

Autoimmune encephalitis ★

Autoimmune encephalitis (AE) is a group of rare and potentially severe neurological disorders characterized by inflammation of the brain caused by an aberrant immune response. The immune system mistakenly targets and attacks healthy brain tissue, resulting in various neurological and psychiatric symptoms.

Types of autoimmune encephalitis include:
- autoimmune limbic encephalitis (paraneoplastic and non-paraneoplastic)
- Rasmussen's encephalitis
- anti-NMDAR (NR1) encephalitis
- glycine-receptor mediated encephalitis
- Bickerstaff brainstem encephalitis

Features
- Cognitive dysfunction: Memory impairment, confusion, and disorientation
- Seizures: Focal or generalized
- Movement disorders: Dystonia, chorea, or myoclonus
- Psychiatric symptoms: Anxiety, depression, hallucinations, or psychosis
- Autonomic dysfunction: Fluctuating blood pressure, heart rate irregularities, or temperature dysregulation
- Sleep disturbances: Insomnia or hypersomnia

Investigation

- Detection of specific autoantibodies in serum or cerebrospinal fluid (CSF)
- Neuroimaging: MRI to identify areas of inflammation or other abnormalities
- Electroencephalogram (EEG): To detect seizure activity or other abnormal brain wave patterns
- Additional tests: Screening for underlying malignancies, infections, or other autoimmune diseases

Management

- High-dose corticosteroids
- Intravenous immunoglobulin
- Plasma exchange (plasmapheresis)

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A 45 year-old man is referred to the nephrologists for investigation of chronic kidney disease. He has a past medical history of hypertension, type 2 diabetes, and Parkinsons disease, and his medication comprises ramipril, metformin, and bromocriptine.

Review of blood tests shows that the glomerular filtration rate (GFR) has steadily fallen from 85 to 44ml/min/1.73m^2 over the last year.

On questioning, his only symptom is chronic back pain which has been getting worse over the last year. On examination, both kidneys are easily palpable.

Routine investigations are as follows:

Hb	12.1 g/dl
MCV	94.2 fl
Platelets	264 x10^9/l
WCC	7.1 x10^9/l
Na	137mmol/l
K	4.6 mmol/l
Urea	13.8 mmol/l
Creatinine	157 mol/l
eGFR	44 ml/min/1.73m^2
ALT	24 IU/l
ALP	78 IU/l
Bilirubin	6 mol/l
Albumin	37 g/l
Total protein	64 g/l
Serum protein electrophoresis	pending
Urine dipstick	negative for blood, protein, leucocytes, and nitrites

Abdominal ultrasound shows bilateral hydronephrosis.

What is the most likely cause of his chronic kidney disease?

○ Multiple myeloma ×

○ Diabetic nephropathy ×

○ Bladder cancer ×

○ Retroperitoneal fibrosis ×

○ Autosomal dominant polycystic kidney disease ×

Submit answer

Reference ranges

Score: **0%**

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ALT	24 IU/l
ALP	78 IU/l
Bilirubin	6 mol/l
Albumin	37 g/l
Total protein	64 g/l
Serum protein electrophoresis	pending
Urine dipstick	negative for blood, protein, leucocytes, and nitrites

Abdominal ultrasound shows bilateral hydronephrosis.

What is the most likely cause of his chronic kidney disease?

Multiple myeloma	12%
Diabetic nephropathy	2%
Bladder cancer	4%
Retroperitoneal fibrosis	67%
Autosomal dominant polycystic kidney disease	15%

Bilateral hydronephrosis suggests that there is an obstruction to the outflow of both kidneys. This typically occurs at the bladder outlet, raising the possibility of a bladder or prostate cancer. The absence of haematuria makes bladder cancer less likely, and the absence of lower urinary tract symptoms makes prostate cancer less likely. However, if another diagnosis were not more forthcoming it would be appropriate to investigate for these conditions.

Bilateral hydronephrosis can more unusually result from obstruction of both ureters. This occurs in retroperitoneal fibrosis, where the ureters are encased in dense fibrous tissue. Back pain is a common symptom. The condition may be idiopathic, or may occur in association with an abdominal aortic aneurysm, or secondary to long-term use of ergot derived dopamine agonists such as bromocriptine, cabergoline, or pergolide.

In the context of back pain and chronic kidney disease, multiple myeloma is a distinct possibility. Serum protein electrophoresis is required to detect the presence of a paraprotein. However, subtracting total protein from albumin (64-37) suggests that the total immunoglobulin level is 27 g/L, and therefore there is unlikely to be a significant level of paraprotein. Equally, there is unlikely to be any urinary Bence Jones protein in the absence of dipstick proteinuria. Finally, one would not expect to see hydronephrosis in myeloma kidney.

The absence of proteinuria makes diabetic nephropathy less likely, and also one would not expect to see evidence of urinary tract obstruction in this condition.

At the age of 45, autosomal dominant polycystic kidney disease can be excluded by the absence of renal cysts on ultrasound.

Discuss (2) Improve

Next question

Parkinson's disease: management ★

Parkinsons disease should only be diagnosed, and management initiated, by a specialist with expertise in movement disorders. However, it is important for all doctors to be aware of the medications used in Parkinson's given the prevalence of this condition. NICE published guidelines in 2017 regarding the management of Parkinson's disease.

For first-line treatment:

- if the motor symptoms are affecting the patient's quality of life: levodopa
- if the motor symptoms are not affecting the patient's quality of life: dopamine agonist (non-ergot derived), levodopa or monoamine oxidase B (MAO-B) inhibitor

Whilst all drugs used to treat Parkinson's can cause a wide variety of side-effects NICE produced tables to help with decision making:

	Levodopa	**Dopamine agonists**	**MAO-B inhibitors**
Motor symptoms	More improvement in motor symptoms	Less improvement in motor symptoms	Less improvement in motor symptoms
Activities of daily living	More improvement in activities of daily living	Less improvement in activities of daily living	Less improvement in activities of daily living
Motor complications	More motor complications	Fewer motor complications	Fewer motor complications
Adverse events	Fewer specified adverse events*	More specified adverse events*	Fewer specified adverse events*

* excessive sleepiness, hallucinations and impulse control disorders

If a patient continues to have symptoms despite optimal levodopa treatment or has developed dyskinesia then NICE recommend the addition of a dopamine agonist, MAO-B inhibitor or catechol-O-methyl transferase (COMT) inhibitor as an adjunct. Again, NICE summarise the main points in terms of decision making:

	Dopamine agonists	**MAO-B inhibitors**	**COMT inhibitors**	**Amantadine**
Motor symptoms	Improvement in motor symptoms	Improvement in motor symptoms	Improvement in motor symptoms	No evidence of improvement in motor symptoms

	Dopamine agonists	MAO-B inhibitors	COMT inhibitors	Amantadine
Activities of daily living	Improvement in activities of daily living	Improvement in activities of daily living	Improvement in activities of daily living	No evidence of improvement in activities of daily living
Off time	More off-time reduction	Off-time reduction	Off-time reduction	No studies reporting this outcome
Adverse events	Intermediate risk of adverse events	Fewer adverse events	More adverse events	No studies reporting this outcome
Hallucinations	More risk of hallucinations	Lower risk of hallucinations	Lower risk of hallucinations	No studies reporting this outcome

Specific points regarding Parkinson's medication

NICE reminds us of the risk of acute akinesia or neuroleptic malignant syndrome if medication is not taken/absorbed (for example due to gastroenteritis) and advise against giving patients a 'drug holiday' for the same reason.

Impulse control disorders have become a significant issue in recent years. These can occur with any dopaminergic therapy but are more common with:

- dopamine agonist therapy
- a history of previous impulsive behaviours
- a history of alcohol consumption and/or smoking

If excessive daytime sleepiness develops then patients should not drive. Medication should be adjusted to control symptoms. Modafinil can be considered if alternative strategies fail.

If orthostatic hypotension develops then a medication review looking at potential causes should be done. If symptoms persist then midodrine (acts on peripheral alpha-adrenergic receptors to increase arterial resistance) can be considered.

Consider glycopyrronium bromide to manage drooling of saliva in people with Parkinson's

disease.

Further information regarding specific anti-Parkinson's medication

Levodopa

- nearly always combined with a decarboxylase inhibitor (e.g. carbidopa or benserazide)
    - this prevents the peripheral metabolism of levodopa to dopamine outside of the brain and hence can reduce side effects
- common adverse effects:
    - dry mouth
    - anorexia
    - palpitations
    - postural hypotension
    - psychosis
- some adverse effects are due to the difficulty in achieving a steady dose of levodopa
    - end-of-dose wearing off: symptoms often worsen towards the end of dosage interval. This results in a decline of motor activity
    - 'on-off' phenomenon: large variations in motor performance, with normal function during the 'on' period, and weakness and restricted mobility during the 'off' period
    - dyskinesias at peak dose: dystonia, chorea and athetosis (involuntary writhing movements)
    - these effects may worsen over time with - clinicians therefore may limit doses until necessary
- it is important not to acutely stop levodopa, for example, if a patient is admitted to hospital
    - if a patient with Parkinson's disease cannot take levodopa orally, they can be given a dopamine agonist patch as rescue medication to prevent acute dystonia

Dopamine receptor agonists

- e.g. bromocriptine, ropinirole, cabergoline, apomorphine
- ergot-derived dopamine receptor agonists (bromocriptine, cabergoline) have been associated with pulmonary, retroperitoneal and cardiac fibrosis. The Committee on Safety of Medicines advice that an echocardiogram, ESR, creatinine and chest x-ray should be obtained prior to treatment and patients should be closely monitored
- patients should be warned about the potential for dopamine receptor agonists to cause impulse control disorders and excessive daytime somnolence
- more likely than levodopa to cause hallucinations in older patients. Nasal congestion and postural hypotension are also seen in some patients

MAO-B (Monoamine Oxidase-B) inhibitors

- e.g. selegiline
- inhibits the breakdown of dopamine secreted by the dopaminergic neurons

Amantadine

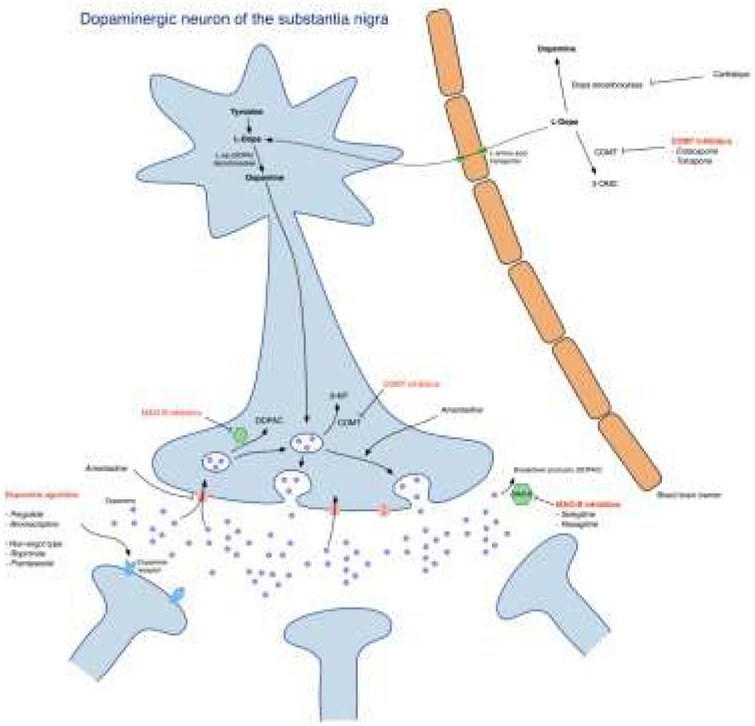
- mechanism is not fully understood, probably increases dopamine release and inhibits its uptake at dopaminergic synapses
- side-effects include ataxia, slurred speech, confusion, dizziness and livedo reticularis

COMT (Catechol-O-Methyl Transferase) inhibitors

- e.g. entacapone, tolcapone
- COMT is an enzyme involved in the breakdown of dopamine, and hence may be used as an adjunct to levodopa therapy
- used in conjunction with levodopa in patients with established PD

Antimuscarinics

- block cholinergic receptors
- now used more to treat drug-induced parkinsonism rather than idiopathic Parkinson's disease
- help tremor and rigidity
- e.g. procyclidine, benzotropine, trihexyphenidyl (benzhexol)


Dopaminergic neuron of the substantia nigra
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COMT
Amantadine
Amantadine
- Selegiline
- Rasagiline

Image sourced from Wikipedia

Diagram showing the mechanism of action of Parkinson's drugs

Next question

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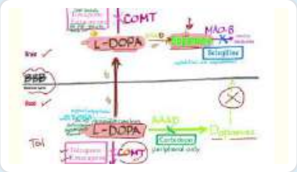
Links

NICE 👍 23 👎 12

2017 Parkinson's disease guidelines

Suggest link Report broken link

Media


COMT
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The use of Levodopa/ Carbidopa in the treatment of Parkinson's disease

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PARKINSON'S
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Parkinson's disease pharmacology

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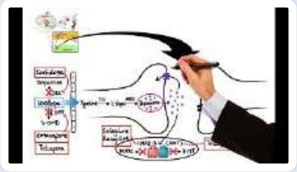

Pharmacology - DRUGS FOR PARKINSON'S DISEASE

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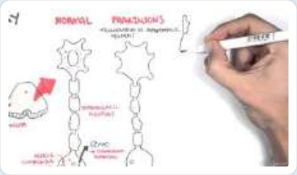
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Pharmacology - Parkinson's Disease

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Parkinson's disease

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SHUFFLING GAIT
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PILL ROLLING TREMOR
COG-WHEEL RIGIDITY
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Understanding Parkinson's Disease

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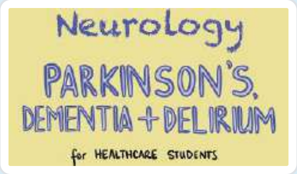
Neurology
PARKINSON'S, DEMENTIA + DELIRIUM
for HEALTHCARE STUDENTS

Parkinson's disease

MedFlix - YouTube 👍 1 👎 2

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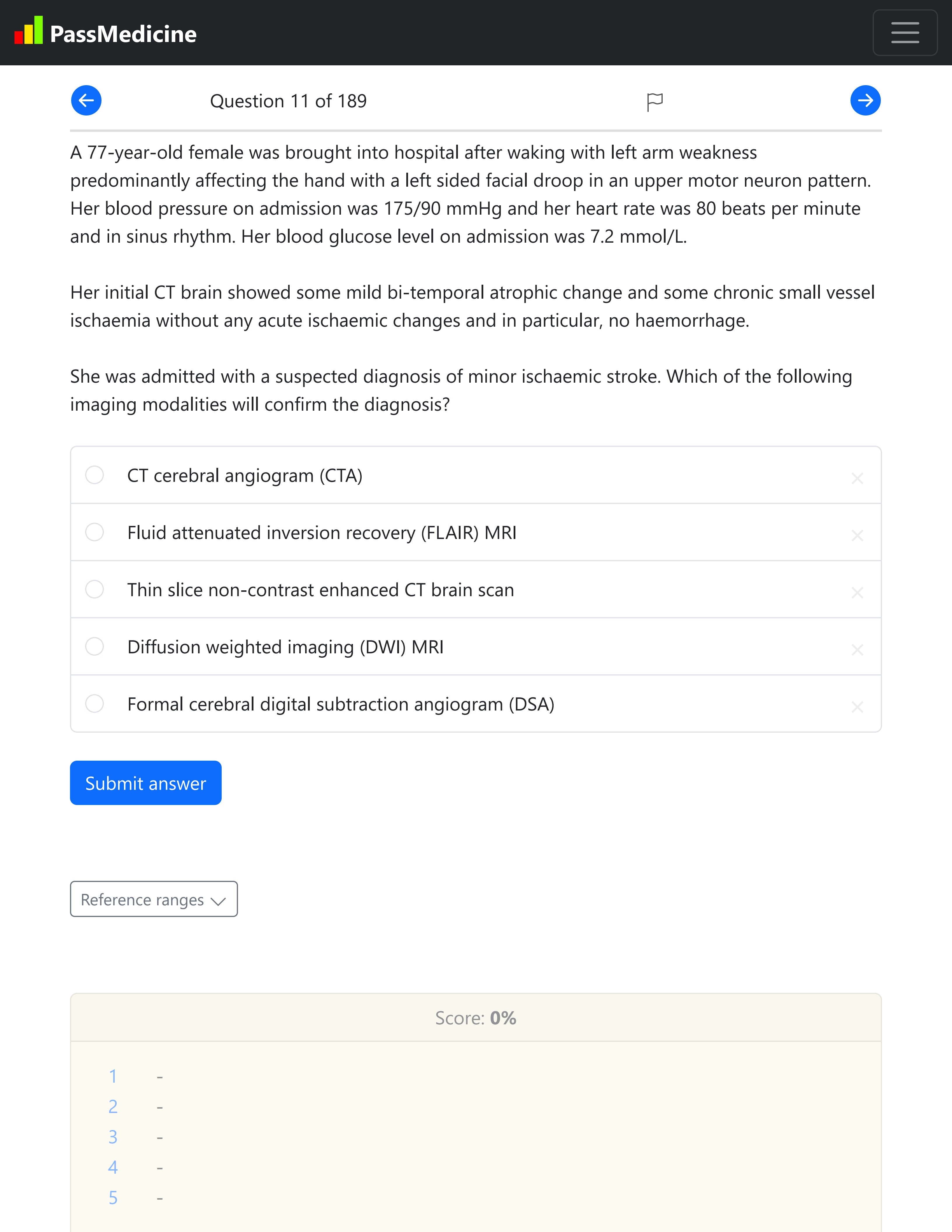

A 77-year-old female was brought into hospital after waking with left arm weakness predominantly affecting the hand with a left sided facial droop in an upper motor neuron pattern. Her blood pressure on admission was 175/90 mmHg and her heart rate was 80 beats per minute and in sinus rhythm. Her blood glucose level on admission was 7.2 mmol/L.

Her initial CT brain showed some mild bi-temporal atrophic change and some chronic small vessel ischaemia without any acute ischaemic changes and in particular, no haemorrhage.

She was admitted with a suspected diagnosis of minor ischaemic stroke. Which of the following imaging modalities will confirm the diagnosis?

- ○ CT cerebral angiogram (CTA) ×
- ○ Fluid attenuated inversion recovery (FLAIR) MRI ×
- ○ Thin slice non-contrast enhanced CT brain scan ×
- ○ Diffusion weighted imaging (DWI) MRI ×
- ○ Formal cerebral digital subtraction angiogram (DSA) ×

Score: **0%**

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Her initial CT brain showed some mild bi-temporal atrophic change and some chronic small vessel ischaemia without any acute ischaemic changes and in particular, no haemorrhage.

She was admitted with a suspected diagnosis of minor ischaemic stroke. Which of the following imaging modalities will confirm the diagnosis?

CT cerebral angiogram (CTA)	8%
Fluid attenuated inversion recovery (FLAIR) MRI	9%
Thin slice non-contrast enhanced CT brain scan	2%
Diffusion weighted imaging (DWI) MRI	79%
Formal cerebral digital subtraction angiogram (DSA)	2%

DWI-MRI is the most sensitive and specific imaging modality for diagnosing acute stroke.

FLAIR MRI is sensitive for chronic ischaemic changes.

Both CTA and DSA may identify significant intravascular stenoses or thrombosis but are insensitive for small areas of acute ischaemic change.

Thin slice CT scans may identify established ischaemia with more sensitivity than a standard CT scan but is still inferior to DWI MRI.

In general, the purpose of the initial CT scan in accident and emergency for suspected ischaemic stroke patients is to exclude a haemorrhage.

Next question >

Stroke: assessment ★

Whilst the diagnosis of stroke may sometimes be obvious in many cases the presenting symptoms may be vague and accurate assessment difficult.

The FAST screening tool (Face/Arms/Speech/Time) is widely known by the general public following a publicity campaign. It has a positive predictive value of 78%.

A variant of FAST called the ROSIER score is useful for medical professionals. It is validated tool recommended by the Royal College of Physicians.

ROSIER score

Exclude hypoglycaemia first, then assess the following:

Assessment	**Scoring**
Loss of consciousness or syncope	- 1 point
Seizure activity	- 1 point
New, acute onset of:	
• asymmetric facial weakness	+ 1 point
• asymmetric arm weakness	+ 1 point
• asymmetric leg weakness	+ 1 point
• speech disturbance	+ 1 point
• visual field defect	+ 1 point

A stroke is likely if > 0.

Investigations

A non-contrast CT head scan is the first line radiological investigation for suspected stroke

- one of the key questions to answer is whether there is an ischaemic stroke or haemorrhagic stroke. Rarely a third pathology such as a tumour may also be detected
- this is especially important given the increasing role of thrombolysis and thrombectomy in acute stroke management
- acute ischaemic strokes
    - may show areas of low density in the grey and white matter of the territory. These changes may take time to develop
    - other signs include the 'hyperdense artery' sign corresponding with the responsible arterial clot - this tends to visible immediately
- acute haemorrhagic strokes
    - typically show areas of hyperdense material (blood) surrounded by low density (oedema)

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 3 👎 4

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Clinical Knowledge Summaries 👍 6 👎 3

Stroke and TIA guidelines

Suggest link

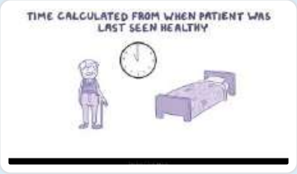
Report broken link

Media

Radiopaedia
Acute Ischaemic Stroke

GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 3 👎 2


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 1 👎 1

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 1 👎 1

Report broken media

Score: **48%**

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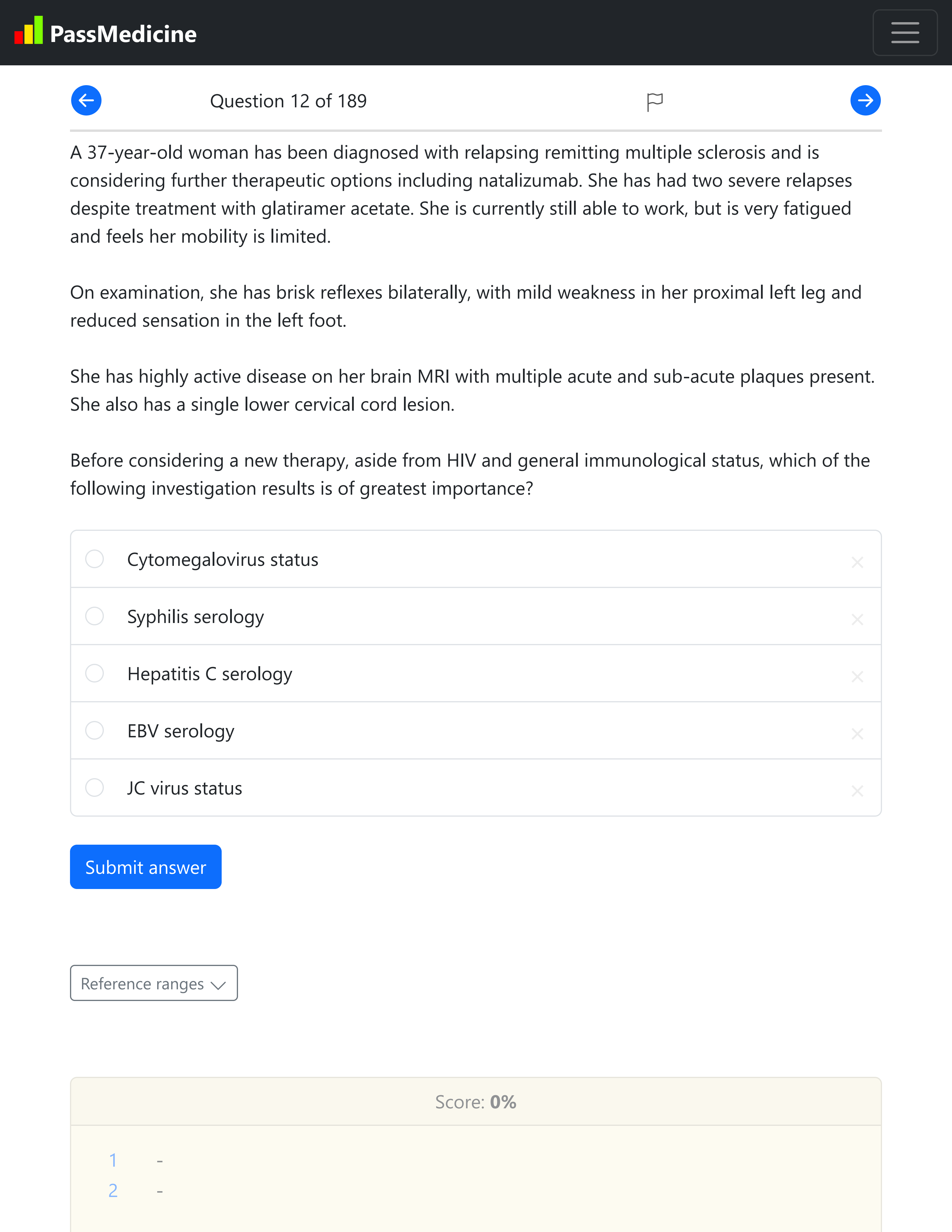

Question 12 of 189

A 37-year-old woman has been diagnosed with relapsing remitting multiple sclerosis and is considering further therapeutic options including natalizumab. She has had two severe relapses despite treatment with glatiramer acetate. She is currently still able to work, but is very fatigued and feels her mobility is limited.

On examination, she has brisk reflexes bilaterally, with mild weakness in her proximal left leg and reduced sensation in the left foot.

She has highly active disease on her brain MRI with multiple acute and sub-acute plaques present. She also has a single lower cervical cord lesion.

Before considering a new therapy, aside from HIV and general immunological status, which of the following investigation results is of greatest importance?

- ○ Cytomegalovirus status
- ○ Syphilis serology
- ○ Hepatitis C serology
- ○ EBV serology
- ○ JC virus status

Score: **0%**

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She has highly active disease on her brain MRI with multiple acute and sub-acute plaques present. She also has a single lower cervical cord lesion.

Before considering a new therapy, aside from HIV and general immunological status, which of the following investigation results is of greatest importance?

Cytomegalovirus status	10%
Syphilis serology	3%
Hepatitis C serology	6%
EBV serology	2%
JC virus status	79%

Natalizumab is a therapy approved for the management of highly active relapsing-remitting multiple sclerosis (RRMS) and is given as a once-monthly infusion. Natalizumab has been shown to greatly reduce relapse rates in RRMS.

It is a humanised monoclonal antibody against the cell adhesion molecule (CAM) 4-integrin. Natalizumab prevents leukocyte adhesion to endothelial VCAM1, thus preventing the migration of immune cells into the CNS.

An extremely rare, severe and almost universally fatal adverse side effect of Natalizumab is progressive multifocal leukoencephalopathy (PML). Only patients who are seropositive for JC virus are at risk of developing PML with exposure to natalizumab, and therefore checking the JC virus status of patients who are considering natalizumab therapy is mandatory.

It is also worth noting that some patients with low titres of JC virus are still commenced on natalizumab under close monitoring and that duration of natalizumab therapy is proportional to the risk of developing PML, i.e..short duration therapy (1-2 years) carries a very low risk of developing PML, even in those with a low positive JC virus titre. Ultimately, patients who are JC virus positive should not receive natalizumab and another therapy should be considered.

👍 👎 Discuss (2) Improve

Next question ›

Multiple sclerosis: management ★

Treatment in multiple sclerosis is focused on reducing the frequency and duration of relapses. There is no cure.

Acute relapse

High-dose steroids (e.g. oral or IV methylprednisolone) may be given for 5 days to shorten the length of an acute relapse. It should be noted that steroids shorten the duration of a relapse and do not alter the degree of recovery (i.e. whether a patient returns to baseline function)

Disease modifying drugs

A number of drugs have been shown to reduce the risk of relapse in patients with MS. Typical indications for disease-modifying drugs include:

- relapsing-remitting disease + 2 relapses in past 2 years + able to walk 100m unaided
- secondary progressive disease + 2 relapses in past 2 years + able to walk 10m (aided or unaided)

Drug options for reducing the risk of relapse in MS

- natalizumab
  - a recombinant monoclonal antibody that antagonises alpha-4 beta-1-integrin found on the surface of leucocytes
  - inhibit migration of leucocytes across the endothelium across the blood-brain barrier
  - generally considered to have the strongest evidence base for preventing relapse of the disease-modifying and hence is often used first-line
  - given intravenously
- ocrelizumab
  - humanized anti-CD20 monoclonal antibody

  - like natalizumab, it is considered a high-efficacy drug that is often used first-line
  - given intravenously
- fingolimod
  - sphingosine 1-phosphate (S1P) receptor modulator
  - prevents lymphocytes from leaving lymph nodes
  - oral formulations are available
- beta-interferon
  - not considered to be as effective as alternative disease-modifying drugs
  - given subcutaneously/intramuscularly
- glatiramer acetate
  - immunomodulating drug - acts as an 'immune decoy'
  - given subcutaneously
  - along with beta-interferon considered an 'older drug' with less effectiveness compared to monoclonal antibodies and S1P) receptor modulators

Some specific problems

Fatigue

- once other problems (e.g. anaemia, thyroid or depression) have been excluded NICE recommend a trial of amantadine
- other options include mindfulness training and CBT

Spasticity

- baclofen and gabapentin are first-line. Other options include diazepam, dantrolene and tizanidine
- physiotherapy is important
- cannabis and botox are undergoing evaluation

Bladder dysfunction

- may take the form of urgency, incontinence, overflow etc
- guidelines stress the importance of getting an ultrasound first to assess bladder emptying - anticholinergics may worsen symptoms in some patients
- if significant residual volume → intermittent self-catheterisation
- if no significant residual volume → anticholinergics may improve urinary frequency

Oscillopsia (visual fields appear to oscillate)

- gabapentin is first-line

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Multiple Sclerosis Society 👍 6 👎 15

Primary care guidelines

NICE 👍 15 👎 7

2014 Multiple Sclerosis guidelines

Suggest link Report broken link

Media

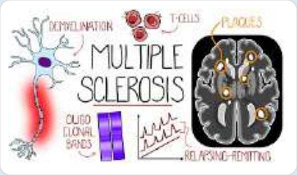
Multiple sclerosis

Podmedics - YouTube 👍 4 👎 0

Multiple Sclerosis
Cytokines

Multiple sclerosis

Soton Brain Hub - YouTube 👍 3 👎 0


DEMYELINATION
T-CELLS
PLAQUES
MULTIPLE SCLEROSIS
OLIGO CLONAL BANDS
RELAPSING-REMITTING

[Multiple sclerosis]

Rhesus Medicine - YouTube 👍 1 👎 0

MULTIPLE SCLEROSIS

[Multiple sclerosis - causes, symptoms, diagnosis, treatment, pathology]

Osmosis - YouTube 👍 7 👎 1

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Score: **48%**

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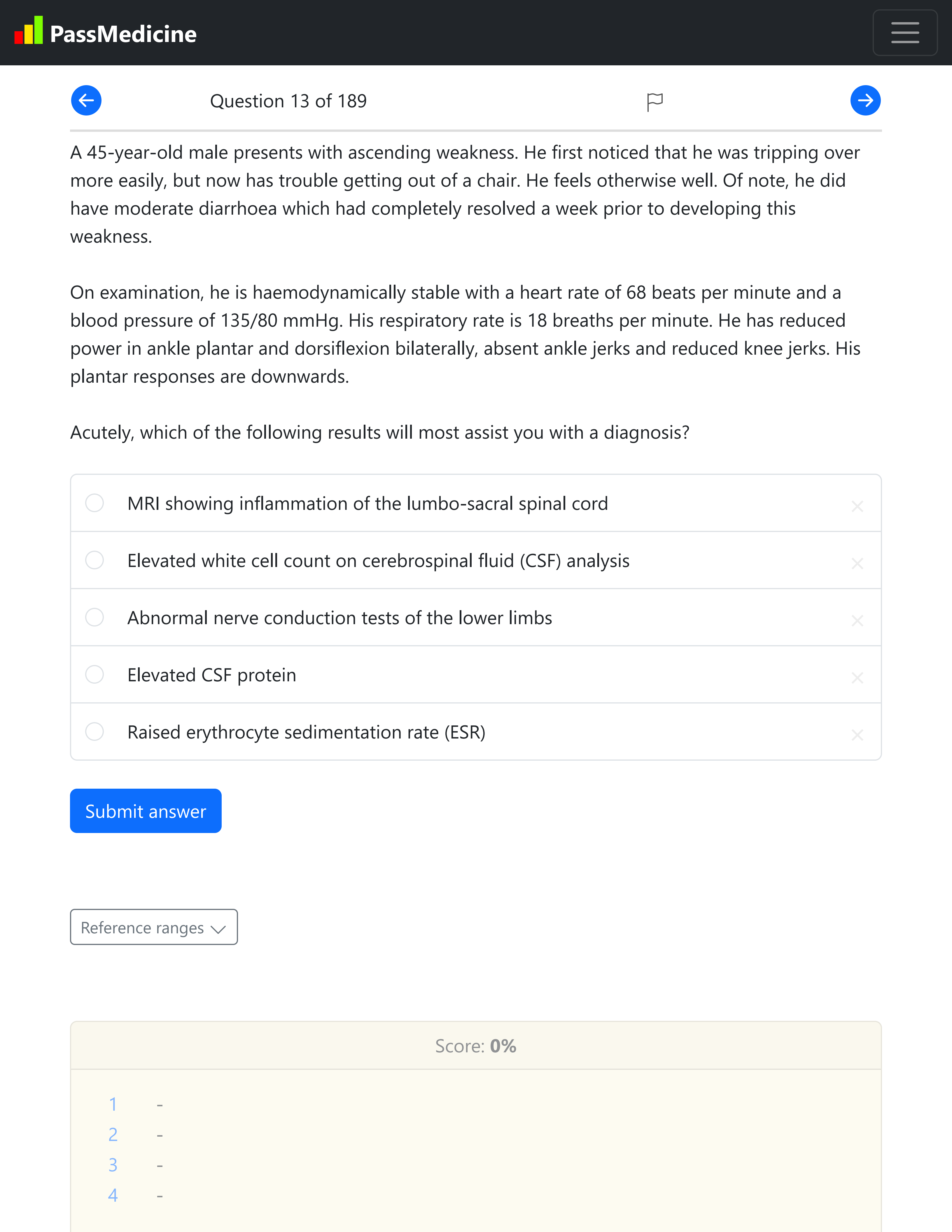

A 45-year-old male presents with ascending weakness. He first noticed that he was tripping over more easily, but now has trouble getting out of a chair. He feels otherwise well. Of note, he did have moderate diarrhoea which had completely resolved a week prior to developing this weakness.

On examination, he is haemodynamically stable with a heart rate of 68 beats per minute and a blood pressure of 135/80 mmHg. His respiratory rate is 18 breaths per minute. He has reduced power in ankle plantar and dorsiflexion bilaterally, absent ankle jerks and reduced knee jerks. His plantar responses are downwards.

Acutely, which of the following results will most assist you with a diagnosis?

- ○ MRI showing inflammation of the lumbo-sacral spinal cord ×
- ○ Elevated white cell count on cerebrospinal fluid (CSF) analysis ×
- ○ Abnormal nerve conduction tests of the lower limbs ×
- ○ Elevated CSF protein ×
- ○ Raised erythrocyte sedimentation rate (ESR) ×

Score: **0%**

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Acutely, which of the following results will most assist you with a diagnosis?

MRI showing inflammation of the lumbo-sacral spinal cord	4%
Elevated white cell count on cerebrospinal fluid (CSF) analysis	5%
Abnormal nerve conduction tests of the lower limbs	35%
Elevated CSF protein	55%
Raised erythrocyte sedimentation rate (ESR)	1%

Acutely, an elevated CSF protein level may be the only indication of an inflammatory aetiology, highly suggestive of Guillain-Barre Syndrome (GBS) or Acute Inflammatory Demyelinating Polyradiculoneuropathy in this clinical setting. The CSF white cell count is not elevated in GBS.

In relation to nerve conduction studies, these often provide normal results within the acute setting and the classic demyelination findings may not be detectable for at least a week.

Discuss (10) | Improve

Next question

Guillain-Barre syndrome: features

Guillain-Barre syndrome describes an immune-mediated demyelination of the peripheral nervous system often triggered by an infection (classically *Campylobacter jejuni*).

Initial symptoms

- around 65% of patients experience back/leg pain in the initial stages of the illness

The characteristic features of Guillain-Barre syndrome is progressive, symmetrical weakness of all the limbs.

- the weakness is classically **ascending** i.e. the legs are affected first
- reflexes are reduced or absent
- sensory symptoms tend to be mild (e.g. distal paraesthesia) with very few sensory signs

Other features

- there may be a history of gastroenteritis
- respiratory muscle weakness
- cranial nerve involvement
  - diplopia
  - bilateral facial nerve palsy
  - oropharyngeal weakness is common
- autonomic involvement
  - urinary retention
  - diarrhoea

Less common findings

- papilloedema: thought to be secondary to reduced CSF resorption

Investigations

- lumbar puncture
  - rise in protein with a normal white blood cell count (albuminocytologic dissociation) - found in 66%
- nerve conduction studies may be performed
  - decreased motor nerve conduction velocity (due to demyelination)
  - prolonged distal motor latency
  - increased F wave latency

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Medcomic 👍 10 👎 8

[Guillain-Barre syndrome]

Patient.info 👍 6 👎 5

Guillain-Barre syndrome review

Suggest link

Report broken link

Media

SYMPTOMS - BASED on NERVES AFFECTED

Guillain-Barre syndrome

Osmosis - YouTube 👍 1 👎 0

NERVE CONDUCTION STUDIES
PERIPHERAL NERVES
GUILLAIN-BARRÉ SYNDROME
WEAKNESS
NEUROPATHY
B-CELLS
ANTIBODIES
CAMPYLOBACTER

Understanding Guillain-Barre Syndrome

Zero To Finals - YouTube 👍 1 👎 0

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Score: **48%**

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Question 14 of 189

A 65-year-old male with a 60 pack year smoking history has been diagnosed with small cell lung cancer. He is currently undergoing chemotherapy. Over the last few months, he has noticed his vision deteriorating and complains of diplopia. He also feels weaker in his upper limbs although his symptoms do fluctuate.

On examination, he has mild ptosis of the eyelids bilaterally and a complex ophthalmoparesis affecting both eyes. He also has reduced power proximally in the upper limbs.

In this clinical syndrome, in addition to electrophysiological studies, which of the following blood test results is likely to assist with the diagnosis?

- ○ Presence of anti-muscle specific kinase antibodies
- ○ Presence of anti-neuronal antibodies
- ○ Presence of voltage gated potassium channel antibodies
- ○ Presence of sodium channel antibodies
- ○ Presence of voltage gated calcium channel antibodies

Submit answer

Reference ranges

Score: **0%**

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Question 14 of 189 ✓

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In this clinical syndrome, in addition to electrophysiological studies, which of the following blood test results is likely to assist with the diagnosis?

Presence of anti-muscle specific kinase antibodies	10%
Presence of anti-neuronal antibodies	2%
Presence of voltage gated potassium channel antibodies	11%
Presence of sodium channel antibodies	1%
Presence of voltage gated calcium channel antibodies	76%

The paraneoplastic myasthenic syndrome described above associated with small-cell lung cancer is Lambert-Eaton myasthenic syndrome (LEMS).

Antibodies to voltage-gated calcium channels (VGCCs) have been reported in 75-100% of LEMS patients who have small cell lung cancer (SCLC) and in 50-90% of LEMS patients who do not have underlying cancer.

Discuss (5) | Improve

Next question ›

Paraneoplastic neurological syndromes ★

Lambert-Eaton myasthenic syndrome

- seen in small cell lung cancer
- antibody directed against voltage gated calcium channel
- similar features to myasthenia gravis
- proximal muscle weakness, hyporeflexia and autonomic features (dry mouth, impotence etc)

Other syndromes may be associated with detectable antibodies, for example:
- anti-Hu: small cell lung cancer - painful sensory neuropathy, cerebellar syndromes, encephalitis
- anti-Yo: ovarian, breast cancer - cerebellar syndrome
- anti-Ri: small cell lung cancer - retinal degeneration

Next question >

Textbooks

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Links

Mayo Clinic 👍 0 👎 0

Paraneoplastic Syndromes: An Approach to Diagnosis and Treatment

Suggest link

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Score: **48%**

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Question 15 of 189

A 75-year-old female on aspirin for known ischaemic heart disease is being investigated for the cause of a recent transient ischaemic attack (TIA) which caused brief left sided weakness. She does not have a history of atrial fibrillation, nor has any been detected on routine telemetry.

Her MRI brain scan does not provide any evidence for acute stroke and her transthoracic echocardiogram does not demonstrate any intra-cardiac thrombus.

A carotid ultrasound study revealed a 75-85% stenosis of the right internal carotid artery. In addition to ordering a CT carotid angiogram to further investigate the lesion, the next best step in her management would be to?

- ○ Add clopidogrel
- ○ Commence best medical management and re-image in 6 months
- ○ Commence an unfractionated heparin infusion
- ○ Commence best medical therapy and refer for a carotid endarterectomy in 6-8 weeks
- ○ Commence best medical therapy and refer for a carotid endarterectomy within 14 days

Submit answer

Reference ranges

Score: **0%**

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Her MRI brain scan does not provide any evidence for acute stroke and her transthoracic echocardiogram does not demonstrate any intra-cardiac thrombus.

A carotid ultrasound study revealed a 75-85% stenosis of the right internal carotid artery. In addition to ordering a CT carotid angiogram to further investigate the lesion, the next best step in her management would be to?

Add clopidogrel	7%
Commence best medical management and re-image in 6 months	3%
Commence an unfractionated heparin infusion	1%
Commence best medical therapy and refer for a carotid endarterectomy in 6-8 weeks	25%
Commence best medical therapy and refer for a carotid endarterectomy within 14 days	64%

Patients with a TIA or small ischaemic stroke with evidence of significant carotid artery stenosis (male patients with a carotid artery stenosis of 50-99% or female patients with a carotid artery stenosis of 70-99%) that corresponds to the same side as the stroke/TIA should be considered for an urgent carotid endarterectomy (CEA) within 14 days.

Discuss (6) Improve

Next question >

Stroke: management of acute ischaemic stroke

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
    - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
    - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:

- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.

- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
    - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
    - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.

- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6

hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

123

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Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 15

Stroke and TIA guidelines

Age and Ageing 👍 20 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

2023 National Clinical Guideline for Stroke

NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

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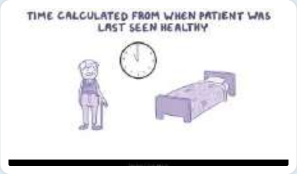
TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

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Radiopaedia
Acute Ischaemic Stroke

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Management of ischaemic stroke

Stroke

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Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

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Question 16 of 189

An 81-year-old lady with a history of congestive cardiac failure was assessed in accident and emergency and clinically diagnosed with an acute ischaemic stroke. Her main deficits were slurred speech and left sided facial droop with some loss of fine motor control in the left hand. On admission, her blood pressure was 185/70mmHg with a heart rate of 95 beats per minute in sinus rhythm.

The initial CT scan of her brain showed some evidence of chronic small vessel ischaemia, but no acute pathology, in particular no haemorrhage was seen. Which of the following combinations of investigations should be carried out during the acute admission?

- ○ MRI, transthoracic echocardiogram (TTE), cardiac telemetry and carotid duplex study ×
- ○ Transoesophageal echocardiogram (TOE), CT cerebral angiogram and carotid duplex study ×
- ○ CT scan, fasting lipids and a video fluoroscopy swallow study ×
- ○ Fasting glucose, Electroencephalogram (EEG), MRI and thrombophilia screen ×
- ○ Thrombophilia screen, MRI and a TTE ×

Submit answer

Reference ranges

Score: **0%**

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- MRI, transthoracic echocardiogram (TTE), cardiac telemetry and carotid duplex study 73%
- Transoesophageal echocardiogram (TOE), CT cerebral angiogram and carotid duplex study 18%
- CT scan, fasting lipids and a video fluoroscopy swallow study 3%
- Fasting glucose, Electroencephalogram (EEG), MRI and thrombophilia screen 2%
- Thrombophilia screen, MRI and a TTE 4%

After a diagnosis of ischaemic stroke is suspected based on clinical assessment and the absence of haemorrhage on CT scan, a number of further investigations should be requested in order to confirm the diagnosis and determine the underlying aetiology of the stroke.

- MRI is the most sensitive and specific modality for identifying an acute ischaemic stroke using diffusion-weighted imaging (DWI). If MRI is unavailable or a patient is ineligible to undergo an MRI, a serial CT scan may show interval changes consistent with an evolving ischaemic stroke.
- The remaining investigations should serve to localise the focus of thrombus or atheroma that has caused the stroke, but importantly, only investigations that will lead to a change in management are necessary. These include a carotid duplex study and/or CT carotid angiogram, a transthoracic echocardiogram (TTE) and cardiac telemetry.
- The carotid studies may identify a relevant significant stenosis necessitating carotid endarterectomy

- The TTE may identify an intraventricular thrombus, valvular pathology or mass that is the cause for thromboembolic stroke necessitating further investigation or the initiation of anticoagulation

- The cardiac telemetry may identify atrial fibrillation requiring consideration of anticoagulation.
- In addition, the above investigations fasting lipids should be measured as well as fasting glucose and or HbA1c in TIA/stroke patients.

A thrombophilia screen or TOE are not required routinely but may be requested in certain patients. A CT cerebral angiogram is needed in some circumstances, but not currently required for the routine investigation of ischaemic stroke.

👍 | 👎 | 💬 Discuss (1) | Improve

Next question >

Stroke: management of acute ischaemic stroke ★

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
    - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
    - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:
- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.
- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
  - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
  - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

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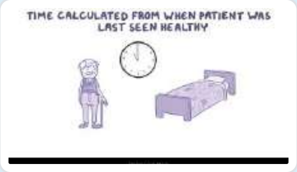
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Stroke

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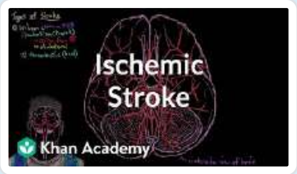
Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

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A 42-year-old man presented with a partial left Horner's syndrome (ptosis with miosis, no anhidrosis), ipsilateral neck pain and reduced visual acuity on the same side.

He reported that the first symptom was neck pain and that his vision worsened over the next two hours. On admission, his blood pressure was 145/80 mmHg and heart rate was 80 beats per minute in sinus rhythm. His chest was clear.

His initial non-contrast enhanced CT brain scan showed a very small area of gliosis in the left frontal area possibly related to a distant injury, but no acute pathology.

Which of the following tests is likely to give you a diagnosis?

○ MRI of the brain ×

○ CT carotid angiogram ×

○ Chest X-Ray ×

○ Erythrocyte sedimentation rate (ESR) and temporal artery biopsy ×

○ CT venogram of the neck and brain ×

Submit answer

Reference ranges

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His initial non-contrast enhanced CT brain scan showed a very small area of gliosis in the left frontal area possibly related to a distant injury, but no acute pathology.

Which of the following tests is likely to give you a diagnosis?

MRI of the brain	7%
CT carotid angiogram	67%
Chest X-Ray	7%
Erythrocyte sedimentation rate (ESR) and temporal artery biopsy	3%
CT venogram of the neck and brain	15%

The oculosympathetic pathway leads a course from the posterior hypothalamus, through the brainstem followed by the spinal cord, over the apex of the lung to the superior cervical ganglion, and then finally ascends along with the internal carotid artery to ultimately reach the pupillary dilator muscle and Mueller's muscle of the lid. These post ganglionic fibres within the carotid sheath are vulnerable to the compressive effects of a mural haematoma or pseudoaneurysm resulting from a carotid artery dissection. Such compression can result in a partial Horner's syndrome.

In addition to this, patients with both carotid and vertebral artery dissections often present with neck pain.

Finally, in patients with a carotid dissection retinal ischaemia is often as a result of thrombi produced within the dissected portion of the vessel that then embolize distally towards the brain or in this case, the ophthalmic artery, a branch of the internal carotid artery.

The investigation of choice to diagnose a carotid dissection is a CT carotid angiogram, however, other such as MR angiograms of the neck are also adequate.

Discuss (4) | Improve

Next question >

Horner's syndrome

Features
- miosis (small pupil)
- ptosis
- enophthalmos* (sunken eye)
- anhidrosis (loss of sweating one side)

Distinguishing between causes
- heterochromia (difference in iris colour) is seen in congenital Horner's
- anhidrosis: see below
- pharmacologic tests
  - can be useful to confirm the diagnosis of Horner syndrome and localise the lesion
  - apraclonidine drops (an alpha-adrenergic agonist) can be used: causes pupillary dilation in Horner's syndrome due to denervation supersensitivity but produces mild pupillary constriction in the normal pupil by down-regulating the norepinephrine release at the synaptic cleft

Central lesions	Pre-ganglionic lesions	Post-ganglionic lesions
Anhidrosis of the face, arm and trunk	Anhidrosis of the face	No anhidrosis
Stroke **S**yringomyelia Multiple **s**clerosis	Pancoast's **t**umour **T**hyroidectomy **T**rauma Cervical rib	**C**arotid artery dissection **C**arotid aneurysm **C**avernous sinus thrombosis **C**luster headache

Central lesions	Pre-ganglionic lesions	Post-ganglionic lesions
Tumour Encephalitis		

*in reality the appearance is due to a narrow palpebral aperture rather than true enophthalmos

123

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HORNER'S SYNDROME

Horner's syndrome

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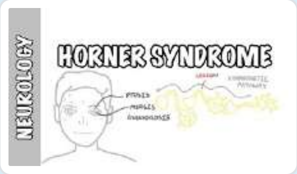
NEUROLOGY
HORNER SYNDROME

Horner's syndrome

Armando Hasudungan - YouTube

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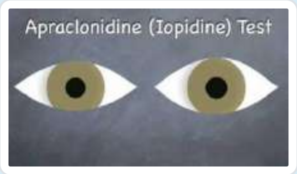
Apraclonidine (Iopidine) Test

[How to Examine Horner's Syndrome]

Individual - YouTube 👍 3 👎 0

[Pancoast tumors]

Osmosis - YouTube 👍 1 👎 0

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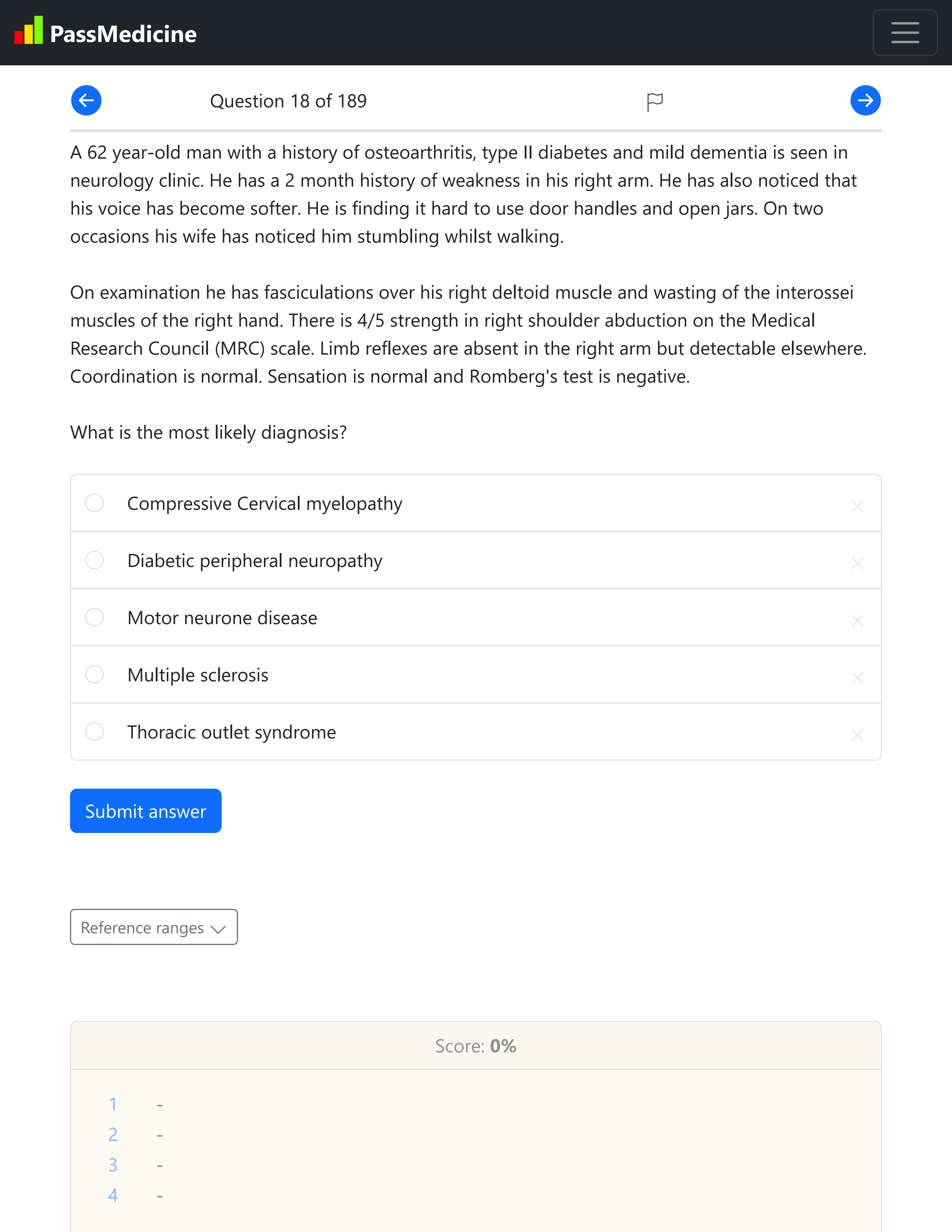

A 62 year-old man with a history of osteoarthritis, type II diabetes and mild dementia is seen in neurology clinic. He has a 2 month history of weakness in his right arm. He has also noticed that his voice has become softer. He is finding it hard to use door handles and open jars. On two occasions his wife has noticed him stumbling whilst walking.

On examination he has fasciculations over his right deltoid muscle and wasting of the interossei muscles of the right hand. There is 4/5 strength in right shoulder abduction on the Medical Research Council (MRC) scale. Limb reflexes are absent in the right arm but detectable elsewhere. Coordination is normal. Sensation is normal and Romberg's test is negative.

What is the most likely diagnosis?

- ○ Compressive Cervical myelopathy
- ○ Diabetic peripheral neuropathy
- ○ Motor neurone disease
- ○ Multiple sclerosis
- ○ Thoracic outlet syndrome

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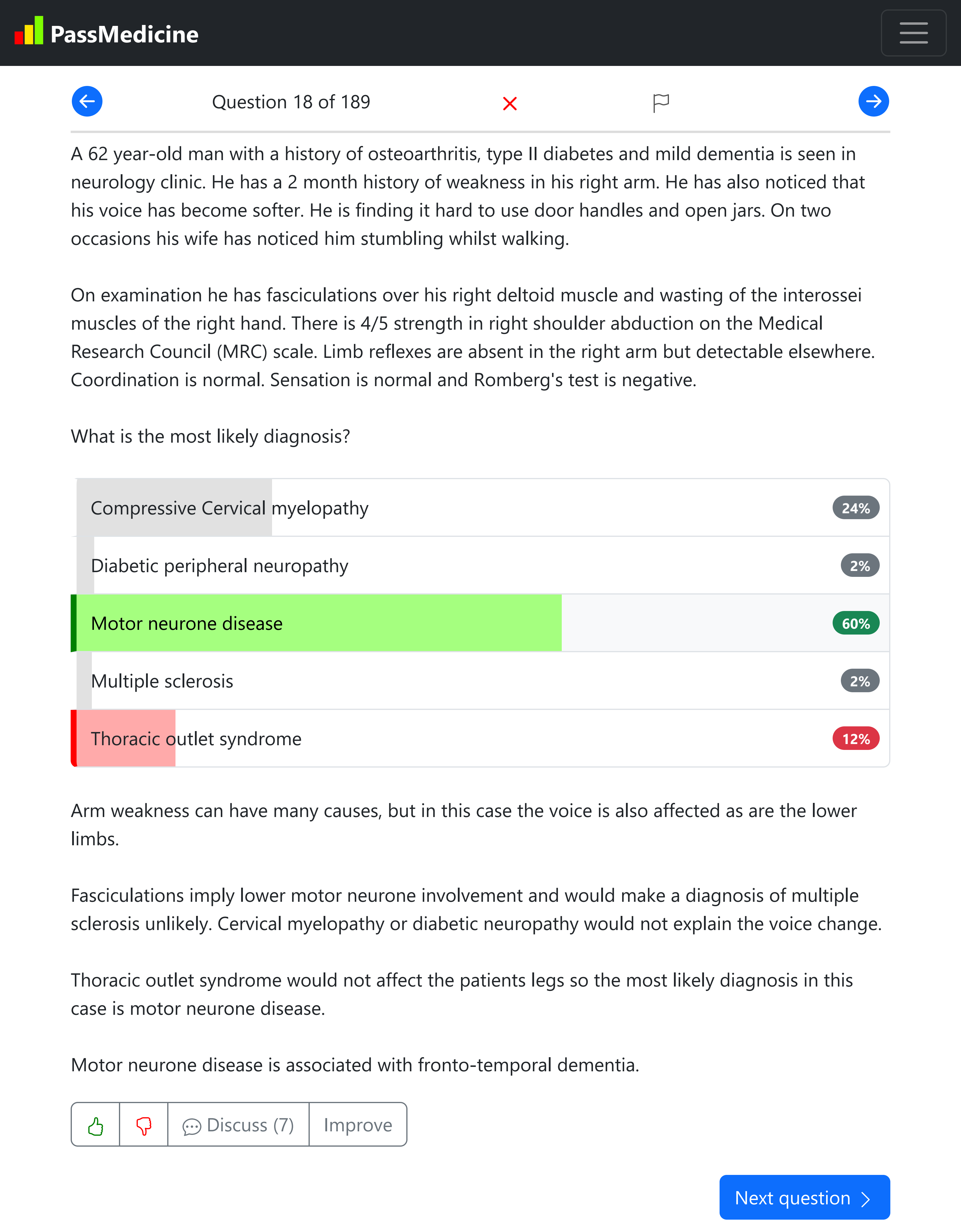

Question 18 of 189 ✗

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What is the most likely diagnosis?

Compressive Cervical myelopathy	24%
Diabetic peripheral neuropathy	2%
🟩 Motor neurone disease	60%
Multiple sclerosis	2%
🟥 Thoracic outlet syndrome	12%

Arm weakness can have many causes, but in this case the voice is also affected as are the lower limbs.

Fasciculations imply lower motor neurone involvement and would make a diagnosis of multiple sclerosis unlikely. Cervical myelopathy or diabetic neuropathy would not explain the voice change.

Thoracic outlet syndrome would not affect the patients legs so the most likely diagnosis in this case is motor neurone disease.

Motor neurone disease is associated with fronto-temporal dementia.

Motor neuron disease: features ★

Motor neuron disease is a neurological condition of unknown cause which can present with both upper and lower motor neuron signs. It rarely presents before 40 years and various patterns of disease are recognised including amyotrophic lateral sclerosis (ALS), progressive muscular atrophy and bulbar palsy.

There are a number of clues which point towards a diagnosis of motor neuron disease:
- asymmetric limb weakness is the most common presentation of ALS
- the mixture of lower motor neuron and upper motor neuron signs
- wasting of the small hand muscles/tibialis anterior is common
- fasciculations
- the absence of sensory signs/symptoms
  - vague sensory symptoms may occur early in the disease (e.g. limb pain) but 'never' sensory signs

Other features
- doesn't affect external ocular muscles
- no cerebellar signs
- abdominal reflexes are usually preserved and sphincter dysfunction if present is a late feature

The diagnosis of motor neuron disease is clinical, but nerve conduction studies will show normal motor conduction and can help exclude a neuropathy. Electromyography shows a reduced number of action potentials with increased amplitude. MRI is usually performed to exclude the differential diagnosis of cervical cord compression and myelopathy

+ Q 123

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Links

Postgraduate Medical Journal 👍 15 👎 12

Review of MND

Royal College of Physicians 👍 17 👎 10

Motor neuron disease: diagnostic pitfalls

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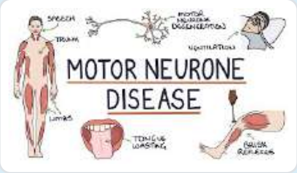
Media



Motor neuron disease

Zero To Finals - YouTube 👍 0 👎 0

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Score: **48%**

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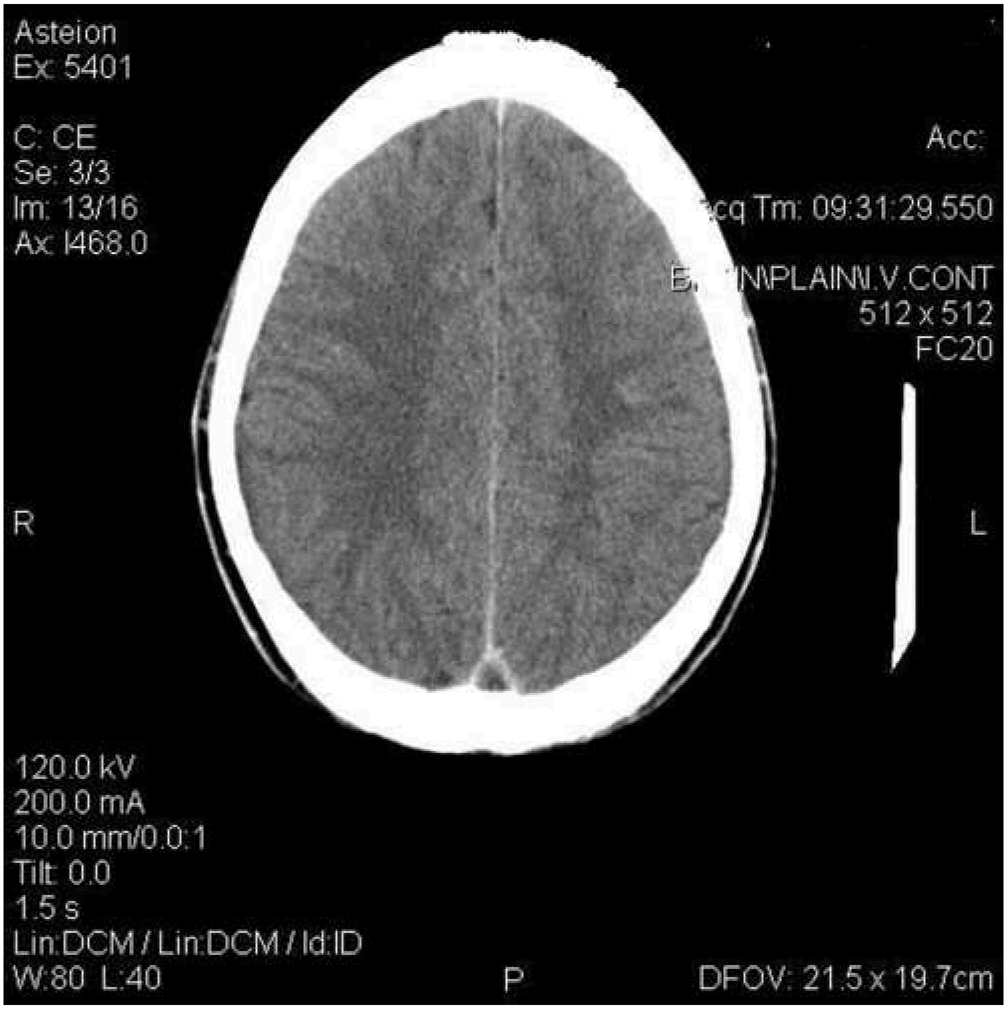
A 19-year-old female is admitted to the Emergency Department after suffering a seizure. Her friends report that she is normally fit and well but had been complaining of a bad headache for the past few hours.

On examination her GCS is 13/15 (M6 V4 E3). No focal neurological deficit is noted. She appears slightly confused and is holding her head in her hands. Her pulse rate is 90/min with a temperature of 37.2°. No neck stiffness is noted.

A CT scan with contrast is requested: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What is the most likely diagnosis?

○ Extradural haemorrhage

○ Superior sagittal sinus thrombosis ×

○ Herpes simplex encephalitis ×

○ Subrarachnoid haemorrhage ×

○ Meningioma ×

Submit answer

Reference ranges

Score: **0%**

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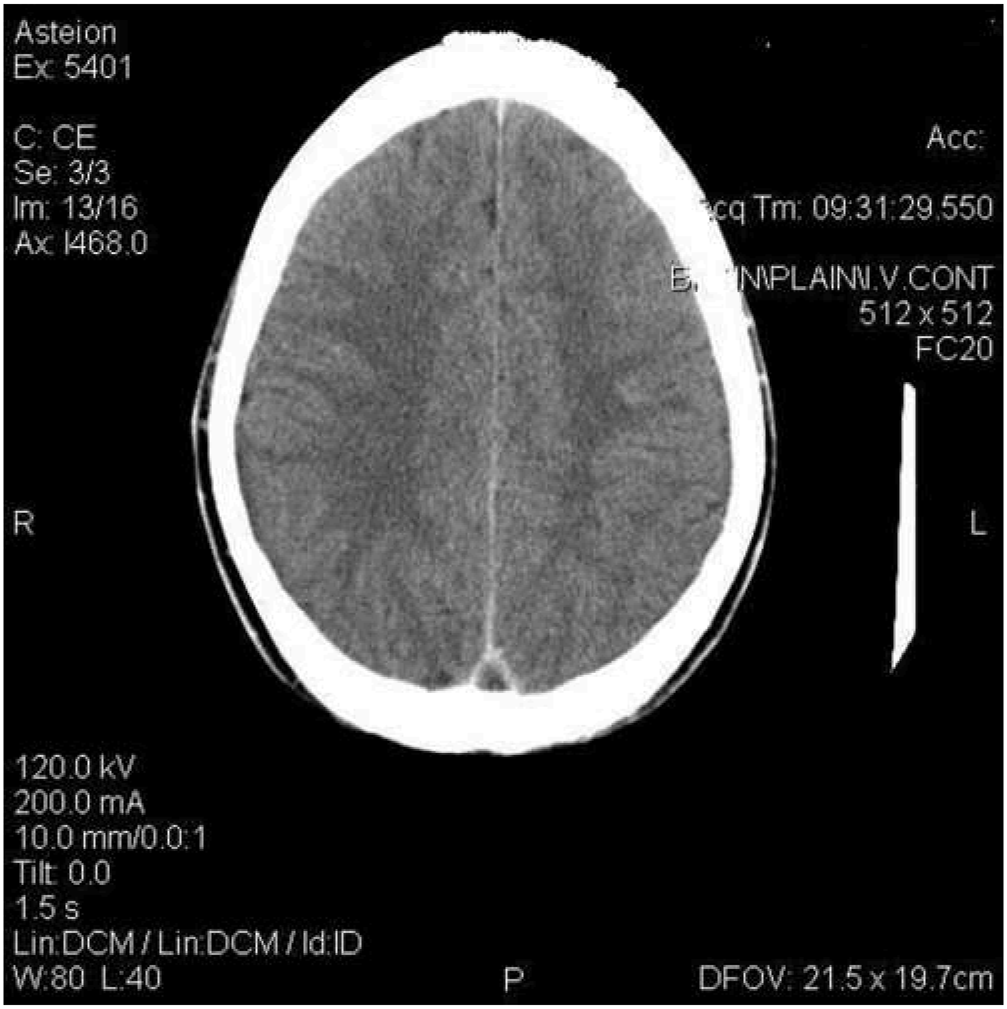
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DFOV: 21.5 x 19.7cm

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What is the most likely diagnosis?

Extradural haemorrhage 1%

Superior sagittal sinus thrombosis	74%
Herpes simplex encephalitis	7%
Subrarachnoid haemorrhage	16%
Meningioma	2%

The CT with contrast demonstates a superior sagittal sinus thrombosis showing the typical empty delta sign. Look at the 'bottom' of the scan for the triangular shaped dural sinus. This should normally be white due to it being filled with contrast. The empty delta sign occurs when the thrombus fails to enhance within the dural sinus and is outlined by enhanced collateral channels in the falx. This sign is seen in only about 25%-30% of cases but is highly diagnostic for sagittal sinus thrombosis.

👍 👎 Discuss (2) Improve

Next question ›

Intracranial venous thrombosis

Overview

- can cause cerebral infarction, much lesson common than arterial causes
- 50% of patients have isolated sagittal sinus thromboses - the remainder have coexistent lateral sinus thromboses and cavernous sinus thromboses

General features

Common features

- headache (may be sudden onset)
- nausea & vomiting
- reduced consciousness

Investigation

- MRI venography is the gold standard
    - CT venography is an alternative
- non-contrast CT head is normal in around 70% of patients
- D-dimer levels may be elevated

Management

- anticoagulation
    - typically with low molecular weight heparin acutely
    - warfarin is still generally used for longer term anticoagulation

Specific syndromes

Sagittal sinus thrombosis

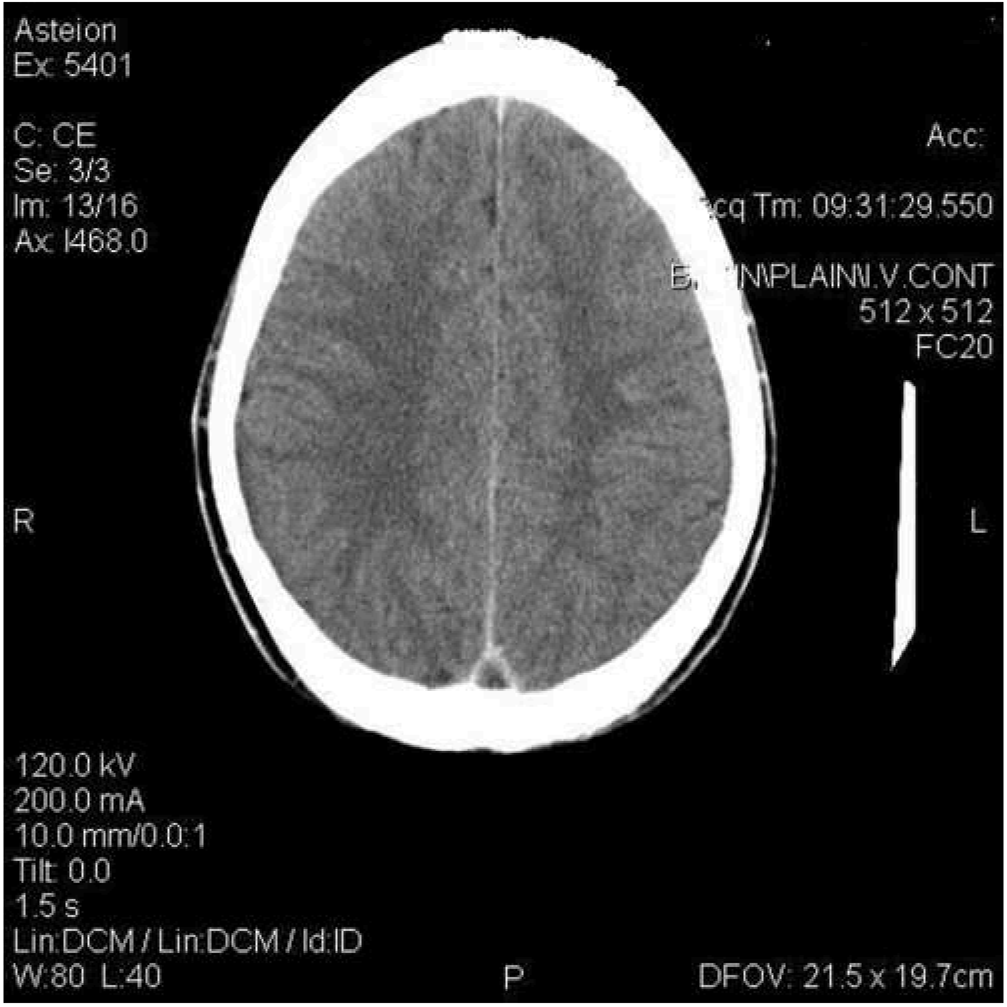
- may present with seizures and hemiplegia
- parasagittal biparietal or bifrontal haemorrhagic infarctions are sometimes seen
- 'empty delta sign' seen on venography

Cavernous sinus thrombosis

- other causes of cavernous sinus syndrome: local infection (e.g. sinusitis), neoplasia, trauma
- periorbital erythema and oedema
- ophthalmoplegia: 6th nerve damage typically occurs before 3rd & 4th
- trigeminal nerve involvement may lead to hyperaesthesia of upper face and eye pain
- central retinal vein thrombosis

Lateral sinus thrombosis

- 6th and 7th cranial nerve palsies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W:80 L:40
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DFOV: 21.5 x 19.7cm

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CT with contrast demonstrating a **superior sagittal sinus thrombosis** showing the typical empty delta sign. Look at the 'bottom' of the scan for the triangular shaped dural sinus. This should normally be white due to it being filled with contrast. The empty delta sign occurs when the thrombus fails to enhance within the dural sinus and is outlined by enhanced collateral channels in the falx. This sign is seen in only about 25%-30% of cases but is highly diagnostic for sagittal sinus thrombosis

+ Q 123

Next question >

Textbooks

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Score: **48%**

1	✗
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20	✓
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24	✓
25	✓

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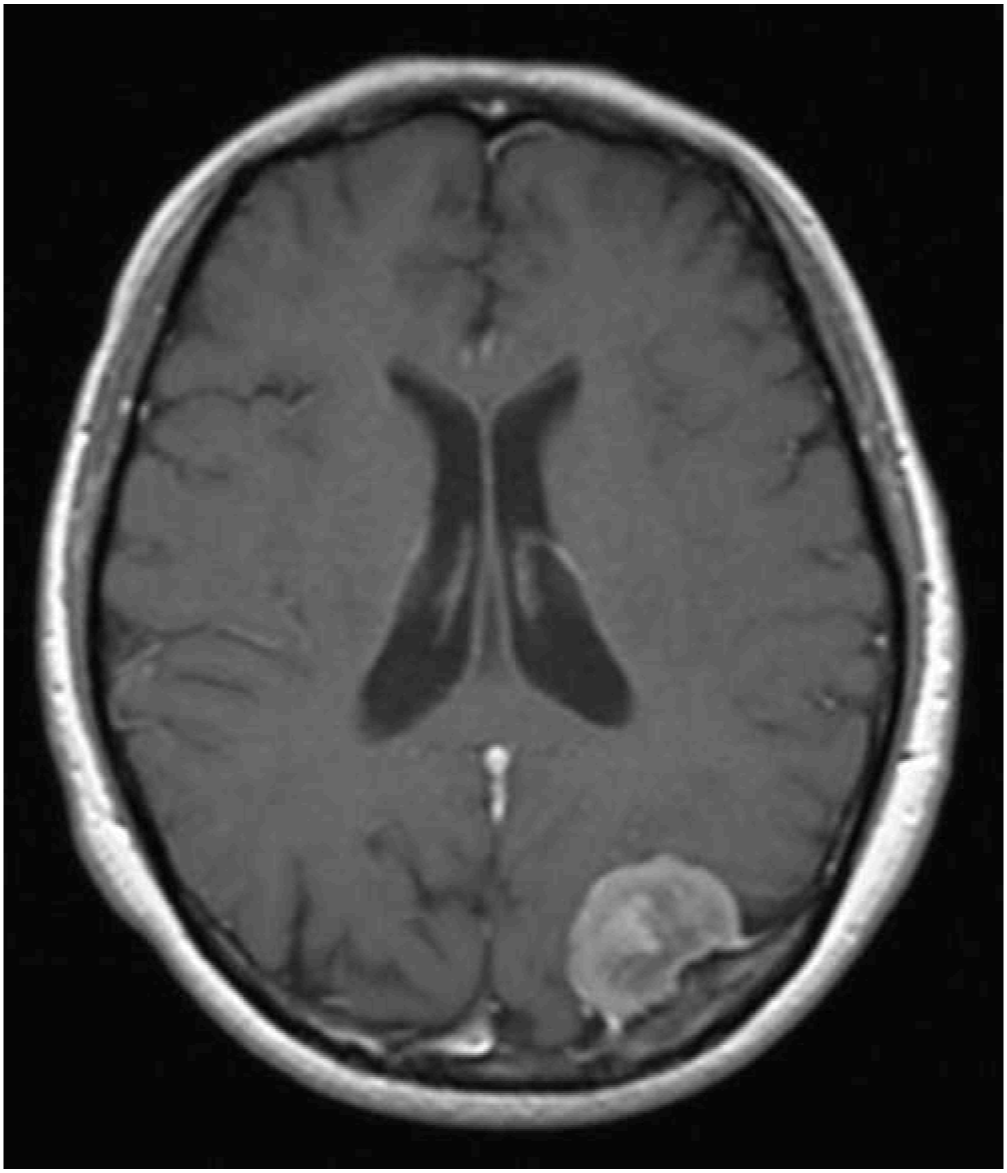
A 54-year-old woman is admitted to the Emergency Department after having a seizure. This occured whilst she was a passenger in a car. There is no history of trauma. By the time of her admission she is alert with a GCS of 15/15. Her partner describes a generalised seizure with a post-ictal phase lasting around 30 minutes.

On examination her heart rate is 90/min, blood pressure 102/60 mmHg and temperature 37.1ºC. There are no focal neurological signs.

A MRI scan is requested:



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What is the most likely diagnosis?

○ Extradural haematoma ×

○ Gliolastoma multiforme ×

○ Subdural haematoma ×

○ Meningioma ×

○ Oligodendroma ×

Submit answer

Reference ranges ⌄

Score: **0%**

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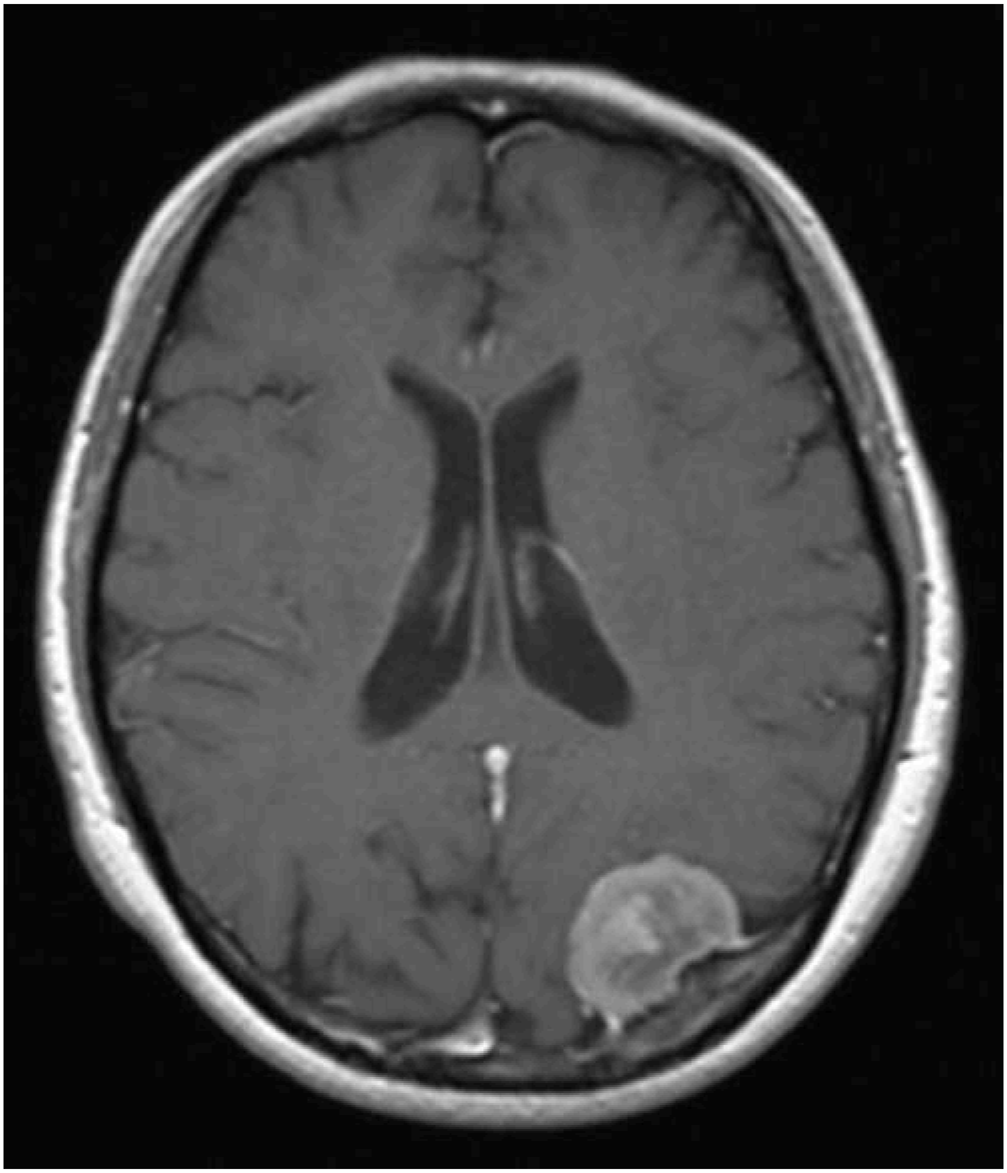

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What is the most likely diagnosis?

Extradural haematoma	1%
Gliolastoma multiforme	17%
Subdural haematoma	2%
Meningioma	77%
Oligodendroma	3%

The MRI shows the typical well-circumscribed appearance of a meningioma. A dural tail can be where the tumour 'connects' to the dura. It is seen in around 65% of meningiomas.

👍 👎 💬 Discuss Improve

Next question ›

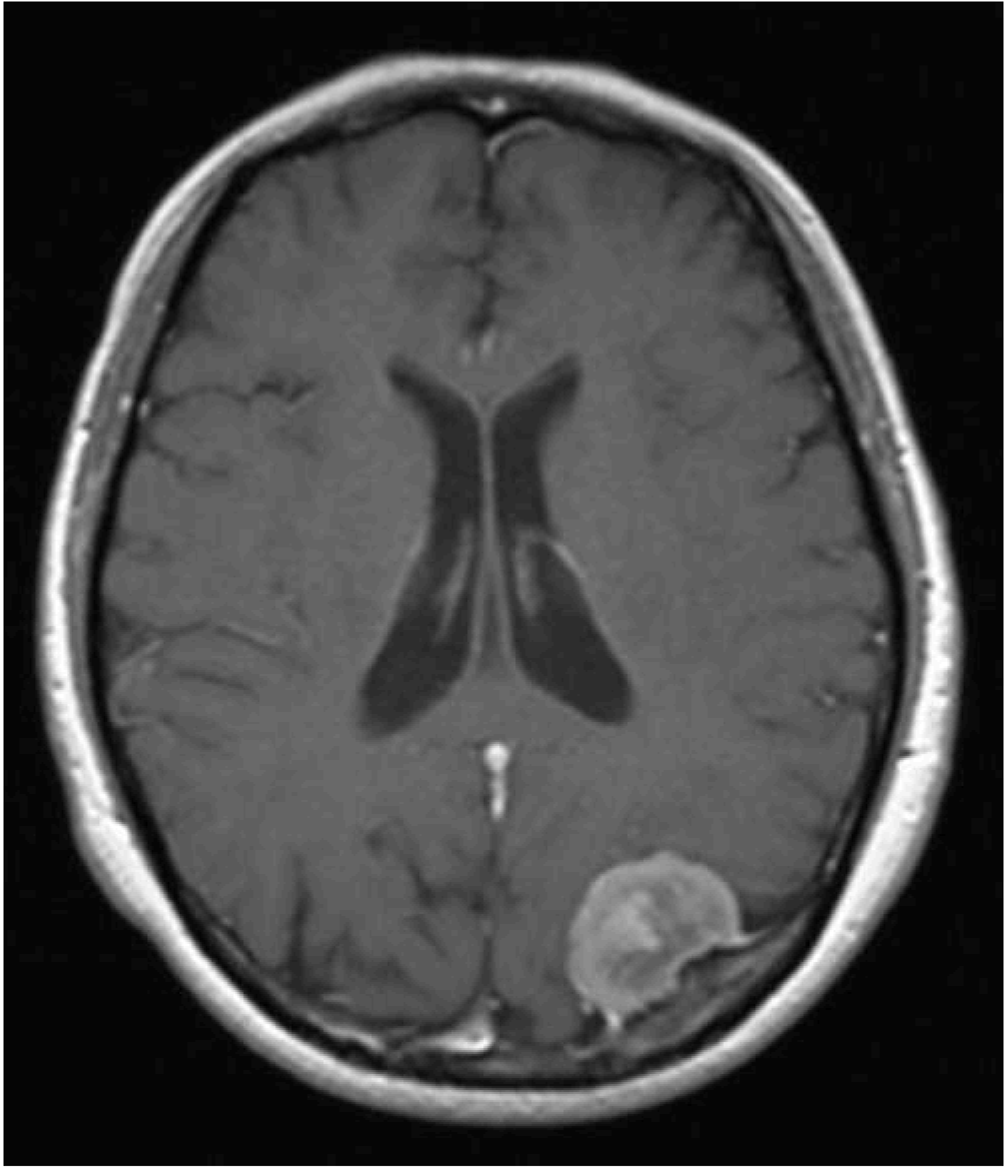
Brain tumours ★

The majority of adult tumours are supratentorial, where as the majority of childhood tumours are infratentorial.

Type of tumour	**Features**
Metastases	Metastatic brain cancer is the most common form of brain tumours. They are often multiple and not treatable with surgical intervention. Tumours that most commonly spread to the brain include: • lung (most common) • breast • bowel • skin (namely melanoma) • kidney
Glioblastoma multiforme	• Glioblastoma is the most common primary tumour in adults and is associated with a poor prognosis (~ 1yr). • On imaging they are solid tumours with central necrosis and a rim that enhances with contrast. Disruption of the blood-brain barrier and

Type of tumour	Features
	therefore are associated with vasogenic oedema. • Histology: Pleomorphic tumour cells border necrotic areas • Treatment is surgical with postoperative chemotherapy and/or radiotherapy. Dexamethasone is used to treat the oedema.
Meningioma	• The second most common primary brain tumour in adults • Meningiomas are typically benign, extrinsic tumours of the central nervous system. They arise from the arachnoid cap cells of the meninges and are typically located next to the dura and cause symptoms by compression rather than invasion. • They typically are located at the falx cerebri, superior sagittal sinus, convexity or skull base. • Histology: Spindle cells in concentric whorls and calcified psammoma bodies • Investigation is with CT (will show contrast enhancement) and MRI, and treatment will involve either observation, radiotherapy or surgical resection.
Vestibular schwannoma	• A vestibular schwannoma (previously termed acoustic neuroma) is a benign tumour arising from the eighth cranial nerve (vestibulocochlear nerve). Often seen in the cerebellopontine angle. It presents with hearing loss, facial nerve palsy (due to compression of the nearby facial nerve) and tinnitus. • Neurofibromatosis type 2 is associated with bilateral vestibular schwannomas. • Histology: Antoni A or B patterns are seen. Verocay bodies (acellular areas surrounded by nuclear palisades) • Treatment may involve observation, radiotherapy or surgery.
Pilocytic astrocytoma	• The most common primary brain tumour in children • Histology: Rosenthal fibres (corkscrew eosinophilic bundle)
Medulloblastoma	• A medulloblastoma is an aggressive paediatric brain tumour that arises within the infratentorial compartment. It spreads through the CSF

Type of tumour	Features
	system. Treatment is surgical resection and chemotherapy. • Histology: Small, blue cells. Rosette pattern of cells with many mitotic figures
Ependymoma	• Commonly seen in the 4th ventricle • May cause hydrocephalus • Histology: perivascular pseudorosettes
Oligodendroma	• Benign, slow-growing tumour common in the frontal lobes • Histology: Calcifications with 'fried-egg' appearance
Haemangioblastoma	• Vascular tumour of the cerebellum • Associated with von Hippel-Lindau syndrome • Histology: foam cells and high vascularity
Pituitary adenoma	• Pituitary adenomas are benign tumours of the pituitary gland. They are either secretory (producing a hormone in excess) or non-secretory. They may be divided into microadenomas (smaller than 1cm) or macroadenoma (larger than 1cm). • Patients will present with the consequences of hormone excess (e.g. Cushing's due to ACTH, or acromegaly due to GH) or depletion. Compression of the optic chiasm will cause a bitemporal hemianopia due to the crossing nasal fibers. • Investigation requires a pituitary blood profile and MRI. Treatment can either be hormonal or surgical (e.g. transphenoidal resection).
Craniopharyngioma	• Most common paediatric supratentorial tumour • A craniopharyngioma is a solid/cystic tumour of the sellar region that is derived from the remnants of Rathke's pouch. It is common in children, but can present in adults also. It may present with hormonal disturbance, symptoms of hydrocephalus or bitemporal hemianopia. • Histology: Derived from remnants of Rathke pouch • Investigation requires pituitary blood profile and MRI. Treatment is typically surgical with or without postoperative radiotherapy.



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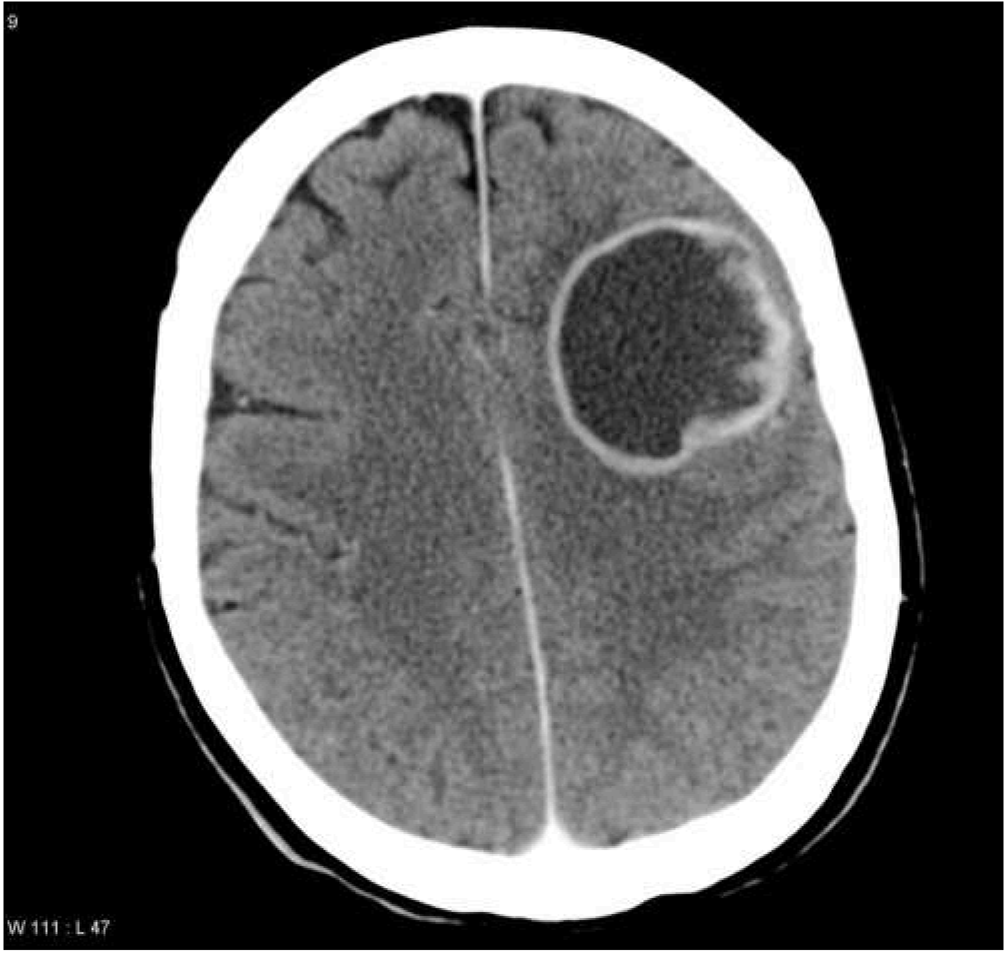
Meningioma - MRI showing the typical well-circumscribed appearance. A dural tail can be where the tumour 'connects' to the dura. It is seen in around 65% of meningiomas.



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Glioblastoma multiforme - CT showing a peripherally enhancing lesion within the left frontal lobe. Note the contrast to the more homogenous meningioma above.

AI + Q 123

Next question >

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PEDIATRIC
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Paediatric brain tumours

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GLIOBLASTOMA
Osmosis
KNOWLEDGE SHOT

Glioblastoma

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Score: **48%**

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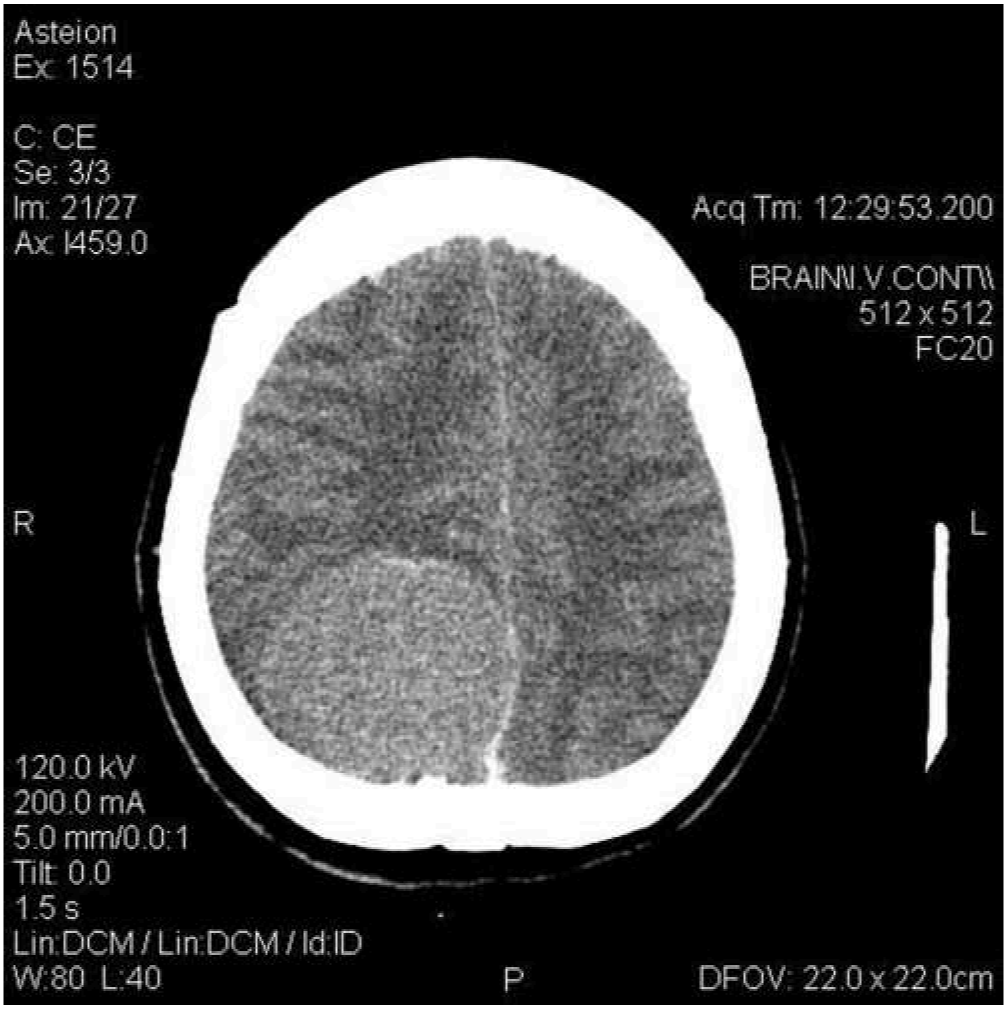
A 45-year-old woman presents to the Emergency Department due to a headache. This has been getting gradually worse over the past 3 months. Her GP has tried a number of therapies including a triptan, amitriptyline and standard analgesia with limited effect. She is a non-smoker and drinks around 30 units of alcohol per week.

Neurological examination is unremarkable.

A CT scan is arranged: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W:80 L:40
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DFOV: 22.0 x 22.0cm

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What is the most likely diagnosis?

○ Extradural haematoma

○ Meningioma

○ Glioblastoma multifome ×

○ Herpes simplex encephalitis ×

○ Subdural haematoma ×

Submit answer

Reference ranges ∨

Score: **0%**

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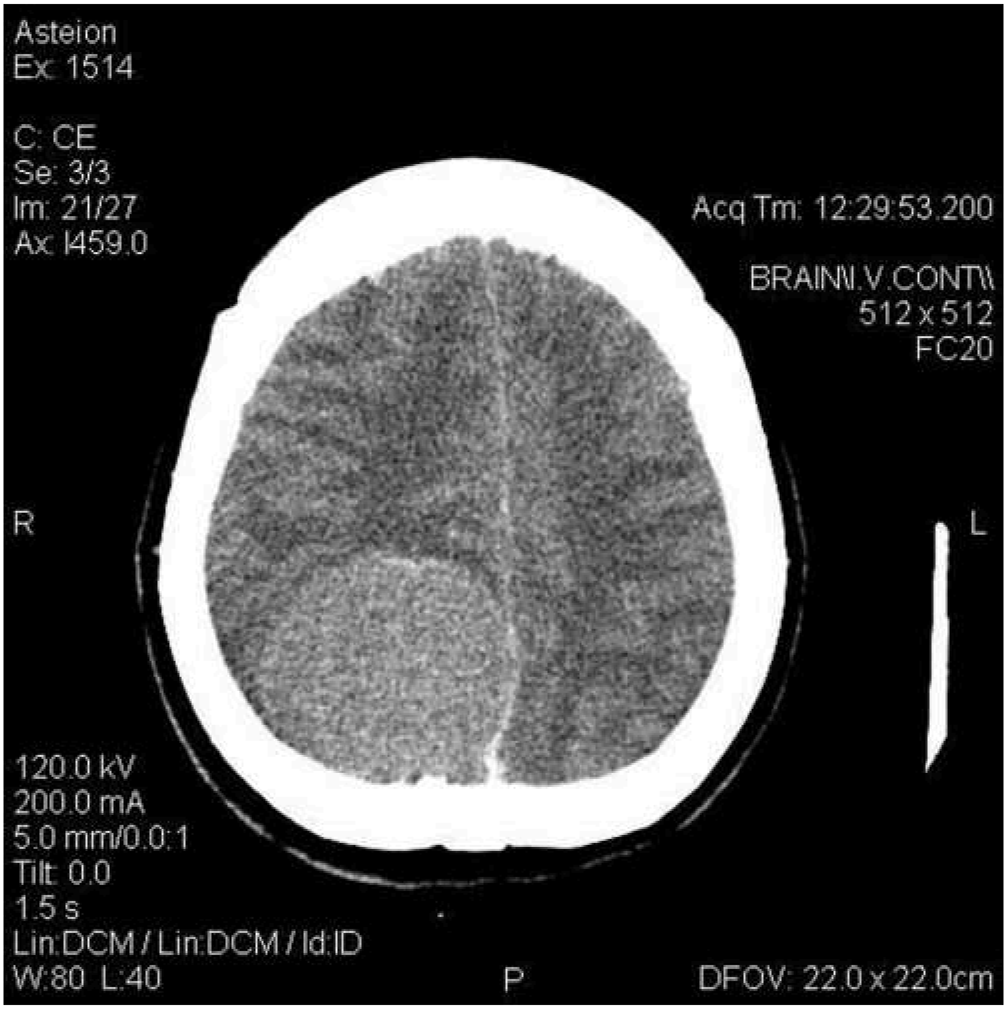
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Lin:DCM / Lin:DCM / Id:ID
W:80 L:40
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DFOV: 22.0 x 22.0cm

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What is the most likely diagnosis?

Extradural haematoma 2%

Meningioma 66%

Glioblastoma multifome	28%
Herpes simplex encephalitis	1%
Subdural haematoma	3%

The CT shows a well defined spherical mass in the right posterior falx cerebri consistent with a meningioma. There is mild oedema and mass effect on the right lateral ventricle. The tumour is straddling the inferior surface of the falx.

A glioblastoma multifome would normally have a more heterogenous appearance than shown here.

Discuss (4) Improve

Next question >

Brain tumours

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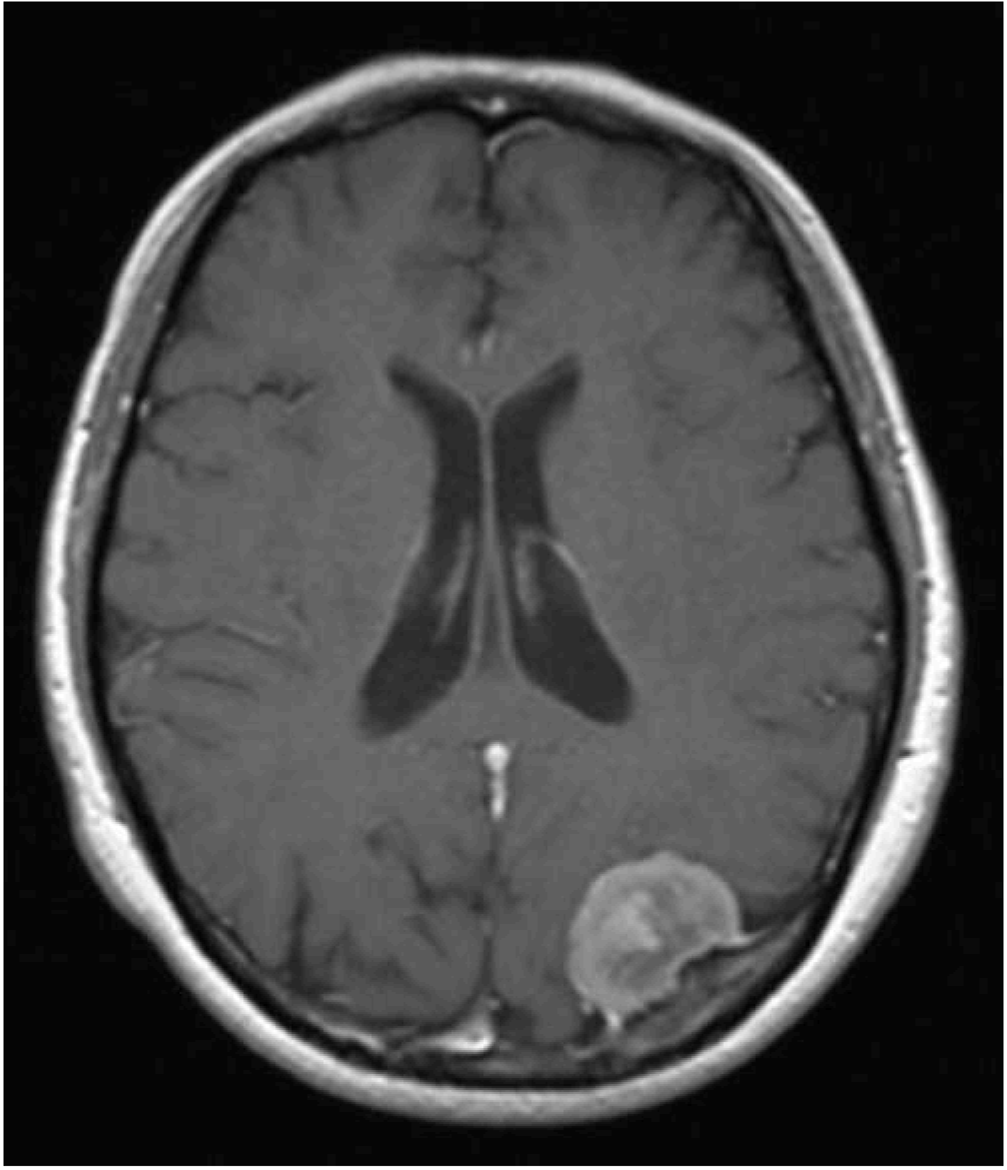

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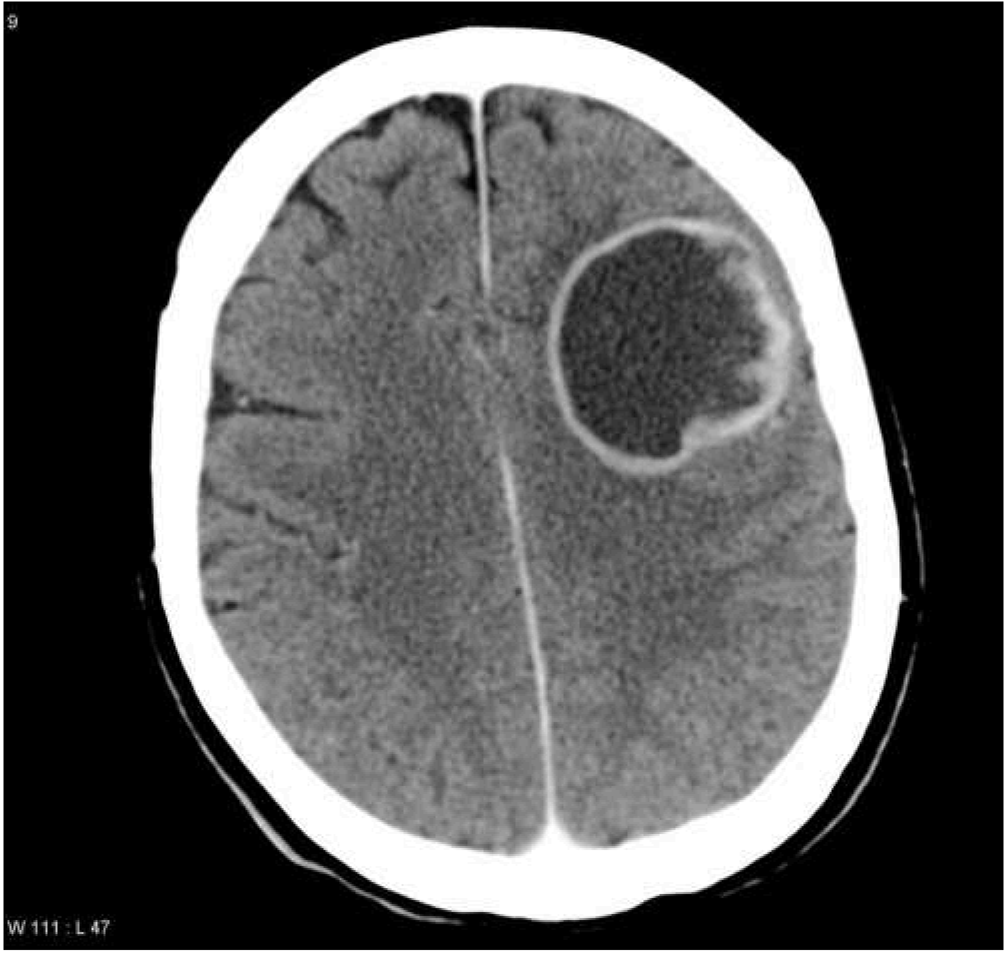
Meningioma - MRI showing the typical well-circumscribed appearance. A dural tail can be where the tumour 'connects' to the dura. It is seen in around 65% of meningiomas.



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Glioblastoma multiforme - CT showing a peripherally enhancing lesion within the left frontal lobe. Note the contrast to the more homogenous meningioma above.

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Glioblastoma

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Score: **48%**

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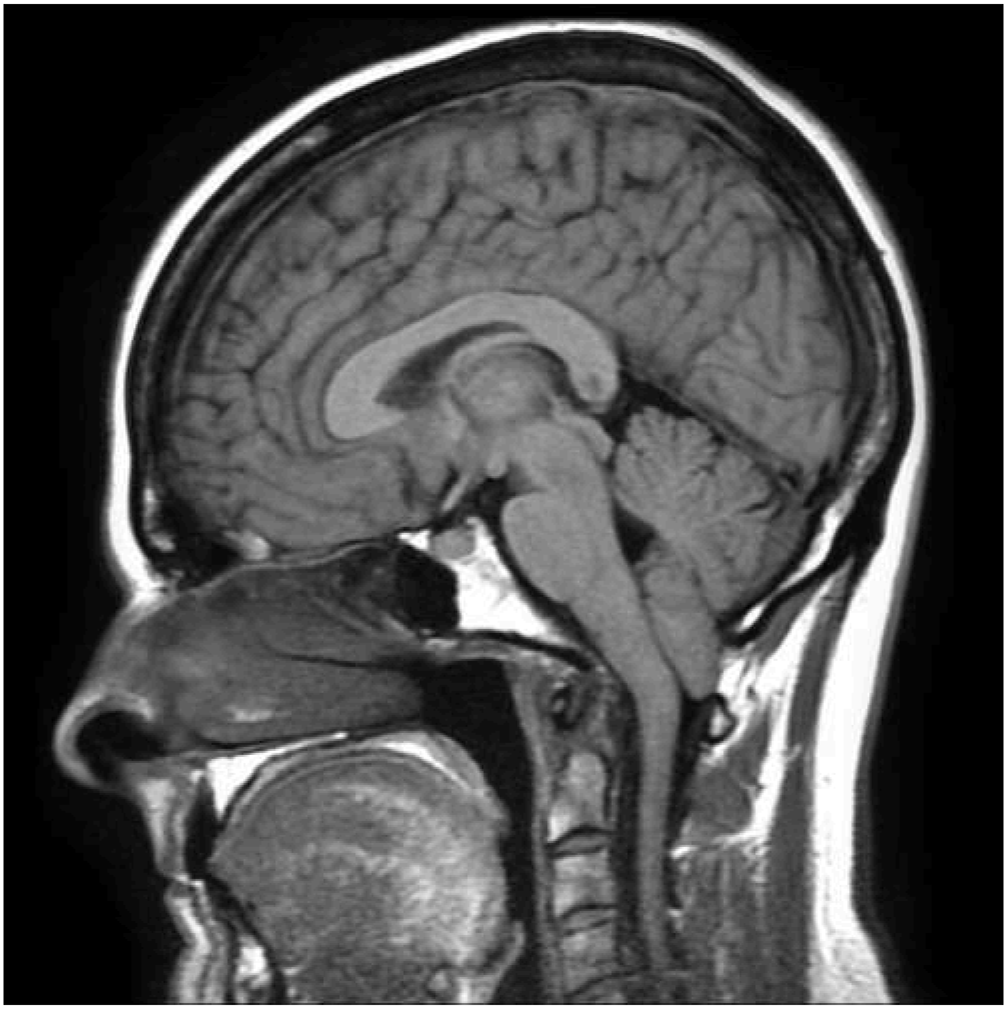
Question 22 of 189

A 34-year-old woman is referred to the neurology clinic for a 6 month history of progressively worsening headaches. The headaches tend to be occipital in nature and are worsened by coughing.

Neurological examination is unremarkable.

Her MRI is shown below:



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What finding is shown on the MRI?

○ Brainstem cavernous angioma

○ Pituitary adenoma

○ Arnold-Chiari malformation ×

○ Arachnoid cyst ×

○ Spina bifida ×

Submit answer

Reference ranges

Score: **0%**

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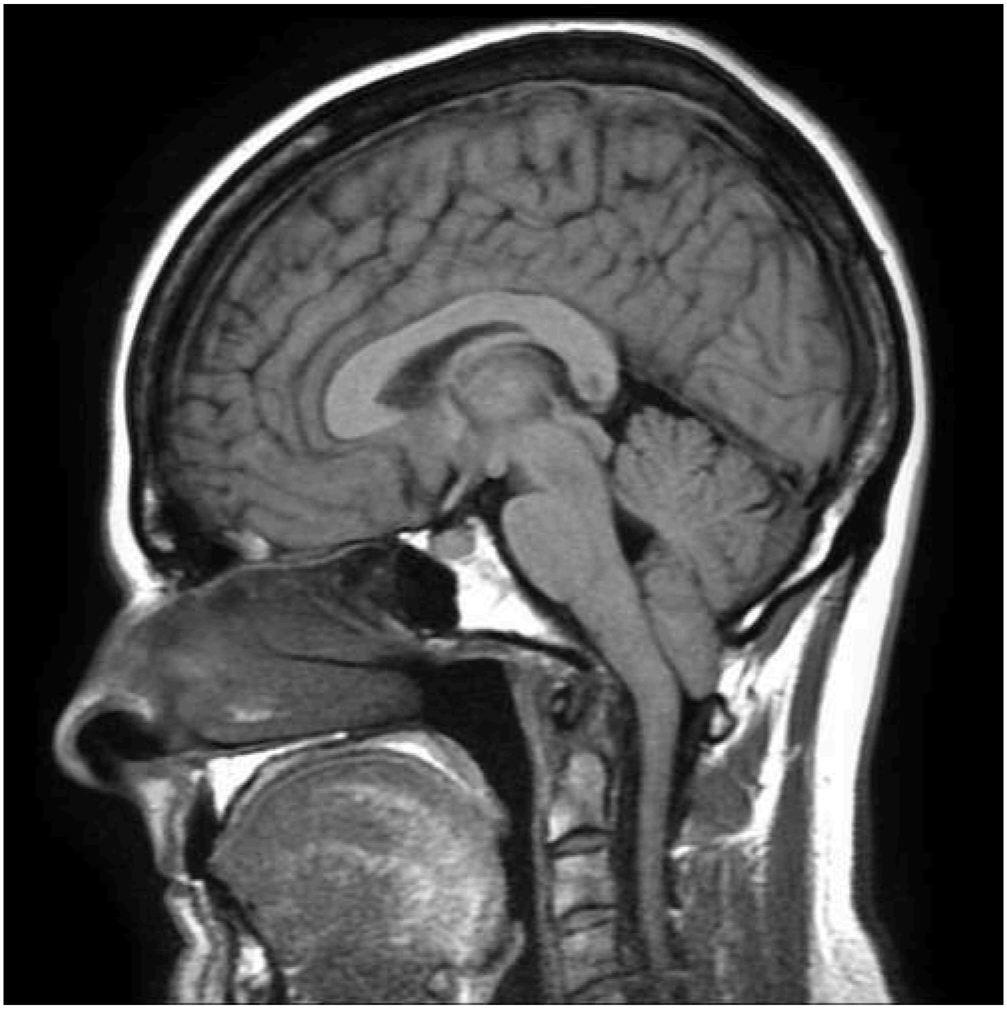

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What finding is shown on the MRI?

Brainstem cavernous angioma	12%
Pituitary adenoma	12%

Arnold-Chiari malformation	67%
Arachnoid cyst	8%
Spina bifida	1%

The MRI shows herniation of the cerebellar tonsils through the foramen magnum consistent with a Chiari type I malformation.

Discuss (4) Improve

Next question

Arnold-Chiari malformation

Arnold-Chiari malformation describes the downward displacement, or herniation, of the cerebellar tonsils through the foramen magnum. Malformations may be congenital or acquired through trauma.

Features
- non-communicating hydrocephalus may develop as a result of obstruction of cerebrospinal fluid (CSF) outflow
- headache
- syringomyelia

123

Next question

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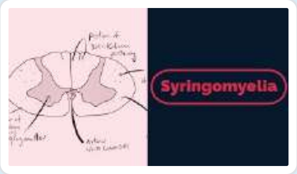
Syringomyelia

Syringomyelia

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BRAIN HERNIATION
Osmosis.org

Brain herniation

Osmosis - YouTube 👍 2 👎 1

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Score: **48%**

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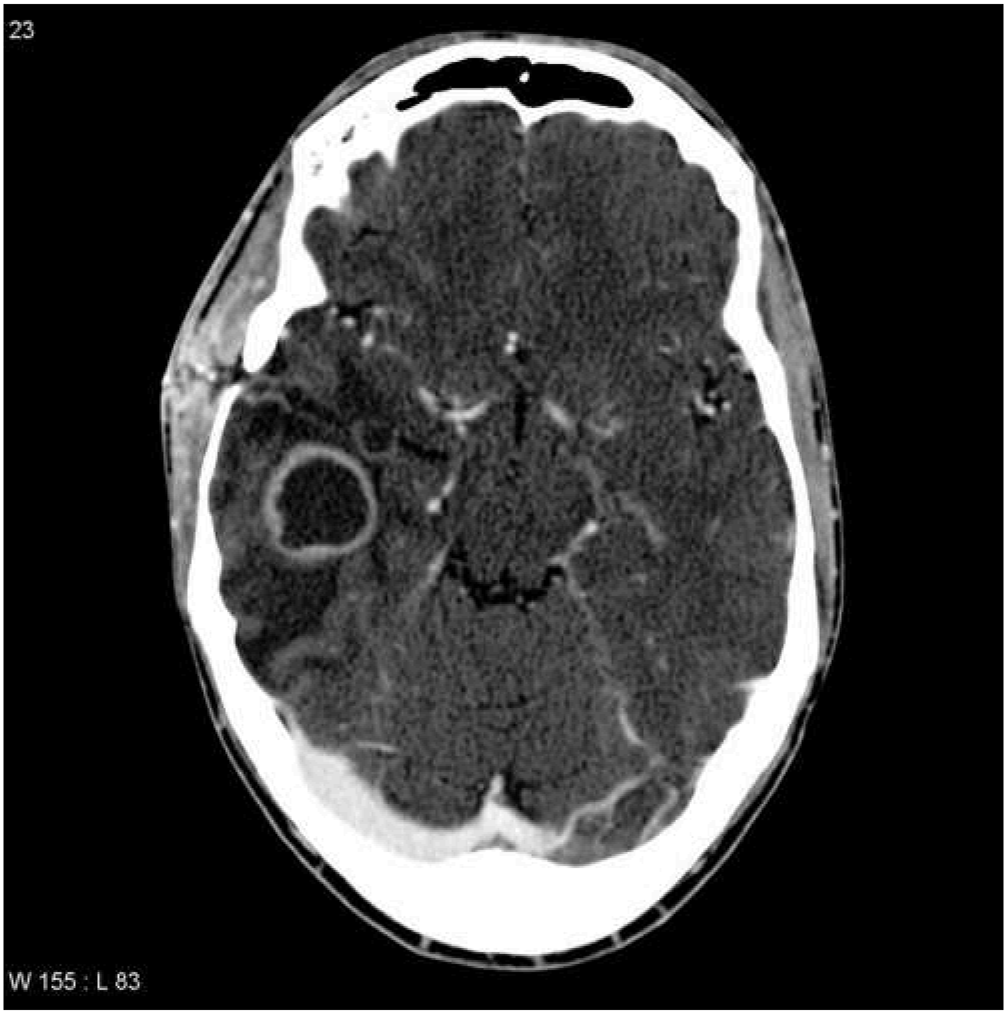
Question 23 of 189

A 23-year-old man is admitted to ITU after arriving from India on a medical transfer plane. He had been travelling in India for around 4 weeks before starting to complain of feeling unwell. His girlfriend describes acting out of character and having numerous seizures prior to being admitted to hospital in Patna, east India.

His girlfriend describes him having a scan in India as well as a procedure in theatre which was abandoned.

A CT head with contrast is performed:



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What is the most likely diagnosis?

○ Neurocysticercosis

○ Meningioma ×

○ Glioblastoma multiforme ×

○ Cerebral abscess ×

○ Herpes simplex encephalitis ×

Submit answer

Reference ranges

Score: **0%**

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Question 23 of 189 ✓

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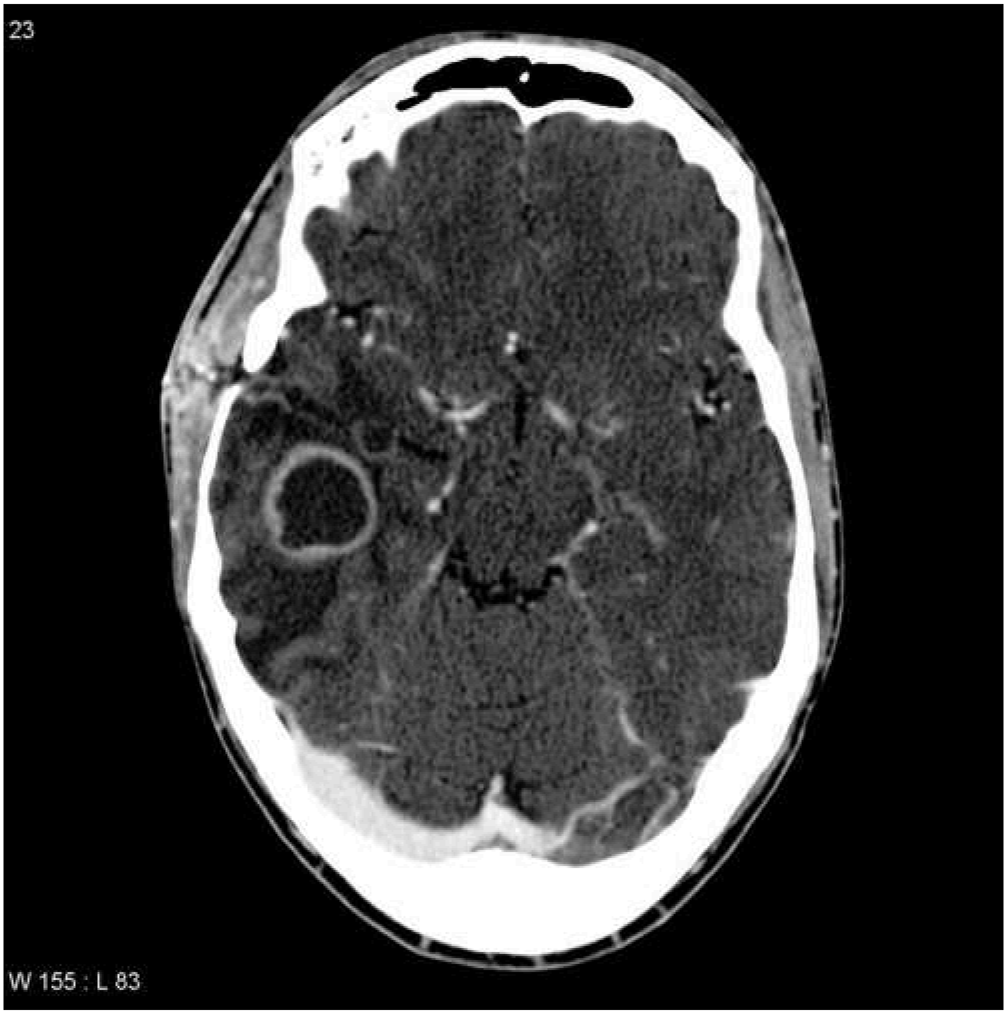

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What is the most likely diagnosis?

Neurocysticercosis	27%

Meningioma	2%
Glioblastoma multiforme	13%
Cerebral abscess	54%
Herpes simplex encephalitis	4%

CT demonstrating a cerebral abscess. CT demonstrates a rounded lesions with vast amounts of surrounding vasogenic oedema resulting in mass effect and distortion of the cerebral peduncle. Contrast confirms the presence of a smooth ring of enhancement, characteristic of an abscess.

The scan shows signs of overlying surgical change consistent with a prior attempted drainage via a burr hole.

This patient underwent repeat surgery with successful drainage of the abscess. He made a slow but uneventful recovery.

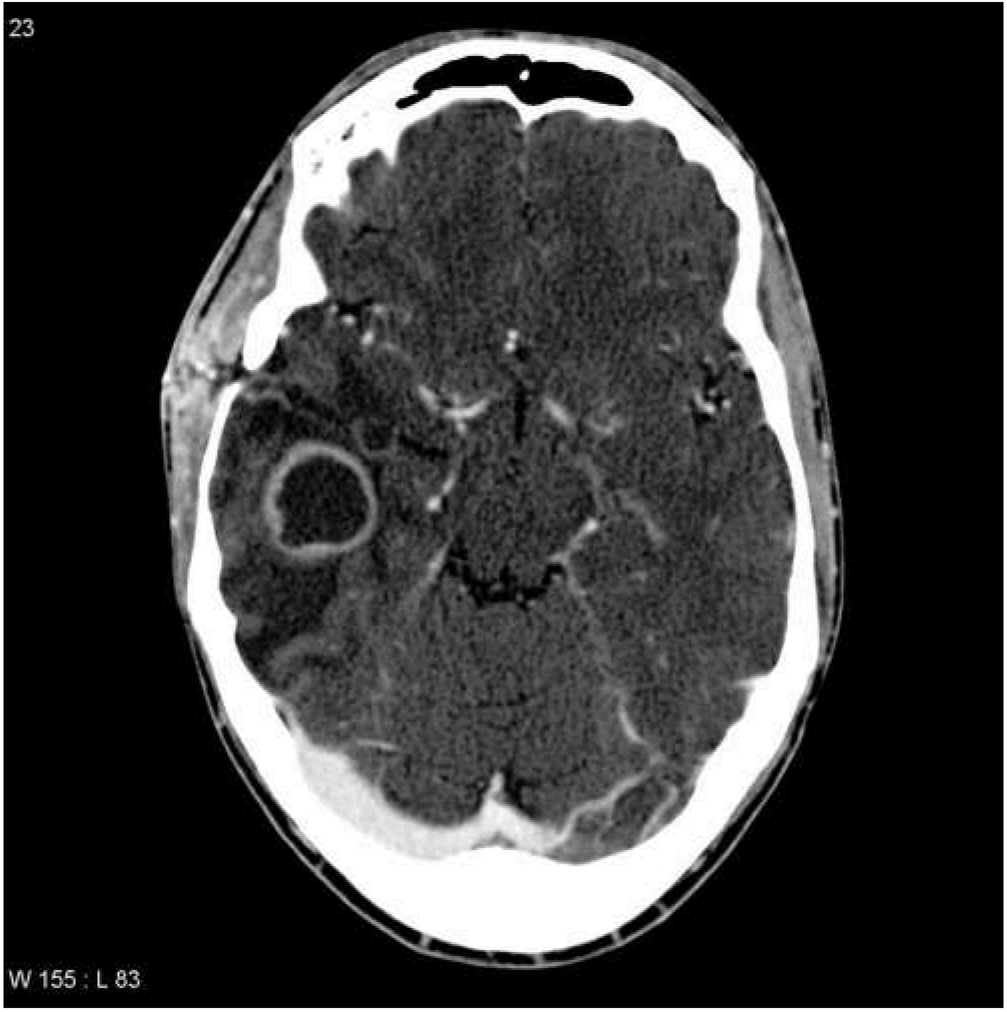
Discuss (10) Improve

Next question >

Intracerebral abscess ★

Features
- fever
- headache
- seizures
- signs of raised intracranial pressure
- focal neurological deficits

Management
- surgical drainage: supratentorial abscesses may be drained via a burr hole
- antibiotic therapy



© Image used on license from Radiopaedia

CT demonstrating a cerebral abscess. CT demonstrates a rounded lesions with vast amounts of surrounding vasogenic oedema resulting in mass effect and distortion of the cerebral peduncle. Contrast confirms the presence of a smooth ring of enhancement, characteristic of an abscess.

+ Q 123

Next question >

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Score: **48%**

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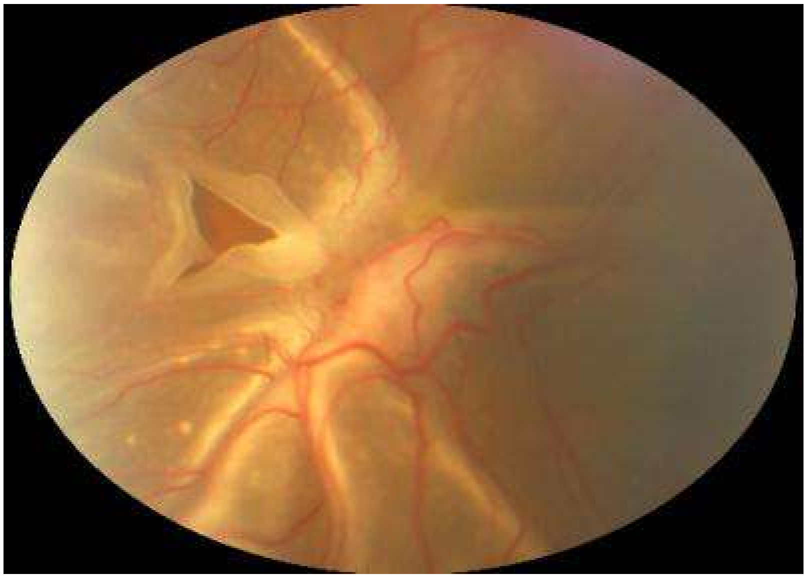
A woman presents with reduced vision in her right eye. She is unsure when it started but can now only make out hand movements with the right eye. Fundoscopy of the affected eye shows the following:



What is the diagnosis?

- ○ Penetrating foreign body
- ○ Myelinated optic disc
- ○ Papilloedema
- ○ Choroidoretinitis
- ○ Retinal detachment

Submit answer

Reference ranges

Score: **0%**

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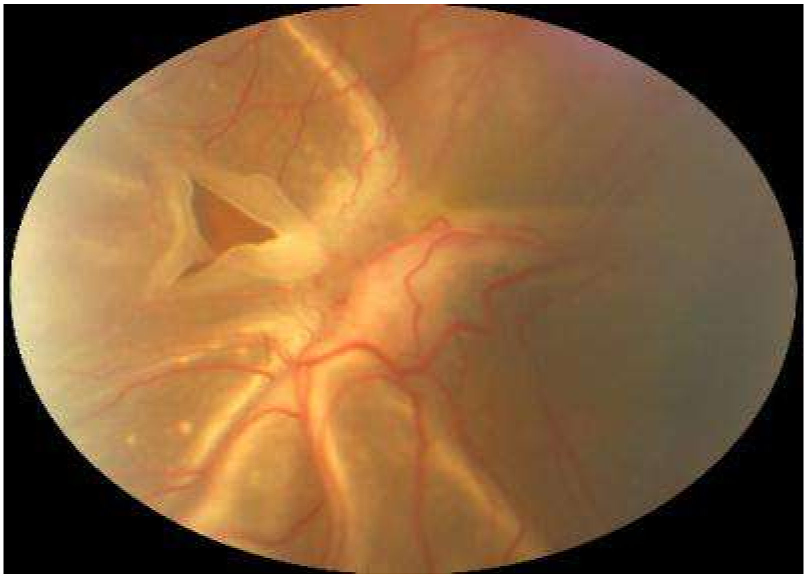

What is the diagnosis?

Penetrating foreign body	12%
Myelinated optic disc	4%
Papilloedema	3%
Choroidoretinitis	6%
Retinal detachment	75%

The correct diagnosis is **Retinal detachment**. This condition presents with symptoms such as sudden or gradual reduction in vision, often described by patients as a 'curtain coming down'. Fundoscopy reveals an elevated retina with folds and a visible retinal tear.

Penetrating foreign body is incorrect because it would typically present with a history of trauma, and the patient would likely experience pain. On examination, there might be visible injury to the cornea or sclera, hyphaema (blood in the anterior chamber), irregular pupil, or low intraocular pressure.

Myelinated optic disc, also known as medullated nerve fibres, is incorrect because it does not typically affect vision. It appears on fundoscopy as whitish patches extending from the optic disc but does not cause elevation or detachment of the retina.

Papilloedema is incorrect because this condition involves swelling of the optic disc due to increased intracranial pressure. Symptoms can include transient visual obscurations and headache. On fundoscopy, there would be blurring of the optic disc margins and elevation of the disc itself rather than the retina.

Finally, **Choroidoretinitis**, inflammation of the choroid and retina usually due to infection such as by *Toxoplasma gondii* or *Cytomegalovirus*, is incorrect because it typically presents with floaters, photopsia (flashes of light), and blurred vision due to inflammatory lesions in the retina. These lesions appear as yellow-white patches during fundoscopy but do not cause retinal detachment unless severe and chronic.

👍 👎 Discuss (5) Improve

Next question >

Sudden loss of vision ★

Sudden loss is a frightening symptom for patients. It may represent an ongoing issue or only be temporary. The term transient monocular visual loss (TMVL) describes a sudden, transient loss of vision that lasts less than 24 hours.

The most common causes of a sudden painless loss of vision are as follows:
- ischaemic/vascular (e.g. thrombosis, embolism, temporal arteritis etc). This includes recognised syndromes e.g. occlusion of central retinal vein and occlusion of central retinal artery
- vitreous haemorrhage
- retinal detachment
- retinal migraine

Ischaemic/vascular
- often referred to as 'amaurosis fugax'
- wide differential including large artery disease (atherothrombosis, embolus, dissection), small artery occlusive disease (anterior ischemic optic neuropathy, vasculitis e.g. temporal arteritis), venous disease and hypoperfusion

- may represent a form of transient ischaemic attack (TIA). It should therefore be treated in a similar fashion, with aspirin 300mg being given
- altitudinal field defects are often seen: 'curtain coming down'
- ischaemic optic neuropathy is due to occlusion of the short posterior ciliary arteries, causing damage to the optic nerve

Central retinal vein occlusion
- incidence increases with age, more common than arterial occlusion
- causes: glaucoma, polycythaemia, hypertension
- severe retinal haemorrhages are usually seen on fundoscopy

Central retinal artery occlusion
- due to thromboembolism (from atherosclerosis) or arteritis (e.g. temporal arteritis)
- features include afferent pupillary defect, 'cherry red' spot on a pale retina

Vitreous haemorrhage
- causes: diabetes, bleeding disorders, anticoagulants
- features may include sudden visual loss, dark spots

Retinal detachment
- features of vitreous detachment, which may precede retinal detachment, include flashes of light or floaters (see below)

Differentiating posterior vitreous detachment, retinal detachment and vitreous haemorrhage

Posterior vitreous detachment	**Retinal detachment**	**Vitreous haemorrhage**
Flashes of light (photopsia) - in the peripheral field of vision Floaters, often on the temporal side of the central vision	Dense shadow that starts peripherally progresses towards the central vision A veil or curtain over the field of vision Straight lines appear curved Central visual loss	Large bleeds cause sudden visual loss Moderate bleeds may be described as numerous dark spots Small bleeds may cause floaters

Next question >

Textbooks

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Links

The Royal Australian College of General Practitioners Sudden loss of vision review	👍	16	👎	20
Patient.info Retinal artery occlusions	👍	14	👎	11
American Academy of Ophthalmology Central Retinal Vein Occlusion	👍	16	👎	10
American Academy of Ophthalmology Central Retinal Artery Occlusion	👍	15	👎	7

Suggest link

Report broken link

Score: **48%**

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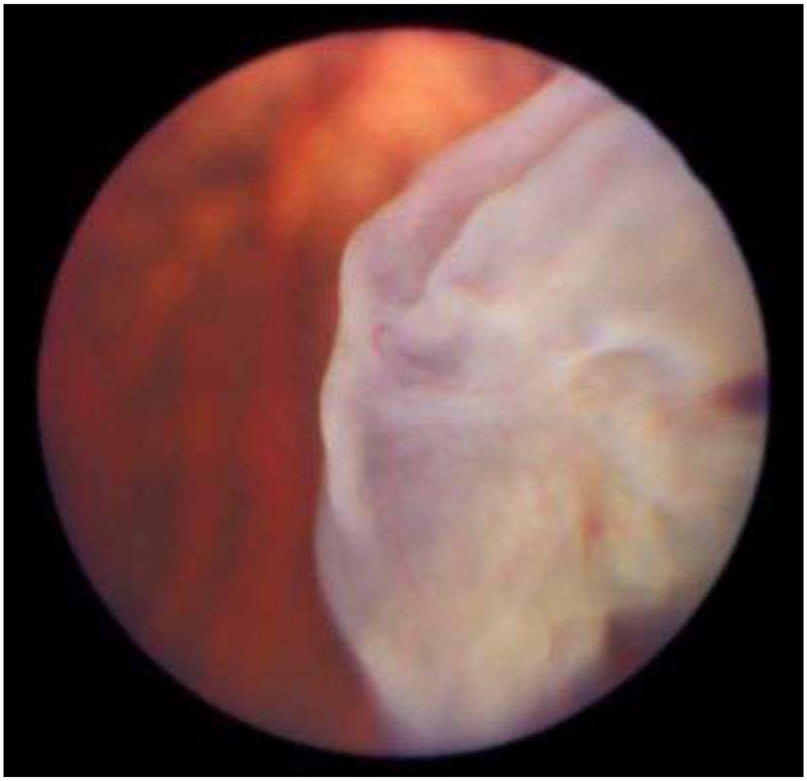
A 71-year-old man presents with sudden painless loss of vision in his left eye: Fundoscopy shows the following:



What is the most likely diagnosis?

- ○ Ciliary body rupture with lens dislocation
- ○ Vitreous haemorrhage
- ○ Ischaemic optic neuropathy
- ○ Central retinal artery occlusion
- ○ Retinal detachment

Submit answer

Reference ranges

Score: **0%**

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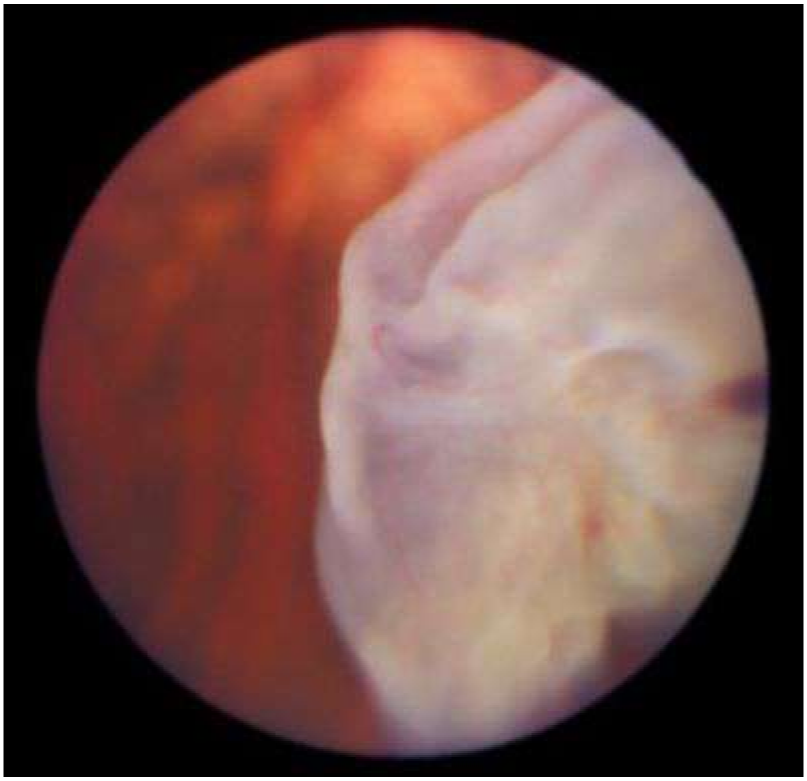
Question 25 of 189 ✓

A 71-year-old man presents with sudden painless loss of vision in his left eye: Fundoscopy shows the following:



What is the most likely diagnosis?

- Ciliary body rupture with lens dislocation 11%
- Vitreous haemorrhage 7%
- Ischaemic optic neuropathy 2%
- Central retinal artery occlusion 3%
- Retinal detachment 77%

Discuss (2) | Improve

Next question >

Sudden loss of vision ★

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- retinal detachment
- retinal migraine

Ischaemic/vascular
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- features include afferent pupillary defect, 'cherry red' spot on a pale retina

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- causes: diabetes, bleeding disorders, anticoagulants
- features may include sudden visual loss, dark spots

Retinal detachment

- features of vitreous detachment, which may precede retinal detachment, include flashes of light or floaters (see below)

Differentiating posterior vitreous detachment, retinal detachment and vitreous haemorrhage

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+ Q 123

Next question ›

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Links

The Royal Australian College of General Practitioners

👍 16 👎 20

Sudden loss of vision review

Patient.info 👍 14 👎 11

Retinal artery occlusions

American Academy of Ophthalmology 👍 16 👎 10

Central Retinal Vein Occlusion

American Academy of Ophthalmology 👍 15 👎 7

Central Retinal Artery Occlusion

Suggest link Report broken link

Score: **48%**

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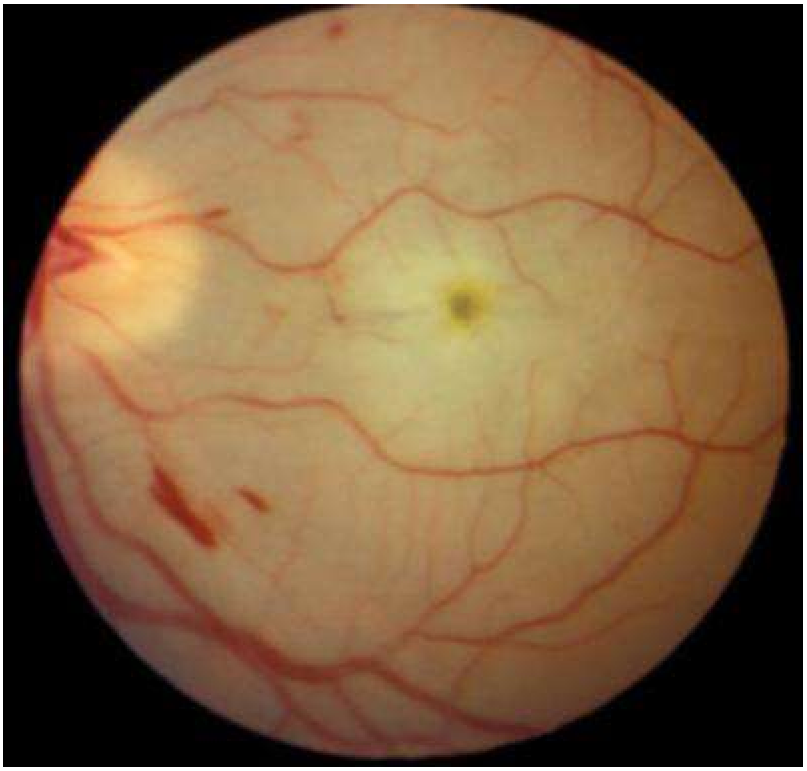
Question 26 of 189

This man presents with unilateral visual loss. On examination he has a relative afferent pupillary defect. Fundoscopy shows the following:



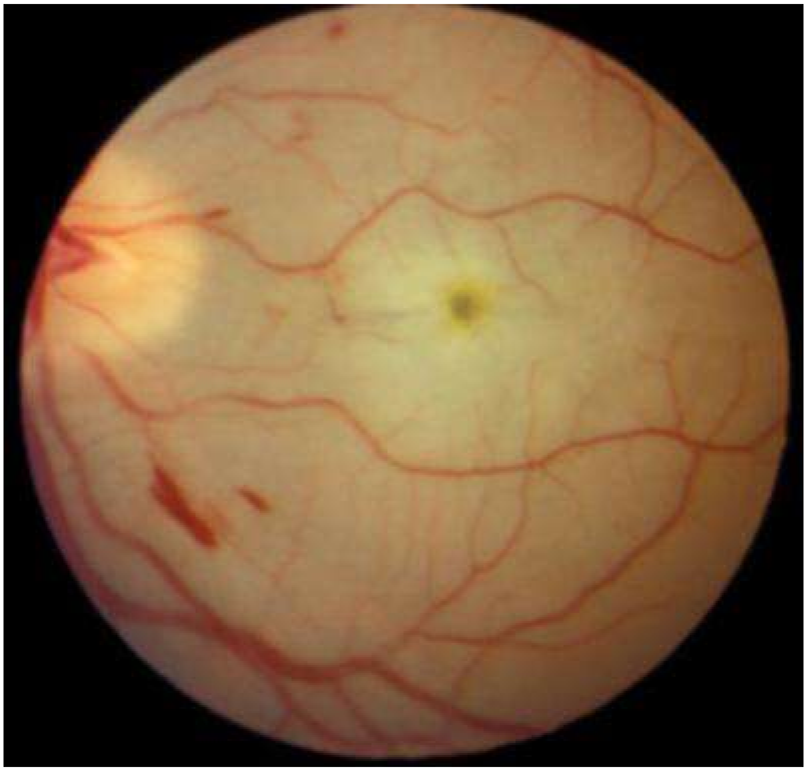
What is the most likely diagnosis?

- ○ Central retinal vein occlusion
- ○ Subhyaloid haemorrhage
- ○ Solar retinopathy
- ○ Central retinal artery occlusion
- ○ Optic neuritis

Submit answer

Reference ranges

This man presents with unilateral visual loss. On examination he has a relative afferent pupillary defect. Fundoscopy shows the following:



What is the most likely diagnosis?

Central retinal vein occlusion	8%
Subhyaloid haemorrhage	3%
Solar retinopathy	4%
Central retinal artery occlusion	69%
Optic neuritis	16%

The correct answer is **Central retinal artery occlusion**. This condition presents with sudden, painless, unilateral vision loss. On examination, there may be a relative afferent pupillary defect (RAPD), also known as Marcus Gunn pupil, which is an abnormal response to light in the affected eye due to a severe decrease in the number of functioning retinal ganglion cells. Fundoscopy typically reveals a pale retina with a cherry-red spot at the fovea due to obstruction of blood flow in the central retinal artery.

Central retinal vein occlusion is incorrect because it presents differently. Patients often complain of sudden or gradual vision loss. On fundoscopy, there are widespread retinal haemorrhages ('blood and thunder' appearance) and cotton wool spots due to blockage of the central retinal vein leading to increased pressure and subsequent leakage from other vessels.

Subhyaloid haemorrhage is also incorrect. It presents with sudden visual loss or floaters but does not typically cause RAPD. On fundoscopy, you would see a pre-retinal (subhyaloid) haemorrhage which appears as a dark red, boat-shaped mass located between the retina and vitreous humour.

The diagnosis of **Solar retinopathy** is unlikely in this case as it results from direct solar radiation damage to the macula leading to central scotoma (blind spot). The typical fundoscopic finding is yellowish dots at the level of RPE in fovea.

Lastly, **Optic neuritis**, commonly associated with multiple sclerosis, typically presents with painful vision loss over days accompanied by dyschromatopsia (impaired colour vision). However, fundoscopy may initially appear normal or show mild disc swelling rather than the findings described here.

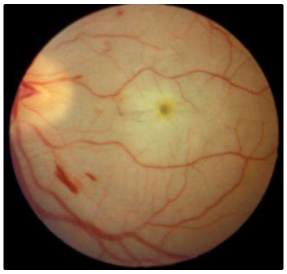
👍 | 👎 | Discuss (1) | Improve

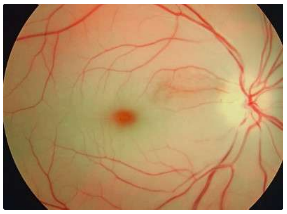
Next question ›

Central retinal artery occlusion ★

Central retinal artery occlusion is a relatively rare cause of sudden unilateral visual loss. It is due to thromboembolism (from atherosclerosis) or arteritis (e.g. temporal arteritis)

Features
- sudden, painless unilateral visual loss
- relative afferent pupillary defect
- 'cherry red' spot on a pale retina





Management is difficult and the prognosis is poor

- any underlying conditions should be identified and treated (e.g. intravenous steroids for temporal arteritis)
- if a patient presents acutely then Intraarterial thrombolysis may be attempted but currently, trials show mixed results.

+ Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

American Academy of Ophthalmology 👍 9 👎 4

Central Retinal Artery Occlusion

Suggest link Report broken link

Score: **28%**

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Question 27 of 189

A 19-year-old university student is reviewed in the general medical clinic with bilateral ptosis. It has developed gradually over the last few months and has now progressed to the extent that he has to tilt his head upwards during lectures. He denies diurnal variation in his symptoms.

His past medical history is unremarkable and he takes no regular medications. He tells you that his mother had a pacemaker fitted due to frequent 'blackouts'.

On examination, you note the presence of bilateral ptosis. He also has impaired abduction and adduction of both eyes. Direct fundoscopy shows black spicules in the periphery of both eyes. An ECG is taken and shows sinus rhythm with first-degree heart block.

Given the likely diagnosis, which additional symptom is he most likely to describe?

- ○ Night blindness ×
- ○ Syncope ×
- ○ Tremor ×
- ○ Flank pain ×
- ○ Diplopia ×

Submit answer

Reference ranges

Score: **48%**

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On examination, you note the presence of bilateral ptosis. He also has impaired abduction and adduction of both eyes. Direct fundoscopy shows black spicules in the periphery of both eyes. An ECG is taken and shows sinus rhythm with first-degree heart block.

Given the likely diagnosis, which additional symptom is he most likely to describe?

🟩 Night blindness	71%
Syncope	6%
Tremor	5%
Flank pain	5%
🟥 Diplopia	13%

The most likely diagnosis is Kearns-Sayre syndrome, a mitochondrial disorder. Individuals usually present <20 years-of-age with progressive external ophthalmoplegia. Ptosis usually develops first, followed by difficulties with horizontal gaze. Patients don't usually complain of diplopia as the extraocular muscle weakness is symmetrical.

Affected patients also usually suffer from pigmentary retinopathy, which causes atrophy of the retinal pigment with bony spicule formation typical of retinitis pigmentosa. As the changes progress, patients often complain of funnel-vision and night blindness.

Cardiac conduction defects usually develop after the onset of ophthalmoplegia and sudden death may occur as a result. Regular cardiac follow-up is essential. Other abnormalities include cerebellar ataxia, sensorineural deafness, muscle weakness and diabetes mellitus.

Discuss (8) Improve

Next question

Retinitis pigmentosa

Retinitis pigmentosa primarily affects the peripheral retina resulting in tunnel vision

Features

- night blindness is often the initial sign
- tunnel vision due to loss of the peripheral retina (occasionally referred to as funnel vision)
- fundoscopy: black bone spicule-shaped pigmentation in the peripheral retina, mottling of the retinal pigment epithelium

Associated diseases

- Refsum disease: cerebellar ataxia, peripheral neuropathy, deafness, ichthyosis
- Usher syndrome
- abetalipoproteinemia
- Lawrence-Moon-Biedl syndrome
- Kearns-Sayre syndrome
- Alport's syndrome

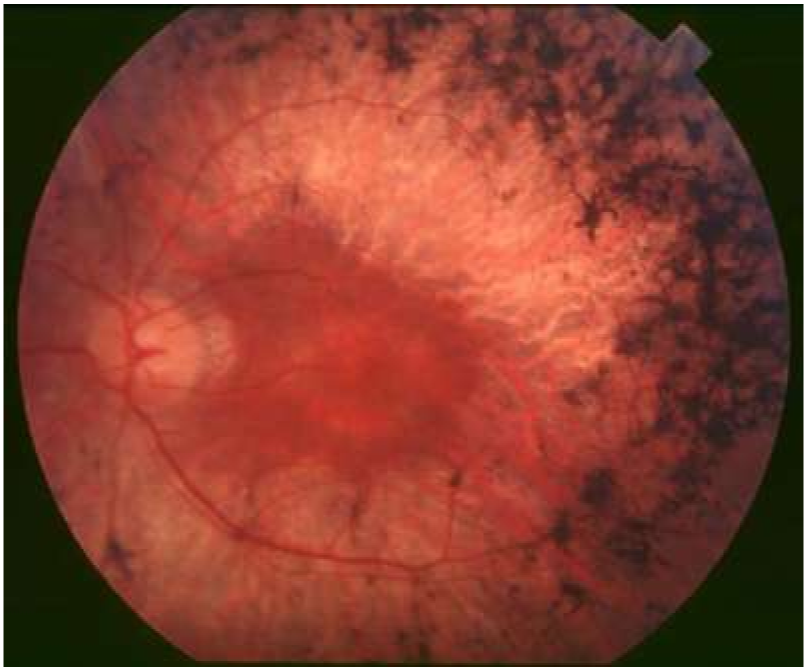

Image sourced from Wikipedia

Fundus showing changes secondary to retinitis pigmentosa

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **28%**

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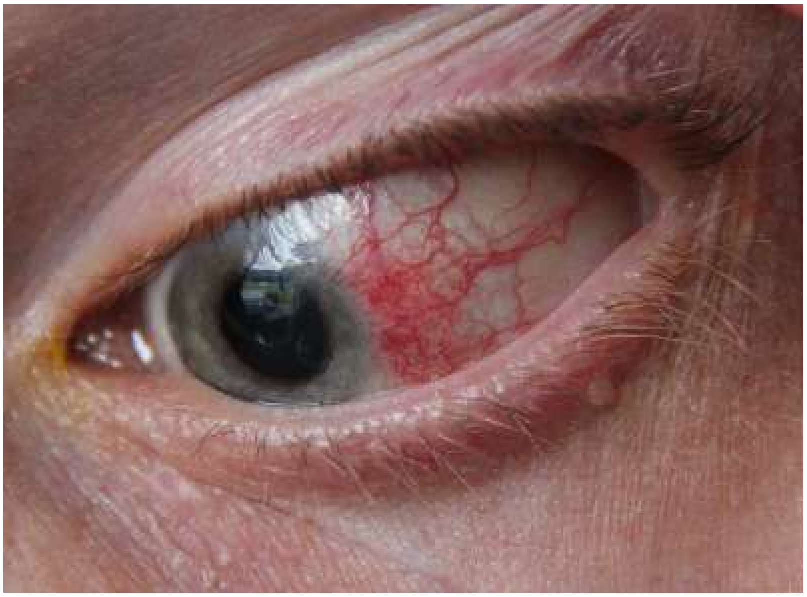
Question 28 of 189

A 44-year-old man attends after waking up with a red eye and some slight crusting. He describes mild pain and finds the eye is tearing excessively. His vision is unaffected.



Slit lamp examination shows congestive vessels with a radial distribution pattern.

He works as a carpenter and has a background of ulcerative colitis but is otherwise fit and well.

What is the most likely diagnosis?

- ○ Conjunctivitis
- ○ Episcleritis
- ○ Exposure keratopathy
- ○ Ocular foreign body
- ○ Scleritis

Submit answer

Reference ranges

Score: **48%**

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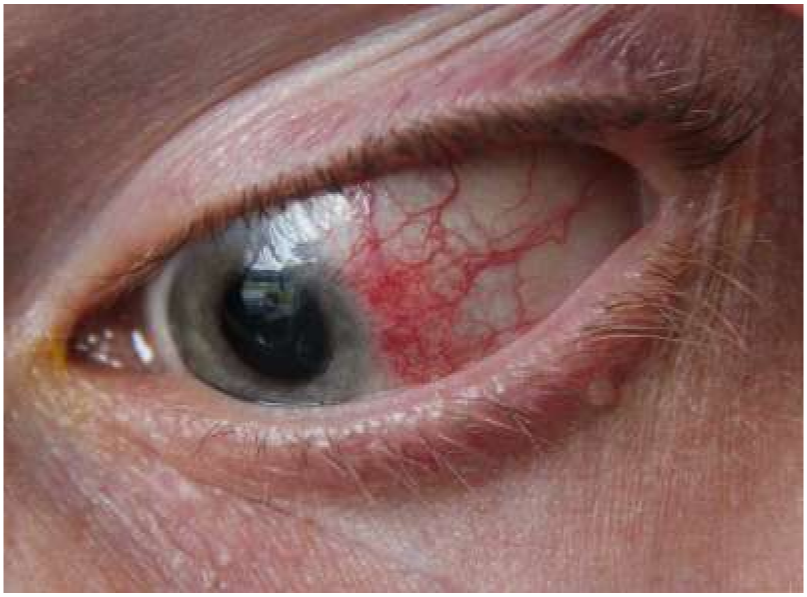


Slit lamp examination shows congestive vessels with a radial distribution pattern.

He works as a carpenter and has a background of ulcerative colitis but is otherwise fit and well.

What is the most likely diagnosis?

Conjunctivitis	6%
Episcleritis	62%
Exposure keratopathy	3%
Ocular foreign body	3%
Scleritis	25%

This is a presentation of episcleritis. Episcleritis is usually painless or with mild pain, there are prominent surface vessels that are mobile and blanch with phenylephrine drops.

Episcleritis is correct. It is an inflammatory condition affecting the episclera and is usually self-limiting. It is not classically associated with severe pain and vision is preserved.

Conjunctivitis is incorrect. This usually presents with purulent discharge and extensive crusting around the eyes. The conjunctiva is often inflamed. It commonly initially affects one eye but can spread easily to affect both. In this case, there is no purulent discharge and so conjunctivitis is less likely.

Exposure keratopathy is incorrect. This is when the cornea is exposed for prolonged periods of time. It can present with a gritty or foreign body sensation and the damaged areas of the cornea will stain with fluorescein. In this case, there is no foreign body sensation and no staining on the cornea.

Ocular foreign body is incorrect. This would commonly be associated with discomfort and a foreign body would be seen on slit lamp examination. This is not seen here.

Scleritis is incorrect. This is also an inflammatory condition but is more classically associated with pain, photophobia, and blurring of the vision. Slit lamp examination usually shows new vessel formation and abnormal vessel distribution. These features are not present for the patient in the stem.

👍 👎 Discuss (1) Improve

Next question ›

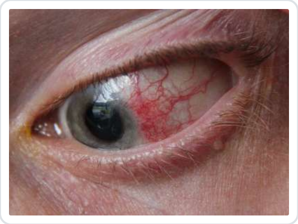
Episcleritis ★

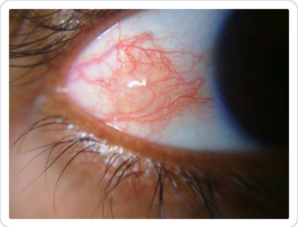
Episcleritis is describes the acute onset of inflammation in the episclera of one or both eyes.

The majority of cases are idiopathic, associated conditions include:
- inflammatory bowel disease
- rheumatoid arthritis

Features
- red eye
- classically not painful (in comparison to scleritis), but mild pain/irritation is common
- watering and mild photophobia may be present
- in episcleritis, the injected vessels are mobile when gentle pressure is applied on the sclera
    - in scleritis, vessels are deeper, hence do not move
- phenylephrine drops may be used to differentiate between episcleritis and scleritis
    - phenylephrine blanches the conjunctival and episcleral vessels but not the scleral vessels

    - if the eye redness improves after phenylephrine a diagnosis of episcleritis can be made
- approximately 50% of cases are bilateral





Management
- conservative
- artificial tears may sometimes be used

+ Q 123

Next question >

B *I* A

Textbooks

High-yield textbook

Extended textbook

Score: **28%**

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A 45-year-old lady was admitted to the Medical Admission Unit with a 16-hour history of weakness. Two days prior to the admission she experienced a sensation of double vision and shortly afterwards had become more unsteady when walking. The weakness developed initially in her arms and was shortly followed by weakness in her legs. Her past medical history included a history of coeliac disease for which she adhered to a strict gluten-free diet, but she was otherwise fit and well.

On examination, she was not distressed and was fully orientated to her surroundings. She had a temperature of 37.6ºC, a heart rate of 88/min, a respiratory rate of 18/min and a blood pressure of 182/82 mmHg. The cardiovascular and respiratory examination was otherwise unremarkable. Examination of the abdomen revealed a mass arising from the symphysis pubis. Examination of her cranial nerves revealed a failure of bilateral external gaze, as well as diplopia on asking the patient to fixate down and in. Her pupils were dilated but reactive. Fundoscopy revealed no abnormalities, and cranial nerve examination was otherwise unremarkable. The tone was reduced in all muscle groups, with a power of grade 3/5 in all muscle groups. Reflexes were absent in all limbs and plantar responses were normal. She was unable to mobilise independently and an ataxia was noted.

Initial investigations revealed the following:

Hb	132 g/l
Platelets	222 * 10^9/l
WBC	5.8 * 10^9/l
ESR	16 mm/hr

Na$^+$	139 mmol/l
K$^+$	3.7 mmol/l
Urea	4.3 mmol/l
Creatinine	77 µmol/l
Bilirubin	11 µmol/l
ALP	101 u/l
ALT	22 u/l
Glucose	6.0 mmol/l
CRP	22 mg/l

Appearance	Clear
Glucose	4.5 mmol/l
Protein	0.6 g/l
White cells	3 / mm^3

CT brain scan: normal appearances, no evidence of haemorrhage, midline shift or space occupying lesion.

What is the single investigation most likely to lead to a diagnosis?

○ Anti Jo1 antibodies ×

○ Anticholinesterase antibodies ×

○ Anti GQ1b antibodies ×

○ Anti GM1 antibodies ×

○ Antinuclear antibodies ×

Submit answer

Reference ranges

Score: **48%**

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CRP	22 mg/l

Appearance	Clear
Glucose	4.5 mmol/l
Protein	0.6 g/l
White cells	3 / mm³

CT brain scan: normal appearances, no evidence of haemorrhage, midline shift or space occupying lesion.

What is the single investigation most likely to lead to a diagnosis?

Anti Jo1 antibodies	6%
Anticholinesterase antibodies	10%
Anti GQ1b antibodies	67%
Anti GM1 antibodies	16%
Antinuclear antibodies	1%

The combination of ophthalmoplegia, ataxia and areflexia is strongly suggestive of Miller-Fisher syndrome, a variant of Guillain-Barre syndrome (GBS). Anti GQ1b antibodies are present in 90% of cases. Anti GM1 antibodies are present in axonal neuropathies including GBS but also other conditions and are not specific to Miller Fisher syndrome. Anti Jo1 antibodies are found in polymyositis whilst anticholinesterase antibodies are found in myasthenia gravis.

👍 👎 Discuss (4) Improve

Next question >

Guillain-Barre syndrome ★

Guillain-Barre syndrome describes an immune-mediated demyelination of the peripheral nervous system often triggered by an infection (classically *Campylobacter jejuni*)

Pathogenesis

- cross-reaction of antibodies with gangliosides in the peripheral nervous system
- correlation between anti-ganglioside antibody (e.g. anti-GM1) and clinical features has been demonstrated
- anti-GM1 antibodies in 25% of patients

Miller Fisher syndrome
- variant of Guillain-Barre syndrome
- associated with ophthalmoplegia, areflexia and ataxia. The eye muscles are typically affected first
- usually presents as a descending paralysis rather than ascending as seen in other forms of Guillain-Barre syndrome
- anti-GQ1b antibodies are present in 90% of cases

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Medcomic 👍 18 👎 4

Guillain-Barre syndrome

Suggest link

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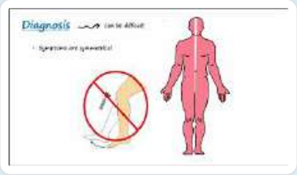
Media

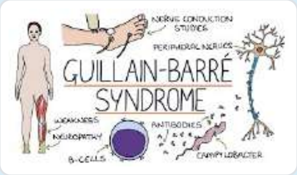


Guillain-Barre Syndrome (GBS) (Described Concisely)

PhysioPathoPharmaco - YouTube 👍 0 👎 0

Guillain-Barre syndrome

Osmosis - YouTube 👍 0 👎 0


NERVE CONDUCTION STUDIES
PERIPHERAL NERVES
GUILLAIN-BARRÉ SYNDROME
WEAKNESS
NEUROPATHY
ANTIBODIES
B-CELLS
CAMPYLOBACTER

Understanding Guillain-Barre Syndrome

Zero To Finals - YouTube 👍 0 👎 1

Report broken media

Score: **28%**

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A 35-year-old man with a vague history of vision problems is reviewed in the ophthalmology clinic. He has not seen a doctor in years and does not report a record of any medical illness.

Fundoscopic examination revealed the following:

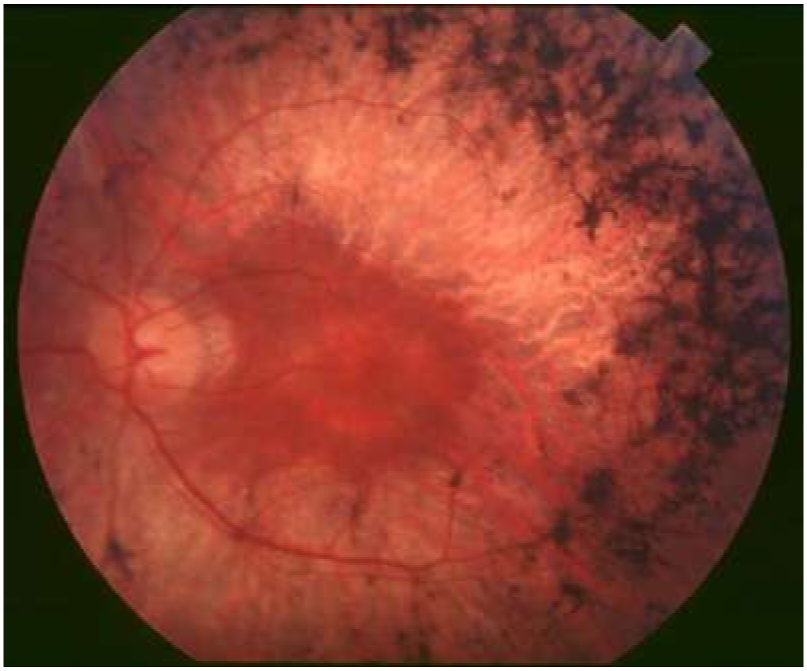

Image sourced from Wikipedia

What is the most likely cause of the fundoscopic findings?

- ○ Hereditary retinal degeneration
- ○ Hypertensive retinopathy
- ○ Non-proliferative diabetic retinopathy
- ○ Proliferative diabetic retinopathy
- ○ Vitreous haemorrhage

Submit answer

Reference ranges

Score: **48%**

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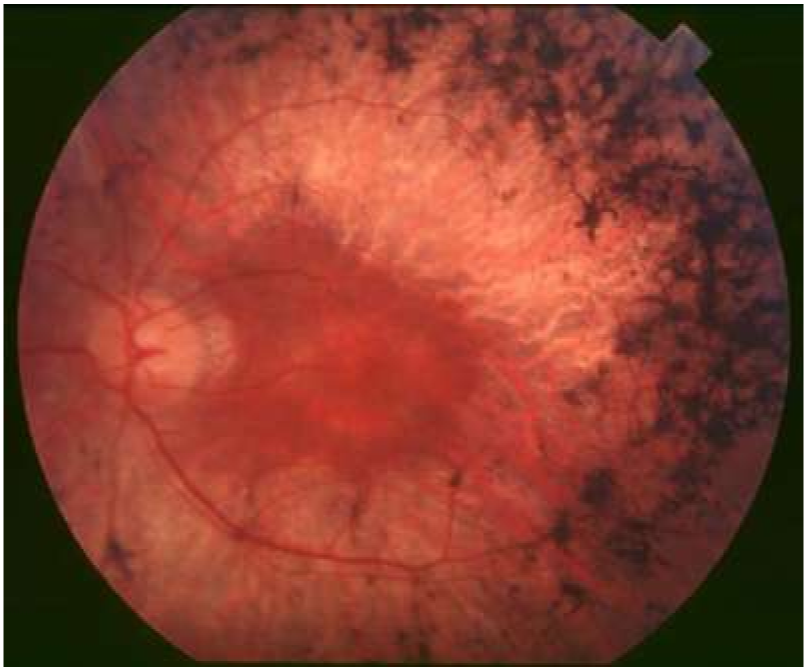

Image sourced from Wikipedia

What is the most likely cause of the fundoscopic findings?

Hereditary retinal degeneration	65%
Hypertensive retinopathy	5%
Non-proliferative diabetic retinopathy	2%
Proliferative diabetic retinopathy	12%
Vitreous haemorrhage	16%

The black bone spicule-shaped pigmentation in the peripheral retina and the mottling of the retinal pigment epithelium seen in this fundoscopic image suggest retinitis pigmentosa.

Retinitis pigmentosa is a hereditary condition characterised by retinal degeneration, affecting predominantly the rods and the peripheral retina. Night blindness is often the initial sign, and tunnel vision is due to the loss of the peripheral retina.

Hypertensive retinopathy is characterised by arteriolar narrowing and AV nipping in the early stages. The flame and blot haemorrhages collected around the fovea can result in a characteristic macular star pattern. Papilloedema is seen in advanced disease.

Non-proliferative diabetic retinopathy is characterised by microaneurysms, blot haemorrhages, hard exudates, cotton wool spots and venous beading or looping with no evidence of retinal neovascularisation or fibrous tissue.

Proliferative diabetic retinopathy is characterised by retinal neovascularisation predominantly around the optic disc or can be seen as neovascularisation elsewhere (NVE). Occasionally fibrous tissue may be seen anterior to the retinal layer.

Vitreous haemorrhage is seen as a red haze of haemorrhage into the vitreous. Dot blot haemorrhages, neovascularisation or boat-shaped retinal tears may also be seen.

👍 👎 Discuss (1) Improve

Next question ›

Retinitis pigmentosa

Retinitis pigmentosa primarily affects the peripheral retina resulting in tunnel vision

Features
- night blindness is often the initial sign
- tunnel vision due to loss of the peripheral retina (occasionally referred to as funnel vision)
- fundoscopy: black bone spicule-shaped pigmentation in the peripheral retina, mottling of the retinal pigment epithelium

Associated diseases
- Refsum disease: cerebellar ataxia, peripheral neuropathy, deafness, ichthyosis
- Usher syndrome
- abetalipoproteinemia
- Lawrence-Moon-Biedl syndrome
- Kearns-Sayre syndrome
- Alport's syndrome

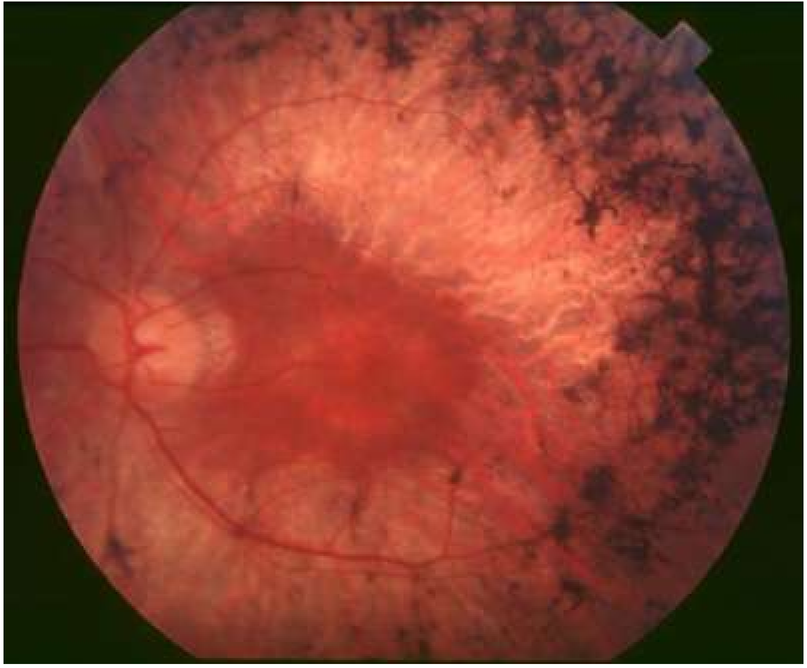

Image sourced from Wikipedia

Fundus showing changes secondary to retinitis pigmentosa

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **28%**

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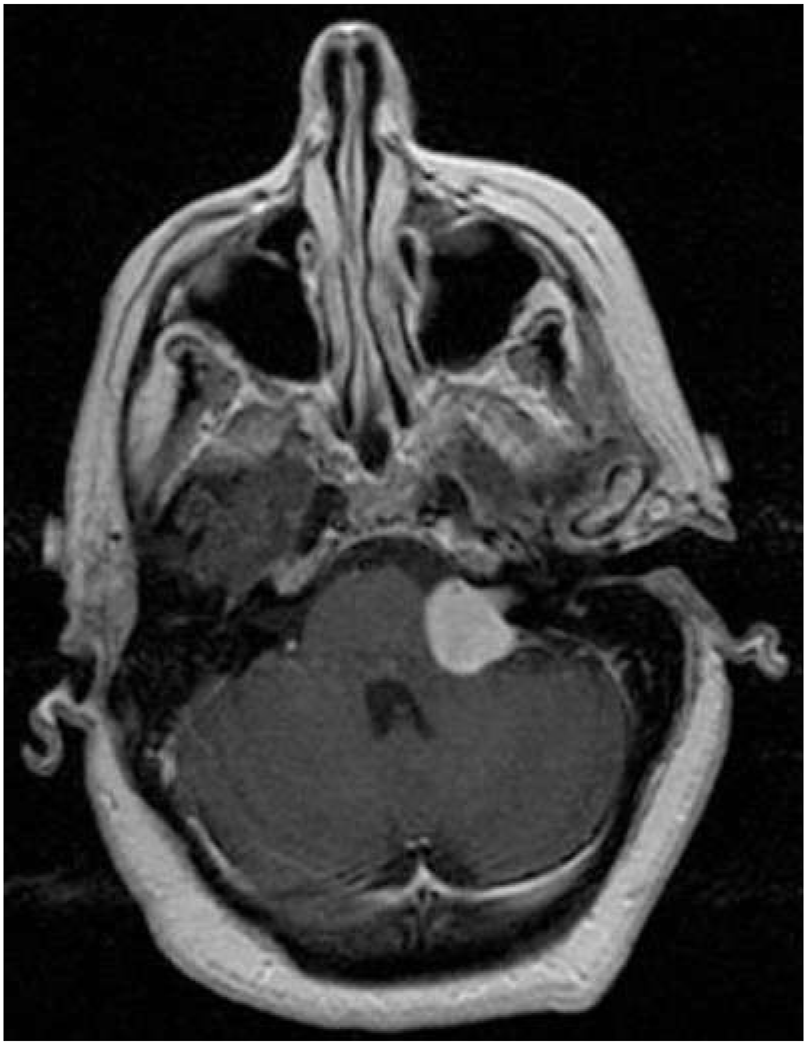
Back to top

A 55-year-old man presents with six weeks history of tinnitus. He has noted some changes in hearing lately. There is no record of any medical illness, and he does not take any regular medications. He is a chronic smoker and has 40 pack year history. His MRI brain showed:



© Image used on license from Radiopaedia

What is the next best step in his management?

- ○ Computed tomography (CT) of thorax
- ○ Intratympanic steroid therapy
- ○ Systemic steroid therapy
- ○ Urgent audiogram

○ Urgent referral to ENT ✗

Submit answer

Reference ranges ⌵

Score: **48%**

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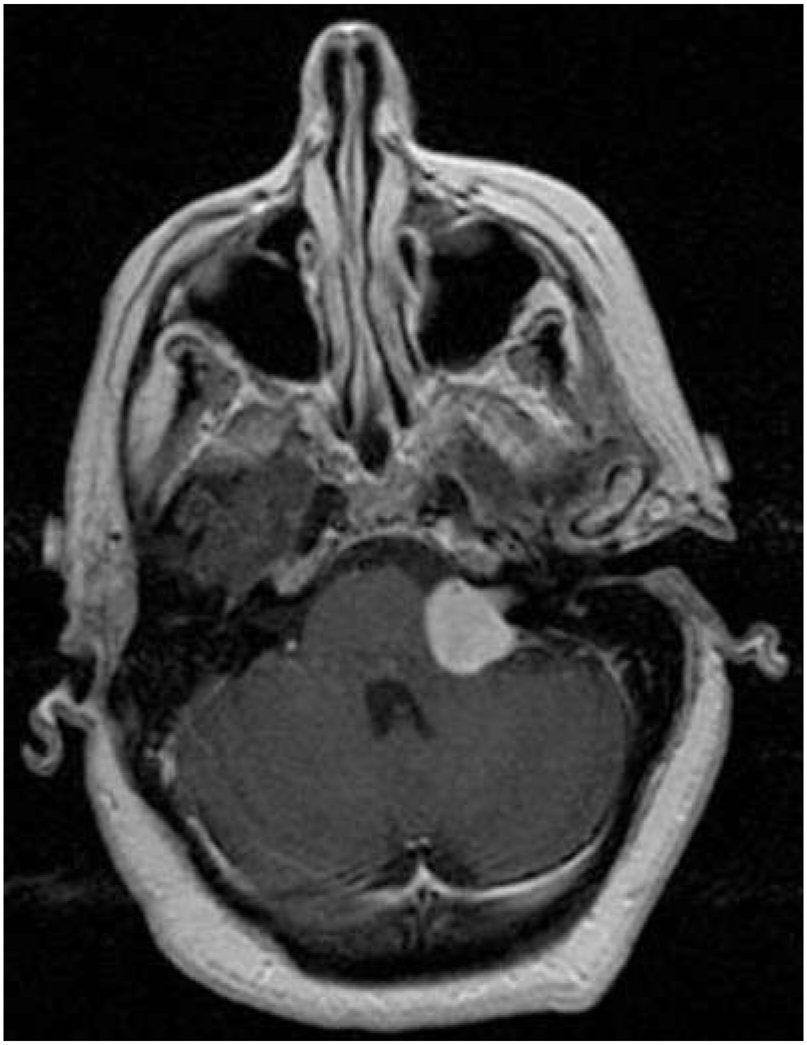


© Image used on license from Radiopaedia

What is the next best step in his management?

Computed tomography (CT) of thorax	8%
Intratympanic steroid therapy	2%
Systemic steroid therapy	8%
Urgent audiogram	8%

Urgent referral to ENT 74%

The MRI brain shows an intracanalicular solid nodular mass with an intense contrast enhancement in the right internal auditory meatus (IAM), which is suggestive of vestibular schwannoma.

Urgent referral to ENT is the correct answer. The patient's presentation and the MRI findings are in keeping with vestibular schwannoma, and the patient requires an urgent referral to ENT for further management.

Computed Tomography of thorax is incorrect. While a metastatic lesion of the cerebellopontine angle is a likely differential given the smoking history, the patient may benefit from a chest radiograph and/or a CT of the thorax to assess for lung neoplasms. However, the presentation and the MRI findings are characteristic of vestibular schwannoma, and the patient needs an urgent referral to ENT.

Intratympanic steroid therapy is incorrect. Intratympanic steroid injection treats vestibulocochlear symptoms of inner ear disease, such as Meniere's disease or idiopathic sudden sensorineural hearing loss. However, this patient's MRI suggests vestibular schwannoma as the likely cause for his hearing loss and requires urgent referral to ENT.

Systemic steroid therapy is incorrect. While systemic steroid therapy may be considered in treating sensorineural hearing loss associated with vestibular schwannoma, there is no adequate evidence to support its routine use in patients with vestibular schwannoma. The patient should be referred to ENT.

Urgent audiogram is incorrect. Patients with sensorineural hearing loss, tinnitus and vertigo should have an audiogram and MRI brain to assess for vestibular schwannoma. However, this patient's MRI brain is diagnostic of vestibular schwannoma and requires urgent ENT referral.

👍 👎 Discuss (1) Improve

Next question >

Vestibular schwannoma (acoustic neuroma) ★

Vestibular schwannomas (sometimes referred to as acoustic neuromas) account for approximately 5% of intracranial tumours and 90% of cerebellopontine angle tumours.

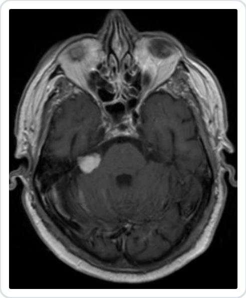
The classical history of vestibular schwannoma includes a combination of vertigo, hearing loss, tinnitus and an absent corneal reflex. Features can be predicted by the affected cranial nerves:

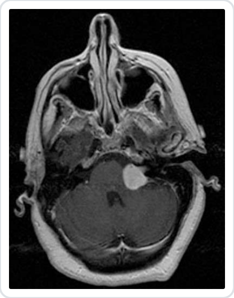
- cranial nerve VIII: vertigo, unilateral sensorineural hearing loss, unilateral tinnitus
- cranial nerve V: absent corneal reflex
- cranial nerve VII: facial palsy

Bilateral vestibular schwannomas are seen in neurofibromatosis type 2.

Patients with a suspected vestibular schwannoma should be referred urgently to ENT. It should be noted though that the tumours are often slow growing, benign and often observed initially.

MRI of the cerebellopontine angle is the investigation of choice. Audiometry is also important as only 5% of patients will have a normal audiogram.





Management is with either surgery, radiotherapy or observation.

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 17 👎 7

Acoustic neuroma

Suggest link Report broken link

Media

AUDITORY NERVE
UNILATERAL SYMPTOMS
CEREBELLO-PONTINE ANGLE
ACOUSTIC NEUROMAS
DIZZINESS
SCHWANN CELLS
FACIAL NUMBNESS

Understanding Acoustic Neuromas

Zero To Finals - YouTube 👍 0 👎 0

Report broken media

Score: **28%**

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Question 32 of 189

An 82-year-old man is referred to neurology clinic with slowness. He presented with his wife who reported that he has been becoming progressively slower in his movements and his facial expressions have become more limited. His symptoms have progressed rapidly following the onset of dizziness a few weeks ago. He has a past medical history of type two diabetes, hypertension, high cholesterol, previous hemicolectomy for diverticulitis. His current medications include ramipril, atorvastatin, paracetamol, amlodipine, metformin, prochlorperazine and gliclazide.

On examination, he has a coarse bilateral tremor at rest, and rigidity in both arms. He has a slow gait as well. What is the most likely diagnosis?

- ○ Parkinson's disease ×
- ○ Drug-induced parkinsonism ×
- ○ Multi-system atrophy ×
- ○ Vascular parkinsonism ×
- ○ Wilson's disease ×

Submit answer

Reference ranges

Score: **48%**

1 ✗
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On examination, he has a coarse bilateral tremor at rest, and rigidity in both arms. He has a slow gait as well. What is the most likely diagnosis?

Parkinson's disease	13%
Drug-induced parkinsonism	59%
Multi-system atrophy	13%
Vascular parkinsonism	14%
Wilson's disease	1%

The answer is drug-induced parkinsonism. There are several hints to why this is more likely. A bilateral tremor of rapid onset makes drug-induced parkinsonism a more probable cause when compared to Parkinson's disease. The recent dizziness suggests that prochlorperazine was only recently introduced, making it more suspicious that it may the cause of the current symptoms. Vascular parkinsonism would have a more step-wise progression with definitive days of significant deterioration, which are not described here. Multi-system atrophy generally has greater autonomic involvement, as well more cerebellar dysfunction. Wilson's disease typically occurs in younger patients, and is also assoaciated with liver dysfunction.

Source:

Chou, Kelvin L. 'Back Diagnosis and Differential Diagnosis of Parkinson Disease.' Ed. Howard I. Hurtig and John F. Dashe. UpToDate. 26 May 2016.

Next question >

Parkinson's disease: features

Parkinson's disease is a progressive neurodegenerative condition caused by degeneration of dopaminergic neurons in the substantia nigra. The reduction in dopaminergic output results in a classical triad of features: bradykinesia, tremor and rigidity. The symptoms of Parkinson's disease are characteristically asymmetrical.

Epidemiology
- around twice as common in men
- mean age of diagnosis is 65 years

Bradykinesia
- poverty of movement also seen, sometimes referred to as hypokinesia
- short, shuffling steps with reduced arm swinging
- difficulty in initiating movement

Tremor
- most marked at rest, 3-5 Hz
- worse when stressed or tired, improves with voluntary movement
- typically 'pill-rolling', i.e. in the thumb and index finger

Rigidity
- lead pipe
- cogwheel: due to superimposed tremor

Other characteristic features
- mask-like facies
- flexed posture
- micrographia
- drooling of saliva
- psychiatric features: depression is the most common feature (affects about 40%); dementia, psychosis and sleep disturbances may also occur
- impaired olfaction
- REM sleep behaviour disorder
- fatigue
- autonomic dysfunction:
    - postural hypotension

Drug-induced parkinsonism has slightly different features to Parkinson's disease:

- motor symptoms are generally rapid onset and bilateral
- rigidity and rest tremor are uncommon

Diagnosis is usually clinical. However, if there is difficulty differentiating between essential tremor and Parkinson's disease NICE recommend considering ^{123}I-FP-CIT single photon emission computed tomography (SPECT).

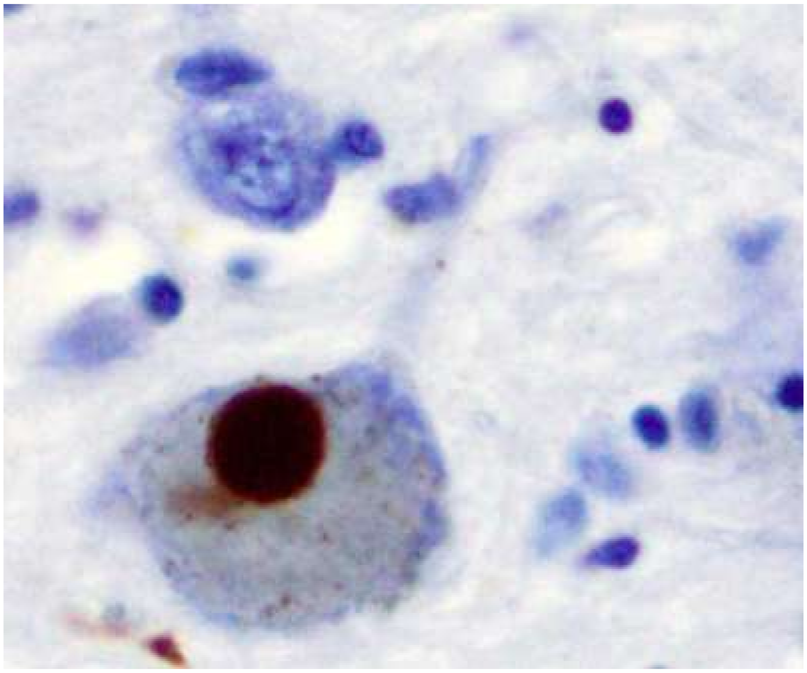


Image sourced from Wikipedia

A Lewy body (stained brown) in a brain cell of the substantia nigra in Parkinson's disease. The brown colour is positive immunohistochemistry staining for alpha-synuclein.

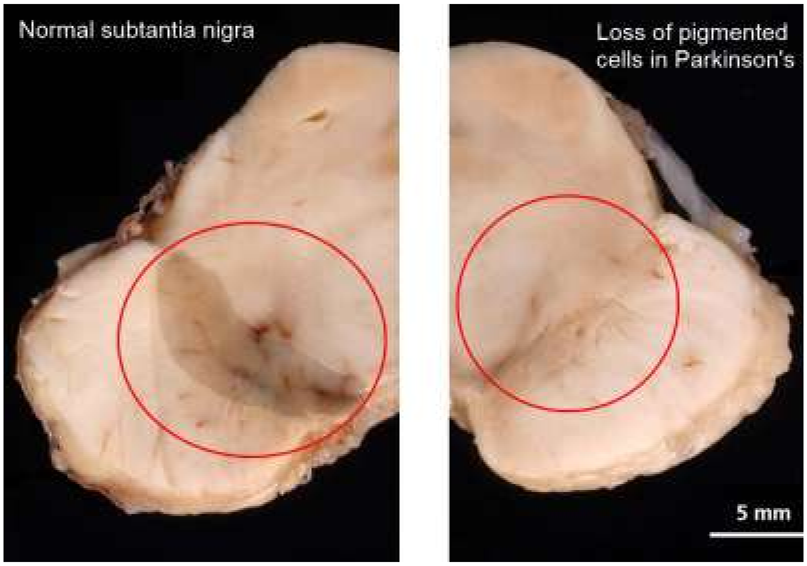


Image sourced from Wikipedia

Discoloration of the substantia nigra due to loss of pigmented nerve cells.

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 8 👎 9

2017 Parkinson's disease guidelines

Suggest link Report broken link

Media

Parkinsonian gait

mhdbelalalsabek - YouTube 👍 6 👎 0

PARKINSON'S
DISEASE
PATHOPHYSIOLOGY

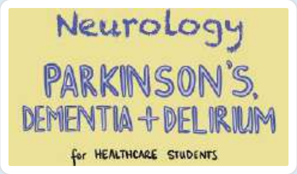
Parkinson's Disease: How is the brain affected?

Soton Brain Hub - YouTube 👍 1 👎 0

Parkinson's Disease Symptoms

[Parkinson's Disease Symptoms]

Soton Brain Hub - YouTube 👍 0 👎 0


Neurology
PARKINSON'S, DEMENTIA + DELIRIUM
for HEALTHCARE STUDENTS

[Parkinson's disease]

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PARKINSON'S DISEASE
Osmosis.org

[Parkinson's disease]

Osmosis - YouTube 👍 2 👎 1

PARKINSON'S DISEASE
SHUFFLING GAIT
α-SYNUCLEIN
PILL ROLLING TREMOR
COG-WHEEL RIGIDITY
DaT SCAN

[Understanding Parkinson's Disease]

Rhesus Medicine - YouTube 👍 1 👎 1

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A 28-year-old woman presents with a gradually worsening headache, nausea and vomiting.

The headache started about 3 days ago coming on over about 3-4 hours and is worse on coughing and lying flat. She has been complaining of some nausea and vomiting which she had attributed to some reheated curry she had the night before.

She is usually fit and well but did suffer a spontaneous below-knee deep vein thrombosis (DVT) 3 years ago which was treated with warfarin for 6 months. She has been taking over-the-counter paracetamol and ibuprofen for the headache and currently has a Mirena intrauterine coil in-situ for contraception. She has no known drug allergies.

She currently works as a sales associate. She smokes 10 cigarettes a day and drink 21 units of alcohol a week.

On examination she looks unwell, pale and nauseated. Her blood pressure was 140/98 mmHg, heart rate 100 bpm, oxygen saturations of 98% on air. Her temperature was 36.1 degrees Celsius.

Heart sounds were normal, and her calves soft and non-tender with no evidence of thromboembolism.

Her chest was clear and her abdominal examination was unremarkable.

On neurological examination there was no cranial nerve abnormalities noted. She had normal tone, power, reflexes and sensation in both her upper and lower limbs. Coordination was intact.

Bloods taken by the Emergency Department were as follows:

Na+	136 mmol/L
K+	3.9 mmol/L
Urea	4.8 mmol/L
Creatinine	76 µmol/L
Hb	12.5 g/dL
WBC	11.0 x 10^9/L
Platelets	350 x 10^9/L
INR	0.9
aPTT	30 seconds

LFTs	Normal

CT head was performed and reported as normal.

Following this a lumbar puncture was performed:

CSF colour	Clear
Opening pressure	250 mmH20
White cell count	5.0 x 10^6/L (all lymphocytes)
Red cell count	15 x 10^6/L
Protein	0.3 g/L
Glucose	4.4mmol/L (Serum glucose 5.3mmol/L)

What is the most likely diagnosis?

○ Intracranial venous thrombosis ×

○ Subarachnoid haemorrhage ×

○ Migraine ×

○ Intracerebral malignancy ×

○ Subdural empyema ×

Submit answer

Reference ranges ⌵

Score: **48%**

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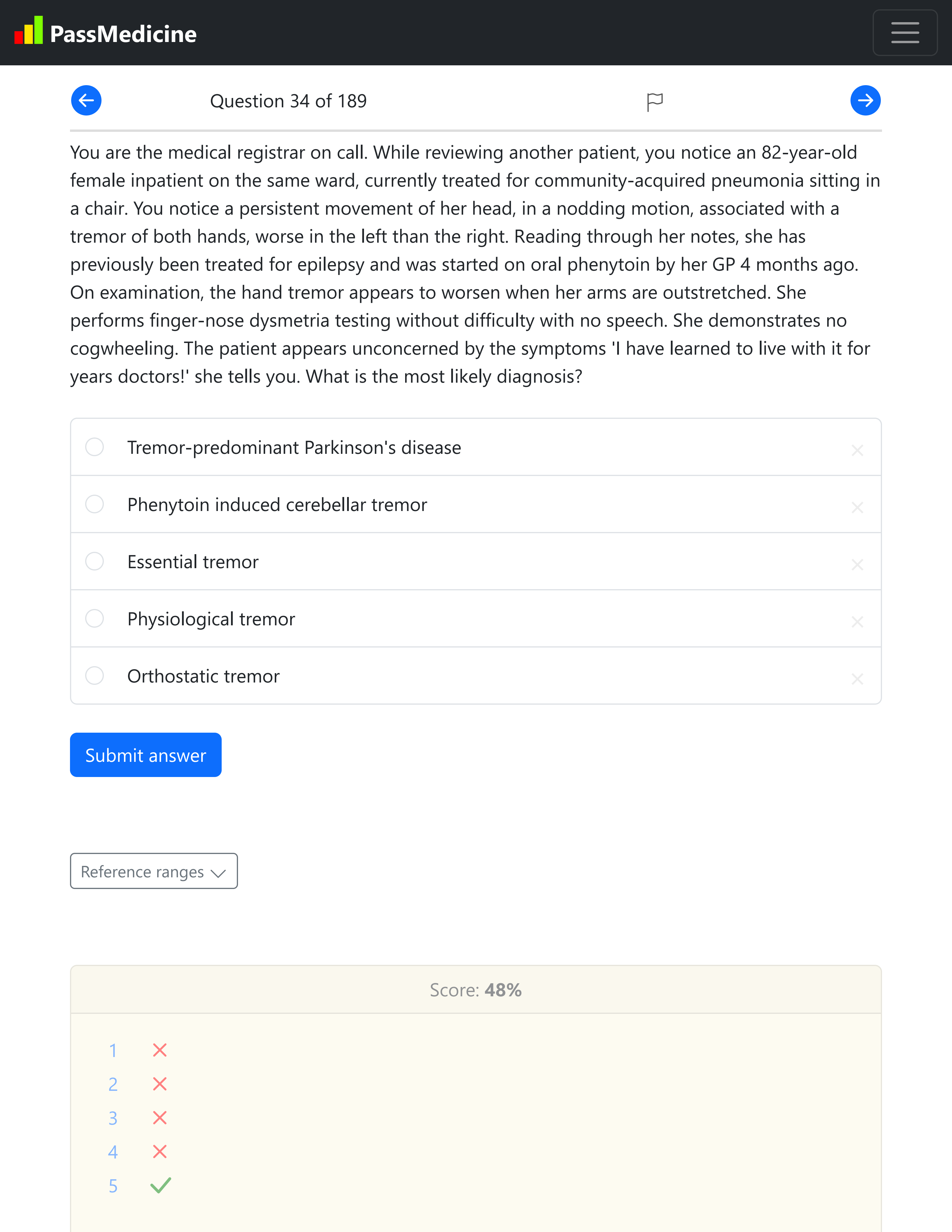

Question 34 of 189

You are the medical registrar on call. While reviewing another patient, you notice an 82-year-old female inpatient on the same ward, currently treated for community-acquired pneumonia sitting in a chair. You notice a persistent movement of her head, in a nodding motion, associated with a tremor of both hands, worse in the left than the right. Reading through her notes, she has previously been treated for epilepsy and was started on oral phenytoin by her GP 4 months ago. On examination, the hand tremor appears to worsen when her arms are outstretched. She performs finger-nose dysmetria testing without difficulty with no speech. She demonstrates no cogwheeling. The patient appears unconcerned by the symptoms 'I have learned to live with it for years doctors!' she tells you. What is the most likely diagnosis?

- ○ Tremor-predominant Parkinson's disease
- ○ Phenytoin induced cerebellar tremor
- ○ Essential tremor
- ○ Physiological tremor
- ○ Orthostatic tremor

Score: **48%**

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What is the most likely diagnosis?

Intracranial venous thrombosis	77%
Subarachnoid haemorrhage	7%
Migraine	8%
Intracerebral malignancy	4%
Subdural empyema	4%

This lady has had a previous venous thromboembolism and presents with a gradually worsening headache with multiple red flags. The history and examination is rather non-specific but it is the CSF opening pressure in the context of previous venous thrombus that most points towards the diagnosis of an intracranial venous thrombosis. This can present with a gradual or severe headache, signs of raised intracranial pressure and cranial nerve palsies.

Subarachnoid haemorrhage is unlikely in the context of the gradual onset of symptoms.

This patient has no previous history of migraine and the investigations are not consistent with this.

She is apyrexial and there is nothing in her presentation to indicate an intracerebral abscess, furthermore the CT is normal.

A normal CT also rules out intracerebral malignancy.

👍 👎 Discuss (10) Improve

Next question >

Intracranial venous thrombosis ★

Overview

- can cause cerebral infarction, much lesson common than arterial causes
- 50% of patients have isolated sagittal sinus thromboses - the remainder have coexistent lateral sinus thromboses and cavernous sinus thromboses

General features

Common features

- headache (may be sudden onset)
- nausea & vomiting
- reduced consciousness

Investigation

- MRI venography is the gold standard
  - CT venography is an alternative
- non-contrast CT head is normal in around 70% of patients
- D-dimer levels may be elevated

Management

- anticoagulation
  - typically with low molecular weight heparin acutely
  - warfarin is still generally used for longer term anticoagulation

Specific syndromes

Sagittal sinus thrombosis

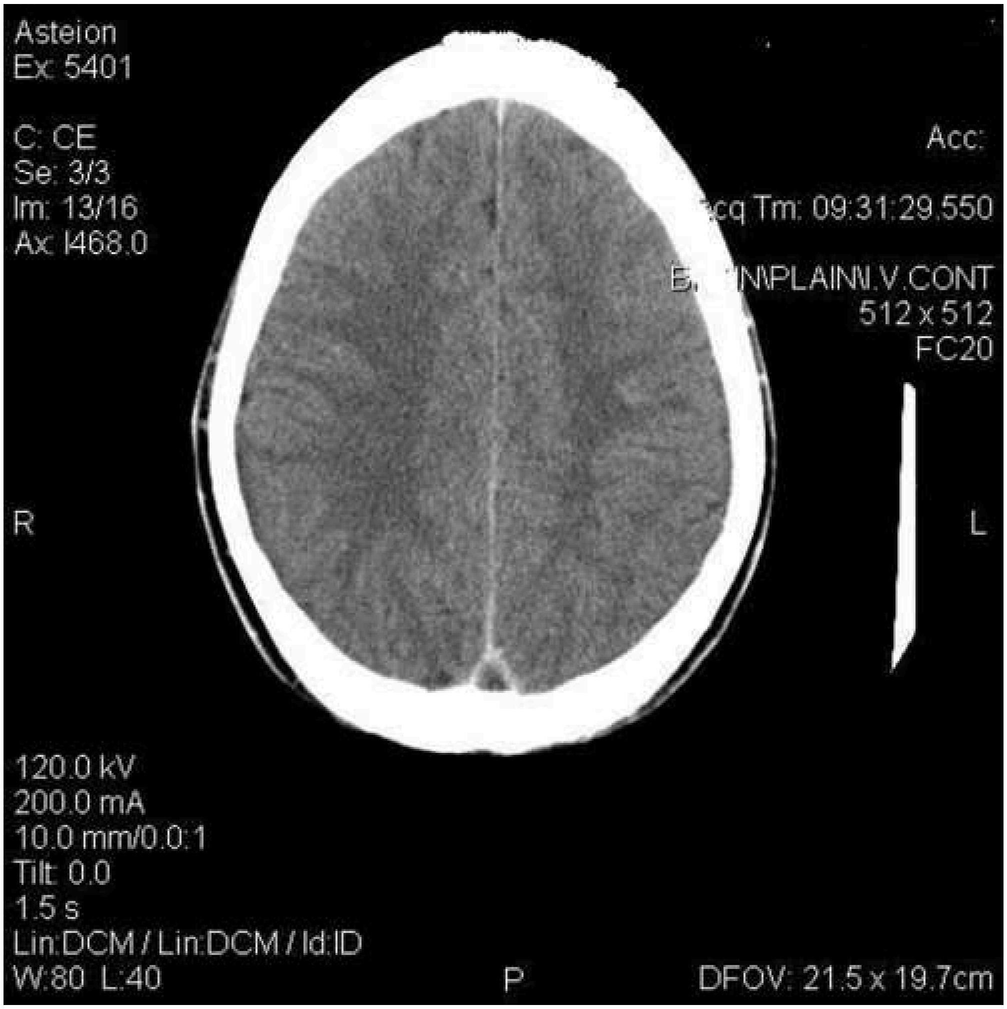
- may present with seizures and hemiplegia
- parasagittal biparietal or bifrontal haemorrhagic infarctions are sometimes seen
- 'empty delta sign' seen on venography

Cavernous sinus thrombosis

- other causes of cavernous sinus syndrome: local infection (e.g. sinusitis), neoplasia, trauma
- periorbital erythema and oedema
- ophthalmoplegia: 6th nerve damage typically occurs before 3rd & 4th
- trigeminal nerve involvement may lead to hyperaesthesia of upper face and eye pain
- central retinal vein thrombosis

Lateral sinus thrombosis

- 6th and 7th cranial nerve palsies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CT with contrast demonstrating a **superior sagittal sinus thrombosis** showing the typical empty delta sign. Look at the 'bottom' of the scan for the triangular shaped dural sinus. This should normally be white due to it being filled with contrast. The empty delta sign occurs when the thrombus fails to enhance within the dural sinus and is outlined by enhanced collateral channels in the falx. This sign is seen in only about 25%-30% of cases but is highly diagnostic for sagittal sinus thrombosis

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **28%**

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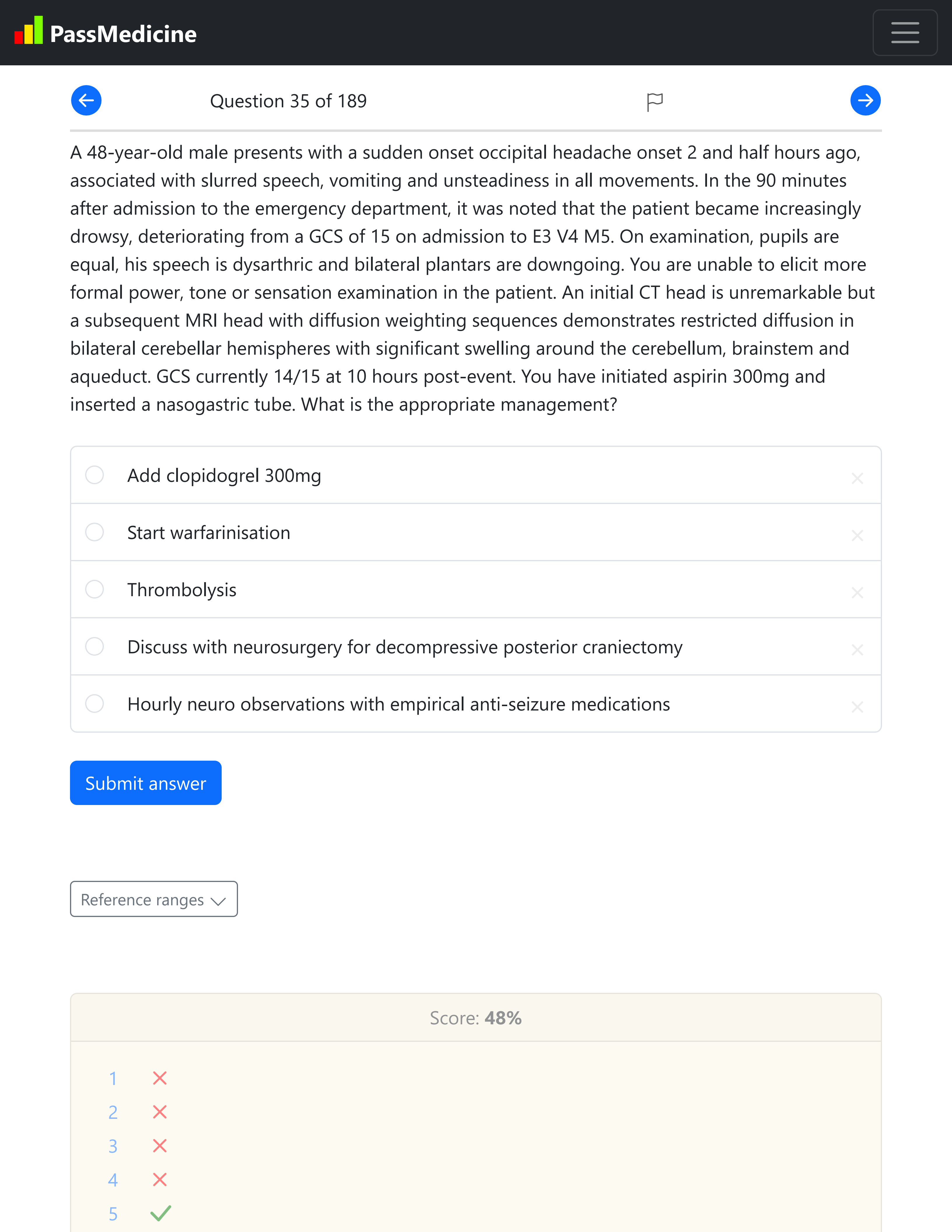

A 48-year-old male presents with a sudden onset occipital headache onset 2 and half hours ago, associated with slurred speech, vomiting and unsteadiness in all movements. In the 90 minutes after admission to the emergency department, it was noted that the patient became increasingly drowsy, deteriorating from a GCS of 15 on admission to E3 V4 M5. On examination, pupils are equal, his speech is dysarthric and bilateral plantars are downgoing. You are unable to elicit more formal power, tone or sensation examination in the patient. An initial CT head is unremarkable but a subsequent MRI head with diffusion weighting sequences demonstrates restricted diffusion in bilateral cerebellar hemispheres with significant swelling around the cerebellum, brainstem and aqueduct. GCS currently 14/15 at 10 hours post-event. You have initiated aspirin 300mg and inserted a nasogastric tube. What is the appropriate management?

- ○ Add clopidogrel 300mg
- ○ Start warfarinisation
- ○ Thrombolysis
- ○ Discuss with neurosurgery for decompressive posterior craniectomy
- ○ Hourly neuro observations with empirical anti-seizure medications

Score: **48%**

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Tremor-predominant Parkinson's disease	5%
Phenytoin induced cerebellar tremor	16%
Essential tremor	75%
Physiological tremor	3%
Orthostatic tremor	1%

The patient's examination is one of isolated tremor with no other cerebellar or neurological features. The clinical features of worsening on posture, head nodding and lack of cerebellar symptoms suggest a diagnosis of essential tremor. She demonstrates no signs of Parkinson's disease, a tremor that does not alleviate with movement and the history describes onset beyond the start of phenytoin. Orthostatic tremor onsets in the trunk and legs when the patient stands. The described features are perhaps too severe for a physiological tremor, which is typically low in amplitude and barely visible. It is unlikely the patient would have received a beta agonist for a community-acquired pneumonia in the absence of obstructive airways disease, hence a drug-induced tremor is also unlikely.

Discuss (4) Improve

Next question

Essential tremor

Essential tremor (previously called benign essential tremor) is an autosomal dominant condition which usually affects both upper limbs

Features
- postural tremor: worse if arms outstretched
- improved by alcohol and rest
- most common cause of titubation (head tremor)

Management
- propranolol is first-line
- primidone is sometimes used

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

HEAD TREMOR
WORSE WITH CAFFEINE
DIFFERENTIALS?
ESSENTIAL TREMOR
BETTER WITH ALCOHOL
FINE TREMOR
SYMMETRICAL

Essential tremor

Zero To Finals - YouTube 👍 1 👎 0

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Score: **28%**

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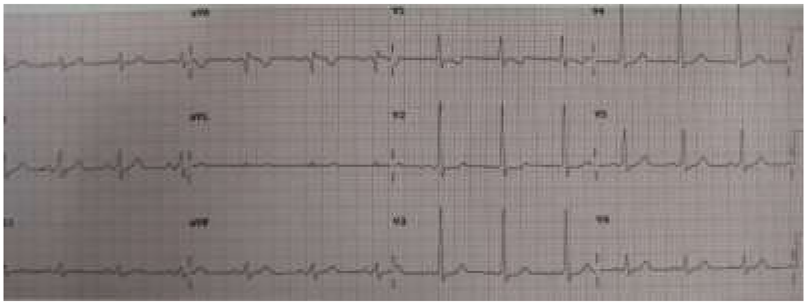
A 14-year-old boy presents his third generalised seizure over the past 72 hours, despite recently being started on sodium valproate by a neurologist for recurrent seizures 6 weeks ago, with worsening vision at night and hearing loss bilaterally. The patient has a number of myoclonic jerks as you arrive. On examination, his heart sounds are unremarkable but you notice a tachycardia at 140 and regular. The ECG is shown below:



© Image used on license from Dr Smith, University of Minnesota

The patient is uncooperative to further neurological examination but you notice sluggishly reactive pupils of equal size. His mother reports that he has been educated in a special needs school for the past 5 years but had been attending the local primary school until aged 9, when he dropped further behind than his peers. Which investigation produces the underlying diagnosis?

○ MRI

○ Lumbar puncture

○ Electroencephalogram (EEG)

○ Cardiac electrophysiology studies

○ Muscle biopsy

Submit answer

Reference ranges

Score: **48%**

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Add clopidogrel 300mg	4%
Start warfarinisation	1%
Thrombolysis	15%
Discuss with neurosurgery for decompressive posterior craniectomy	74%
Hourly neuro observations with empirical anti-seizure medications	6%

This patient is critically unwell. He has presented with a posterior circulation infarct significant oedema around a very tight part of the neuro-anatomy. Although pupils are currently equal and reactive, the drop in GCS is a major concern. It may not be possible to illicit upper motor neurone signs in the hyperacute phase after a stroke. The most concerning complication at this stage is a progression towards obstructive hydrocephalus from CSF flow blockage at the aqueduct or 3rd or 4th ventricle. Prophylactic posterior craniectomy must be considered in such a young patient, discussion with neurosurgery is essential.

Discuss (10) | Improve

Next question >

Stroke: management of acute ischaemic stroke

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
  - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
  - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:

- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.

- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
  - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
  - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue

- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 11 👎 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 15

Stroke and TIA guidelines

Age and Ageing 👍 20 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

2023 National Clinical Guideline for Stroke

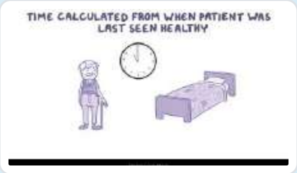
NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link

Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia
Acute Ischaemic Stroke

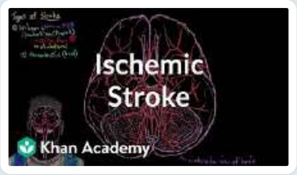
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
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Iscchaemic stroke

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Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

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Report broken media

Score: **28%**

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Question 37 of 189

A 61-year-old male presents with his sixth episode of binocular visual phenomenon over the past 14 months. He describes these episodes as 'lights' and 'white dots' in his field of vision. He denies any limb or facial weakness or sensory loss. He denies having a headache. He is an active smoker, with a 60 pack year smoking history and has known hypertension on ramipril 5mg OD. Your neurological exam is unremarkable; CT head demonstrates no acute infarct or haemorrhage. MRI head is unremarkable.

What is the most likely diagnosis?

- ○ Posterior circulation transient ischaemic attacks (TIAs)
- ○ Posterior circulation strokes
- ○ Acephalgic migraine
- ○ Multiple sclerosis
- ○ Occipital space occupying lesion

Submit answer

Reference ranges

Score: **48%**

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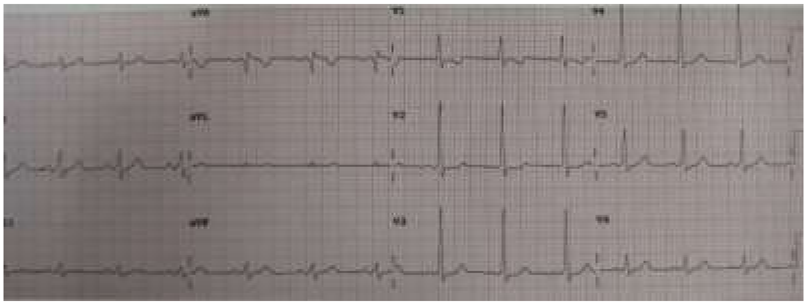
A 14-year-old boy presents his third generalised seizure over the past 72 hours, despite recently being started on sodium valproate by a neurologist for recurrent seizures 6 weeks ago, with worsening vision at night and hearing loss bilaterally. The patient has a number of myoclonic jerks as you arrive. On examination, his heart sounds are unremarkable but you notice a tachycardia at 140 and regular. The ECG is shown below:



© Image used on license from Dr Smith, University of Minnesota

The patient is uncooperative to further neurological examination but you notice sluggishly reactive pupils of equal size. His mother reports that he has been educated in a special needs school for the past 5 years but had been attending the local primary school until aged 9, when he dropped further behind than his peers. Which investigation produces the underlying diagnosis?

MRI	19%
Lumbar puncture	2%
Electroencephalogram (EEG)	12%
Cardiac electrophysiology studies	7%
Muscle biopsy	60%

The patient is a young male presenting with cognitive impairment developing after a period of normal development, seizures, myoclonic jerks, Wolff-Parkinson-White syndrome and worsening vision (consistent with optic atrophy). The unifying diagnosis is that of myoclonic epilepsy with ragged red fibres (MERRF), which is a mitochondrial DNA disorder diagnosed by ragged red fibres on muscle biopsy.

Discuss (12) | Improve

Next question ›

Mitochondrial diseases

Whilst most DNA is found in the cell nucleus, a small amount of double-stranded DNA is present in the mitochondria. It encodes protein components of the respiratory chain and some special types of RNA

Mitochondrial inheritance has the following characteristics:

- inheritance is only via the maternal line as the sperm contributes no cytoplasm to the zygote
- none of the children of an affected male will inherit the disease
- all of the children of an affected female will inherit the disease
- generally, encode rare neurological diseases
- poor genotype:phenotype correlation - within a tissue or cell there can be different mitochondrial populations - this is known as heteroplasmy

Histology

- muscle biopsy classically shows 'red, ragged fibres' due to increased number of mitochondria

Examples include:

- Leber's optic atrophy
    - symptoms typically develop at around the age of 30 years
    - central scotoma → loss of colour vision → rapid onset of significant visual impairment
- MELAS syndrome: mitochondrial encephalomyopathy lactic acidosis and stroke-like episodes
- MERRF syndrome: myoclonus epilepsy with ragged-red fibres
- Kearns-Sayre syndrome: onset in patients < 20 years old, external ophthalmoplegia, retinitis pigmentosa. Ptosis may be seen
- sensorineural hearing loss

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 5 👎 6

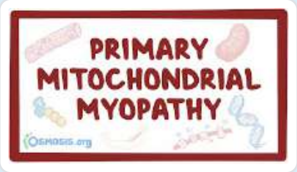
2013 How to spot mitochondrial disease in adults

Rare Disease Video Library - NORD 👍 3 👎 4

Primary Mitochondrial Myopathy

Suggest link Report broken link

Media


PRIMARY
MITOCHONDRIAL
MYOPATHY
Osmosis.org

Primary mitochondrial myopathy

Osmosis - YouTube 👍 6 👎 1

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Score: **28%**

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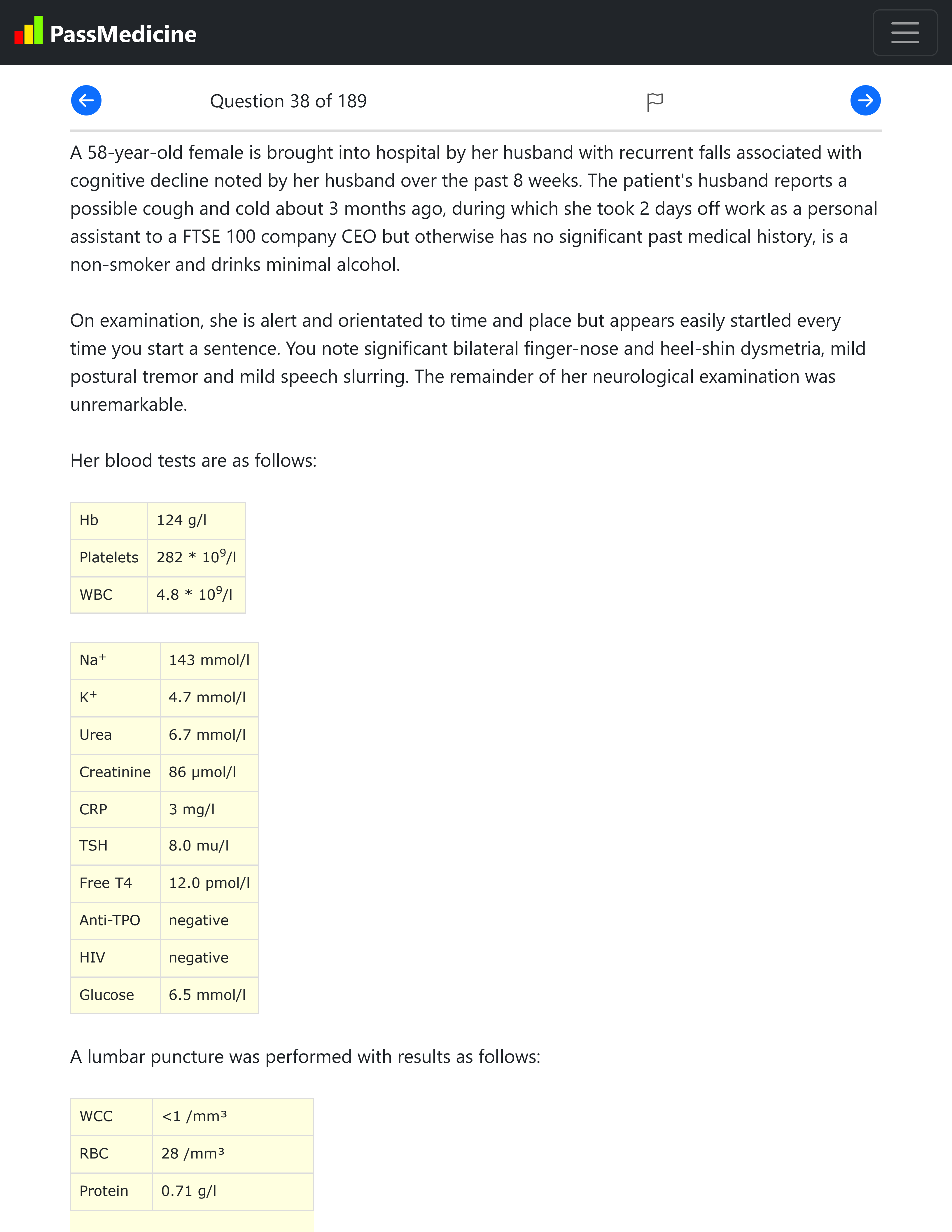

A 58-year-old female is brought into hospital by her husband with recurrent falls associated with cognitive decline noted by her husband over the past 8 weeks. The patient's husband reports a possible cough and cold about 3 months ago, during which she took 2 days off work as a personal assistant to a FTSE 100 company CEO but otherwise has no significant past medical history, is a non-smoker and drinks minimal alcohol.

On examination, she is alert and orientated to time and place but appears easily startled every time you start a sentence. You note significant bilateral finger-nose and heel-shin dysmetria, mild postural tremor and mild speech slurring. The remainder of her neurological examination was unremarkable.

Her blood tests are as follows:

Hb	124 g/l
Platelets	282 * 10^9/l
WBC	4.8 * 10^9/l

Na^+	143 mmol/l
K^+	4.7 mmol/l
Urea	6.7 mmol/l
Creatinine	86 µmol/l
CRP	3 mg/l
TSH	8.0 mu/l
Free T4	12.0 pmol/l
Anti-TPO	negative
HIV	negative
Glucose	6.5 mmol/l

A lumbar puncture was performed with results as follows:

WCC	<1 /mm³
RBC	28 /mm³
Protein	0.71 g/l

Glucose	3.4 mmol/l
Culture	no organisms grown
Viral PCR	awaited
Cytology	awaited
14-3-3	positive

An EEG demonstrated brief periodic spikes but these were not correlated with any seizure activity clinically. An MRI head demonstrated no parenchymal abnormalities except mildly increased signal in the cortical sulci.

What is the most likely diagnosis?

- ○ Creutzfeldt-Jakob disease ×
- ○ Viral encephalitis ×
- ○ Acute disseminated encephalomyelitis (ADEM) ×
- ○ Progressive multifocal leucoencephalopathy (PML) ×
- ○ Hashimoto's encephalopathy ×

Submit answer

Reference ranges ⌵

Score: **48%**

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A 61-year-old male presents with his sixth episode of binocular visual phenomenon over the past 14 months. He describes these episodes as 'lights' and 'white dots' in his field of vision. He denies any limb or facial weakness or sensory loss. He denies having a headache. He is an active smoker, with a 60 pack year smoking history and has known hypertension on ramipril 5mg OD. Your neurological exam is unremarkable; CT head demonstrates no acute infarct or haemorrhage. MRI head is unremarkable.

What is the most likely diagnosis?

Posterior circulation transient ischaemic attacks (TIAs)	29%
Posterior circulation strokes	3%
Acephalgic migraine	62%
Multiple sclerosis	4%
Occipital space occupying lesion	2%

The patient gives a strong history of positive visual symptoms such as flashing lights and dots, with no negative such as weakness or sensory loss. This would be far more suggestive of a migrainous syndrome or primary retinal pathology than cerebrovascular insufficiency, where negative symptoms would be prominent. In addition, a diagnosis of TIA or stroke should be suspicious in any patient with repeated stereotyped, similar symptoms over a long period of time. It is unlikely any patient will present with recurrent multiple TIAs without stroke over a 14 month period.

Acephalgic migraine is a frequently missed diagnosis of exclusion, where patients experience auras without a headache. Up to 38% of migraineurs experience migraine attacks without and with a headache, while 4% of migraineurs exclusively experience migraines without headaches[1].

1. Russell MB, Olesen J. A nosographic analysis of the migraine aura in a general population. Brain. 1996;119 (Pt 2):355.

Discuss (3) | Improve

Next question >

Migraine: diagnostic criteria

The International Headache Society has produced the following diagnostic criteria for migraine without aura:

Point	Criteria
A	At least 5 attacks fulfilling criteria B-D
B	Headache attacks lasting 4-72 hours* (untreated or unsuccessfully treated)
C	Headache has at least two of the following characteristics: • 1. unilateral location* • 2. pulsating quality (i.e., varying with the heartbeat) • 3. moderate or severe pain intensity • 4. aggravation by or causing avoidance of routine physical activity (e.g., walking or climbing stairs)
D	During headache at least one of the following: • 1. nausea and/or vomiting* • 2. photophobia and phonophobia
E	Not attributed to another disorder (history and examination do not suggest a secondary headache disorder or, if they do, it is ruled out by appropriate investigations or headache attacks do not occur for the first time in close temporal relation to the other disorder)

*In children, attacks may be shorter-lasting, headache is more commonly bilateral, and gastrointestinal disturbance is more prominent.

Migraine with aura (seen in around 25% of migraine patients) tends to be easier to diagnose with a typical aura being progressive in nature and may occur hours prior to the headache. Typical aura include a transient hemianopic disturbance or a spreading scintillating scotoma ('jagged crescent'). Sensory symptoms may also occur

If we compare these guidelines to the **NICE criteria** the following points are noted:

- NICE suggests migraines may be unilateral or bilateral
- NICE also give more detail about typical auras:

Auras may occur with or without headache and:
- are fully reversible
- develop over at least 5 minutes
- last 5-60 minutes

The following aura symptoms are atypical and may prompt further investigation/referral;
- motor weakness
- double vision
- visual symptoms affecting only one eye
- poor balance
- decreased level of consciousness.

AI + Q 123

Next question >

B *I*

Textbooks

High-yield textbook

Extended textbook

Links

International Headache Society 👍 7 👎 5

2019 The International Classification of Headache Disorders

Suggest link

Report broken link

Media

Management of Migraine
Podmedics

Migraine

Podmedics - YouTube 👍 4 👎 0

Migraine

Townsend Teaching - YouTube 👍 0 👎 0

Report broken media

Score: **28%**

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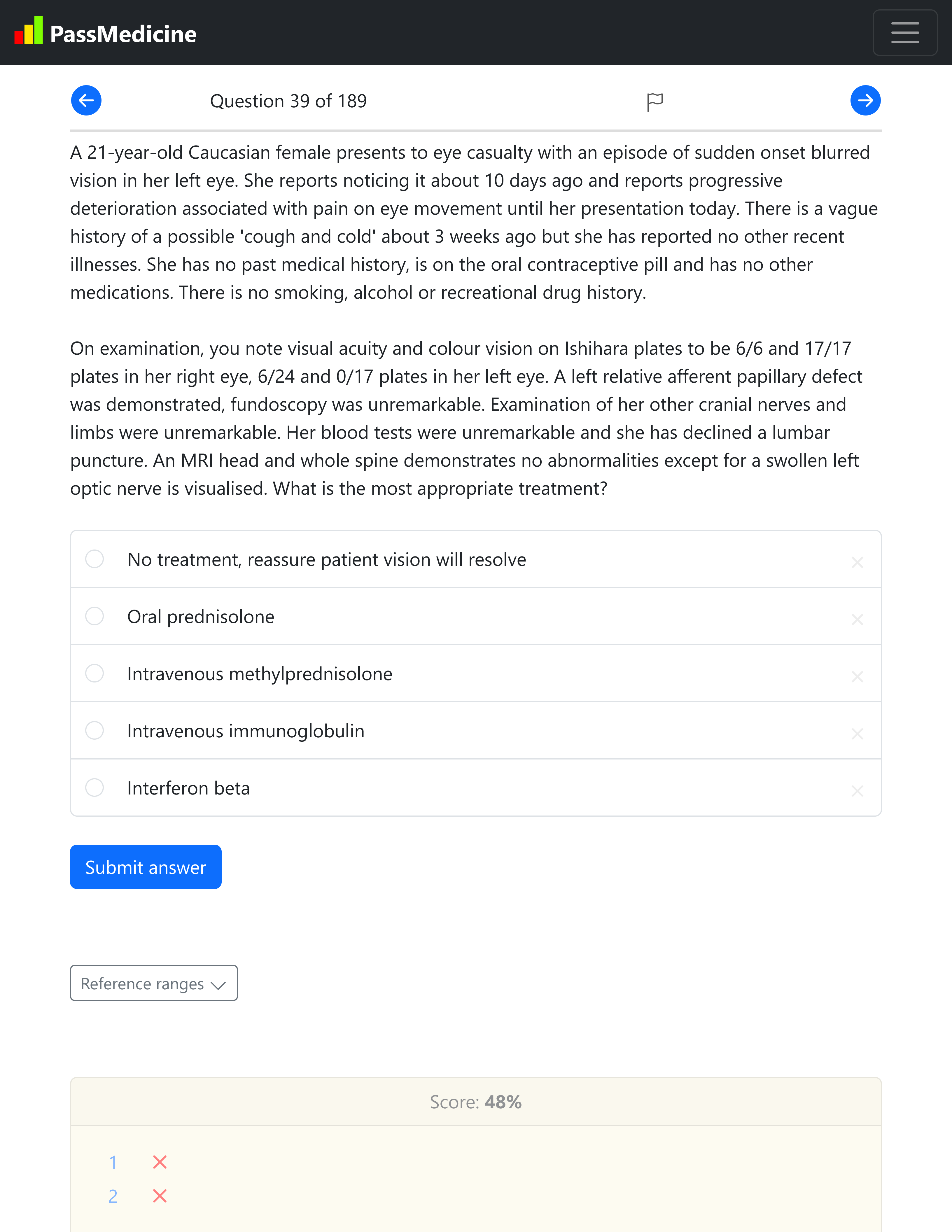

A 21-year-old Caucasian female presents to eye casualty with an episode of sudden onset blurred vision in her left eye. She reports noticing it about 10 days ago and reports progressive deterioration associated with pain on eye movement until her presentation today. There is a vague history of a possible 'cough and cold' about 3 weeks ago but she has reported no other recent illnesses. She has no past medical history, is on the oral contraceptive pill and has no other medications. There is no smoking, alcohol or recreational drug history.

On examination, you note visual acuity and colour vision on Ishihara plates to be 6/6 and 17/17 plates in her right eye, 6/24 and 0/17 plates in her left eye. A left relative afferent papillary defect was demonstrated, fundoscopy was unremarkable. Examination of her other cranial nerves and limbs were unremarkable. Her blood tests were unremarkable and she has declined a lumbar puncture. An MRI head and whole spine demonstrates no abnormalities except for a swollen left optic nerve is visualised. What is the most appropriate treatment?

- ○ No treatment, reassure patient vision will resolve
- ○ Oral prednisolone
- ○ Intravenous methylprednisolone
- ○ Intravenous immunoglobulin
- ○ Interferon beta

Score: **48%**

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A 58-year-old female is brought into hospital by her husband with recurrent falls associated with cognitive decline noted by her husband over the past 8 weeks. The patient's husband reports a possible cough and cold about 3 months ago, during which she took 2 days off work as a personal assistant to a FTSE 100 company CEO but otherwise has no significant past medical history, is a non-smoker and drinks minimal alcohol.

On examination, she is alert and orientated to time and place but appears easily startled every time you start a sentence. You note significant bilateral finger-nose and heel-shin dysmetria, mild postural tremor and mild speech slurring. The remainder of her neurological examination was unremarkable.

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Glucose	3.4 mmol/l
Culture	no organisms grown
Viral PCR	awaited
Cytology	awaited
14-3-3	positive

An EEG demonstrated brief periodic spikes but these were not correlated with any seizure activity clinically. An MRI head demonstrated no parenchymal abnormalities except mildly increased signal in the cortical sulci.

What is the most likely diagnosis?

Creutzfeldt-Jakob disease	68%
Viral encephalitis	5%
Acute disseminated encephalomyelitis (ADEM)	16%
Progressive multifocal leucoencephalopathy (PML)	8%
Hashimoto's encephalopathy	4%

This middle aged female demonstrates a number of worrying features suggestive of Creutzfeld Jakob disease (CJD): rapid cognitive decline, myoclonus, extrapyramidal signs (ataxia), startle response, positive 14-3-3 on CSF and periodic spiking on EEG. The MRI findings likely represent 'cortical ribboning', with high signal on the cortical sulci surfaces, which along with increased signal in putamen and caudate head, is suggestive of CJD.

She is also in the expected age group for CJD, typically presenting between 57 and 62 years old.

A number of features in the history and investigations are red herrings: it is unusual for a viral encephalitis, related to a probable viral respiratory tract infection, to be indolent for 8 weeks and continuing to progress in a non-immunosuppressed patient. The MRI findings and time course are also not suggestive of ADEM, which produces many focal areas of central demyelination within 1 to 3 weeks of a viral illness.

PML results in central demyelinating and is caused by activation of JC papovavirus in immunosuppressed patients, typically HIV positive individuals, and hence unexpected in this patient. The raised TSH likely represents subclinical hypothyroidism in this patient: while Hashimoto's encephalopathy can produce personality change, aggressive and disorientation, myoclonus and ataxia, 14-3-3 is typically negative in CSF with positive anti-TPO antibodies. A diagnosis of CJD is thus most likely diagnosis, with the patient progressing along a typically fatal clinical course.

Discuss (6) | Improve

Next question

Creutzfeldt-Jakob disease

Creutzfeldt-Jakob disease (CJD) is rapidly progressive neurological condition caused by prion proteins. These proteins induce the formation of amyloid folds resulting in tightly packed beta-pleated sheets resistant to proteases.

Features

- dementia (rapid onset)
- myoclonus

Investigation

- CSF is usually normal
- EEG: biphasic, high amplitude sharp waves (only in sporadic CJD)
- MRI: hyperintense signals in the basal ganglia and thalamus

Sporadic CJD

- accounts for 85% of cases
- 10-15% of cases are familial
- mean age of onset is 65 years

New variant CJD

- younger patients (average age of onset = 25 years)
- psychological symptoms such as anxiety, withdrawal and dysphonia are the most common presenting features
- the 'prion protein' is encoded on chromosome 20 - it's role is not yet understood
- methionine homozygosity at codon 129 of the prion protein is a risk factor for developing CJD - all patients who have so far died have had this
- median survival = 13 months

Other prion diseases

- kuru

- fatal familial insomnia
- Gerstmann Straussler-Scheinker disease

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

PRIONS
(SPONGIFORM
ENCEPHALOPATHY)

Prions (Spongiform encephalopathy)

Osmosis - YouTube 👍 7 👎 0

Report broken media

Score: **28%**

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Question 40 of 189

A 32-year-old female patient presents with 48 hours of new onset vertiginous symptoms and speech slurring. She has no past medical history and is a non-smoker. On examination, you notice a left partial ptosis and miosis. Finger-nose coordination is impaired with her left arm and she also has reduced sensation to your cold tuning fork on her left face, right arm and right leg. What is the most likely diagnosis?

- ○ Bilateral cerebellar infarcts
- ○ Right middle cerebral artery ischaemic infarct
- ○ Left middle cerebral artery ischaemic infarct
- ○ Lateral medullar brainstem infarct
- ○ Left basal ganglia haemorrhage

Submit answer

Reference ranges

Score: **48%**

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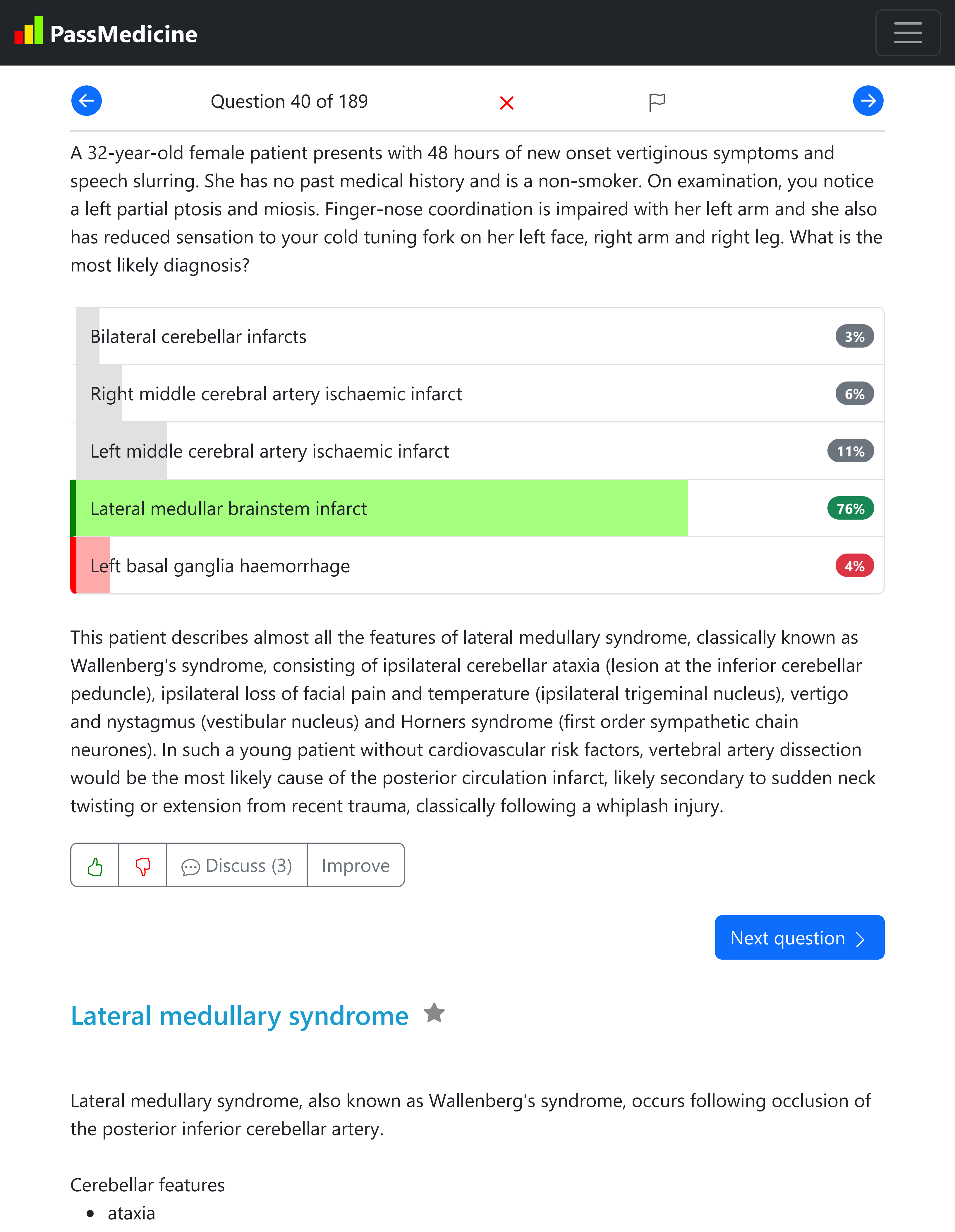

Question 40 of 189

A 32-year-old female patient presents with 48 hours of new onset vertiginous symptoms and speech slurring. She has no past medical history and is a non-smoker. On examination, you notice a left partial ptosis and miosis. Finger-nose coordination is impaired with her left arm and she also has reduced sensation to your cold tuning fork on her left face, right arm and right leg. What is the most likely diagnosis?

Bilateral cerebellar infarcts 3%

Right middle cerebral artery ischaemic infarct 6%

Left middle cerebral artery ischaemic infarct 11%

Lateral medullar brainstem infarct 76%

Left basal ganglia haemorrhage 4%

This patient describes almost all the features of lateral medullary syndrome, classically known as Wallenberg's syndrome, consisting of ipsilateral cerebellar ataxia (lesion at the inferior cerebellar peduncle), ipsilateral loss of facial pain and temperature (ipsilateral trigeminal nucleus), vertigo and nystagmus (vestibular nucleus) and Horners syndrome (first order sympathetic chain neurones). In such a young patient without cardiovascular risk factors, vertebral artery dissection would be the most likely cause of the posterior circulation infarct, likely secondary to sudden neck twisting or extension from recent trauma, classically following a whiplash injury.

Lateral medullary syndrome

Lateral medullary syndrome, also known as Wallenberg's syndrome, occurs following occlusion of the posterior inferior cerebellar artery.

Cerebellar features

- ataxia

- nystagmus

Brainstem features

- ipsilateral: dysphagia, facial numbness, cranial nerve palsy e.g. Horner's
- contralateral: limb sensory loss

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

Brainstem Stroke

Brainstem stroke syndromes

Soton Brain Hub - YouTube 👍 6 👎 0

Report broken media

Score: **28%**

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A 16-year-old male is referred to your clinic after reporting feeling increasingly tired at school over the past 2 months. His parents are similarly concerned that their son seems to be relying on large volumes of Lucozade to keep himself alert during this stressful period of secondary school examinations. He reports no headaches. The patient was born following a normal pregnancy and development until he was 12 years old, when he underwent chemotherapy and radiotherapy treatment for an optic chiasm glioma, forcing him to miss one academic year of school. On completion of his treatment, he returned to school and has been achieving good grades. There is no other past medical or family history.

On examination, the patient is short for his age (at 2nd centile) and you note an absence of facial hair or other secondary sexual characteristics. He is thin with a BMI of 13.7 kg/m². Chest, cardiovascular and abdominal examinations are unremarkable. There are no skin lesions. Neurological examination including cranial nerve is unremarkable.
His initial blood tests are as follows:

Hb	142 g/l
MCV	89 fl
Platelets	410 * 10^9/l
WBC	7.4 * 10^9/l
Na^+	139 mmol/l
K^+	4.6 mmol/l
Urea	5.1 mmol/l
Creatinine	44µmol/l
FSH	low
ACTH	low
TSH	0.13 mu/l

What is the most likely diagnosis?

○ Cranial radiation injury

○ Glioma recurrence in optic chiasm

○ Klinefelter's syndrome

○ Turner's syndrome ×

○ Haemochromatosis ×

Submit answer

Reference ranges ⌵

Score: **48%**

1	✗
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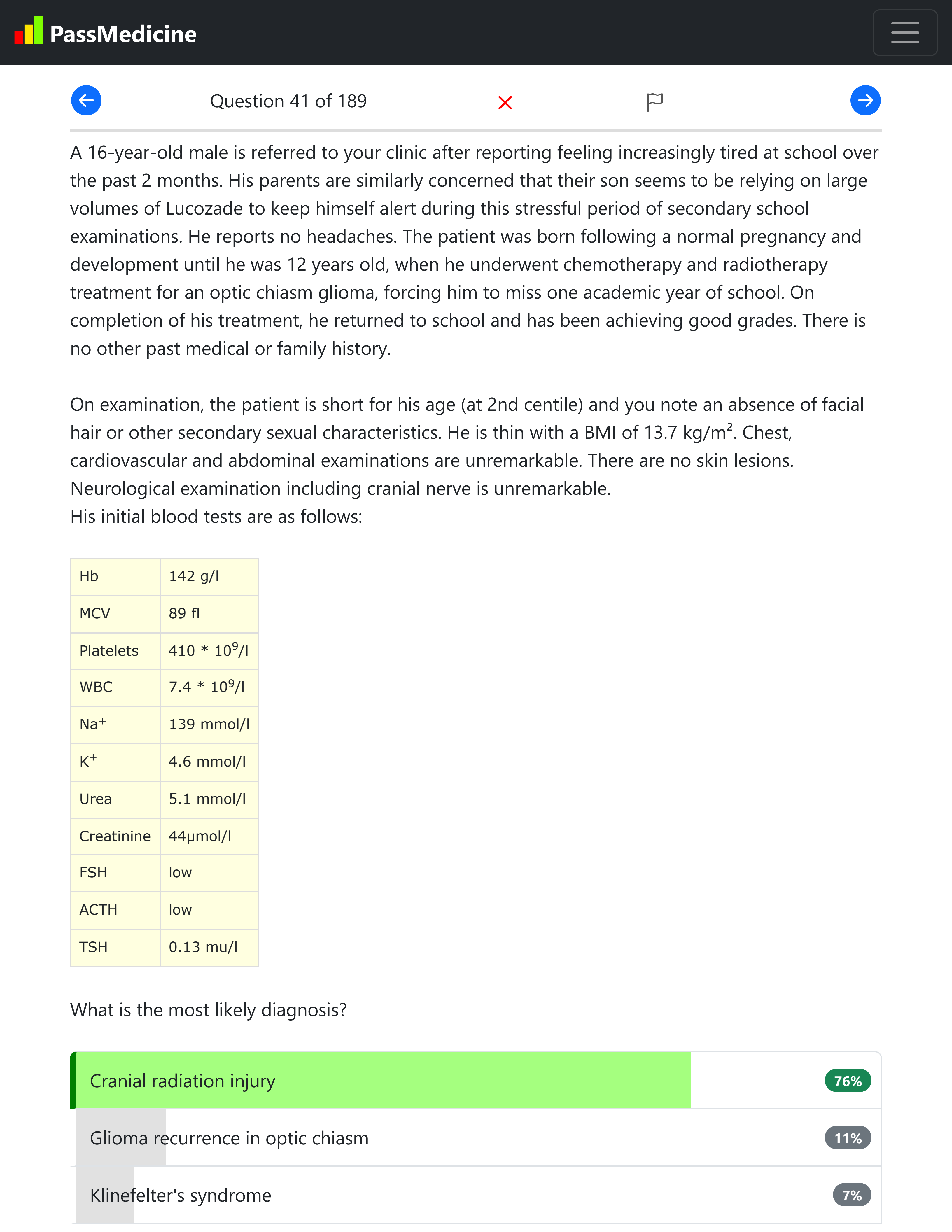

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Na^+	139 mmol/l
K^+	4.6 mmol/l
Urea	5.1 mmol/l
Creatinine	44μmol/l
FSH	low
ACTH	low
TSH	0.13 mu/l

What is the most likely diagnosis?

Cranial radiation injury 76%

Glioma recurrence in optic chiasm 11%

Klinefelter's syndrome 7%

Turner's syndrome 4%

Haemochromatosis 1%

A combination of clinical features and pituitary function tests reveal panhypopituitarism. Delayed puberty does not appear to be gonadal but instead central in cause. While haemochromatosis may be a cause of panhypopituitarism, other features such as skin pigmentation, liver cirrhosis, arthralgia and type 1 diabetes mellitus would be expected. Turner's syndrome appears in females only due to an XO karyotype. Klinefelter's can cause delayed puberty secondary to a failure of Leydig cell development but would be associated typically with clinical features such as hypothyroidism, diabetes mellitus, with increased pituitary hormone release in response to gonadal dysfunction.

The location of the optic chiasm adjacent to the hypothalamic-pituitary structures and previous medical treatment should raise suspicion of cranial radiation injury, common in childhood survivors of intracranial cancers treated with radiotherapy. There is little to suggest recurrence of the tumour, which would typically present with proptosis and visual disturbance.

👍 👎 Discuss (4) Improve

Next question >

Brain tumours

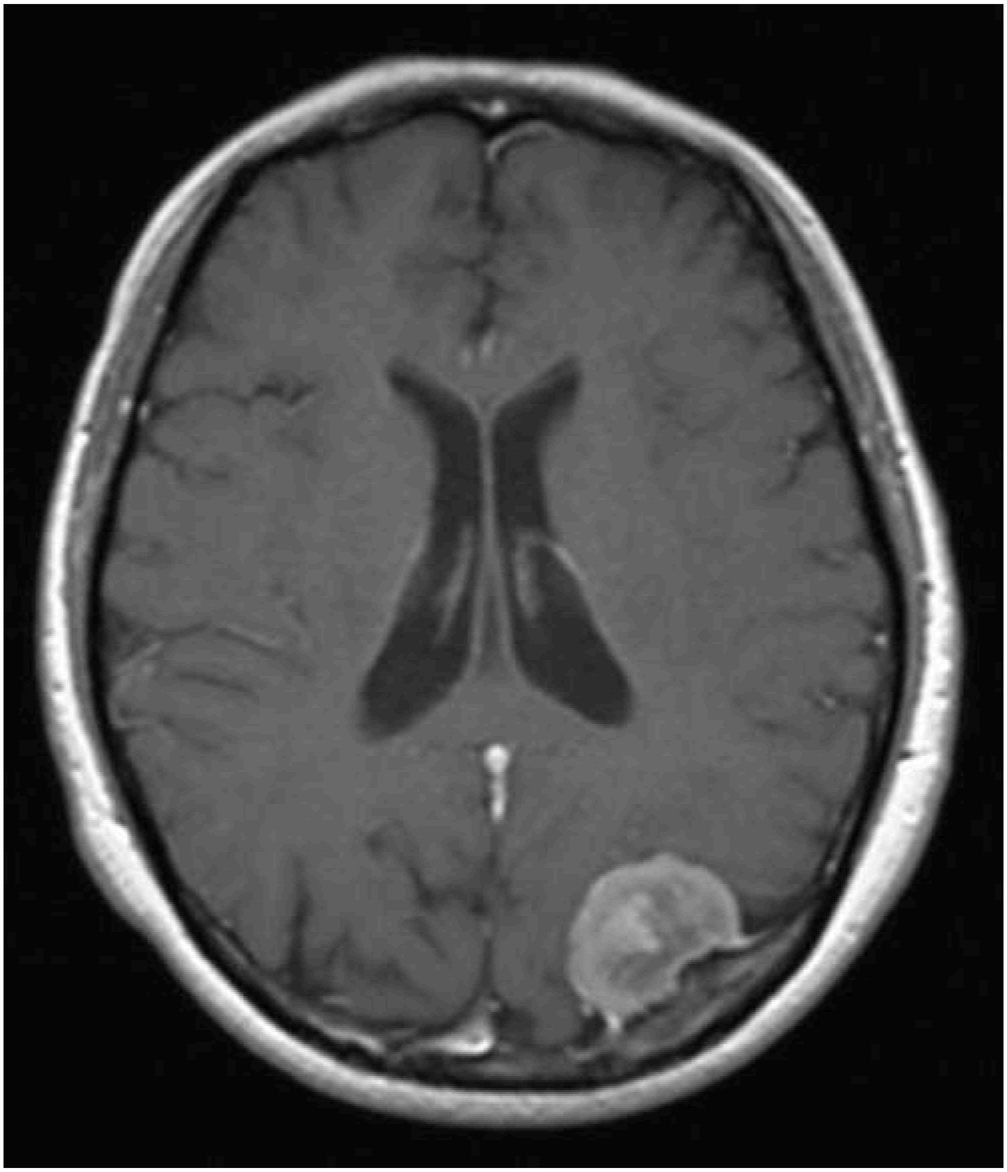
The majority of adult tumours are supratentorial, where as the majority of childhood tumours are infratentorial.

Type of tumour	**Features**
Metastases	Metastatic brain cancer is the most common form of brain tumours. They are often multiple and not treatable with surgical intervention. Tumours that most commonly spread to the brain include: • lung (most common) • breast • bowel • skin (namely melanoma) • kidney

Type of tumour	Features
Glioblastoma multiforme	• Glioblastoma is the most common primary tumour in adults and is associated with a poor prognosis (~ 1yr). • On imaging they are solid tumours with central necrosis and a rim that enhances with contrast. Disruption of the blood-brain barrier and therefore are associated with vasogenic oedema. • Histology: Pleomorphic tumour cells border necrotic areas • Treatment is surgical with postoperative chemotherapy and/or radiotherapy. Dexamethasone is used to treat the oedema.
Meningioma	• The second most common primary brain tumour in adults • Meningiomas are typically benign, extrinsic tumours of the central nervous system. They arise from the arachnoid cap cells of the meninges and are typically located next to the dura and cause symptoms by compression rather than invasion. • They typically are located at the falx cerebri, superior sagittal sinus, convexity or skull base. • Histology: Spindle cells in concentric whorls and calcified psammoma bodies • Investigation is with CT (will show contrast enhancement) and MRI, and treatment will involve either observation, radiotherapy or surgical resection.
Vestibular schwannoma	• A vestibular schwannoma (previously termed acoustic neuroma) is a benign tumour arising from the eighth cranial nerve (vestibulocochlear nerve). Often seen in the cerebellopontine angle. It presents with hearing loss, facial nerve palsy (due to compression of the nearby facial nerve) and tinnitus. • Neurofibromatosis type 2 is associated with bilateral vestibular schwannomas. • Histology: Antoni A or B patterns are seen. Verocay bodies (acellular areas surrounded by nuclear palisades) • Treatment may involve observation, radiotherapy or surgery.

Type of tumour	**Features**
Pilocytic astrocytoma	• The most common primary brain tumour in children • Histology: Rosenthal fibres (corkscrew eosinophilic bundle)
Medulloblastoma	• A medulloblastoma is an aggressive paediatric brain tumour that arises within the infratentorial compartment. It spreads through the CSF system. Treatment is surgical resection and chemotherapy. • Histology: Small, blue cells. Rosette pattern of cells with many mitotic figures
Ependymoma	• Commonly seen in the 4th ventricle • May cause hydrocephalus • Histology: perivascular pseudorosettes
Oligodendroma	• Benign, slow-growing tumour common in the frontal lobes • Histology: Calcifications with 'fried-egg' appearance
Haemangioblastoma	• Vascular tumour of the cerebellum • Associated with von Hippel-Lindau syndrome • Histology: foam cells and high vascularity
Pituitary adenoma	• Pituitary adenomas are benign tumours of the pituitary gland. They are either secretory (producing a hormone in excess) or non-secretory. They may be divided into microadenomas (smaller than 1cm) or macroadenoma (larger than 1cm). • Patients will present with the consequences of hormone excess (e.g. Cushing's due to ACTH, or acromegaly due to GH) or depletion. Compression of the optic chiasm will cause a bitemporal hemianopia due to the crossing nasal fibers. • Investigation requires a pituitary blood profile and MRI. Treatment can either be hormonal or surgical (e.g. transphenoidal resection).
Craniopharyngioma	• Most common paediatric supratentorial tumour • A craniopharyngioma is a solid/cystic tumour of the sellar region that is derived from the remnants of Rathke's pouch. It is common in children, but can present in adults also. It may present with hormonal disturbance, symptoms of hydrocephalus or bitemporal hemianopia. • Histology: Derived from remnants of Rathke pouch

Type of tumour	Features
	• Investigation requires pituitary blood profile and MRI. Treatment is typically surgical with or without postoperative radiotherapy.



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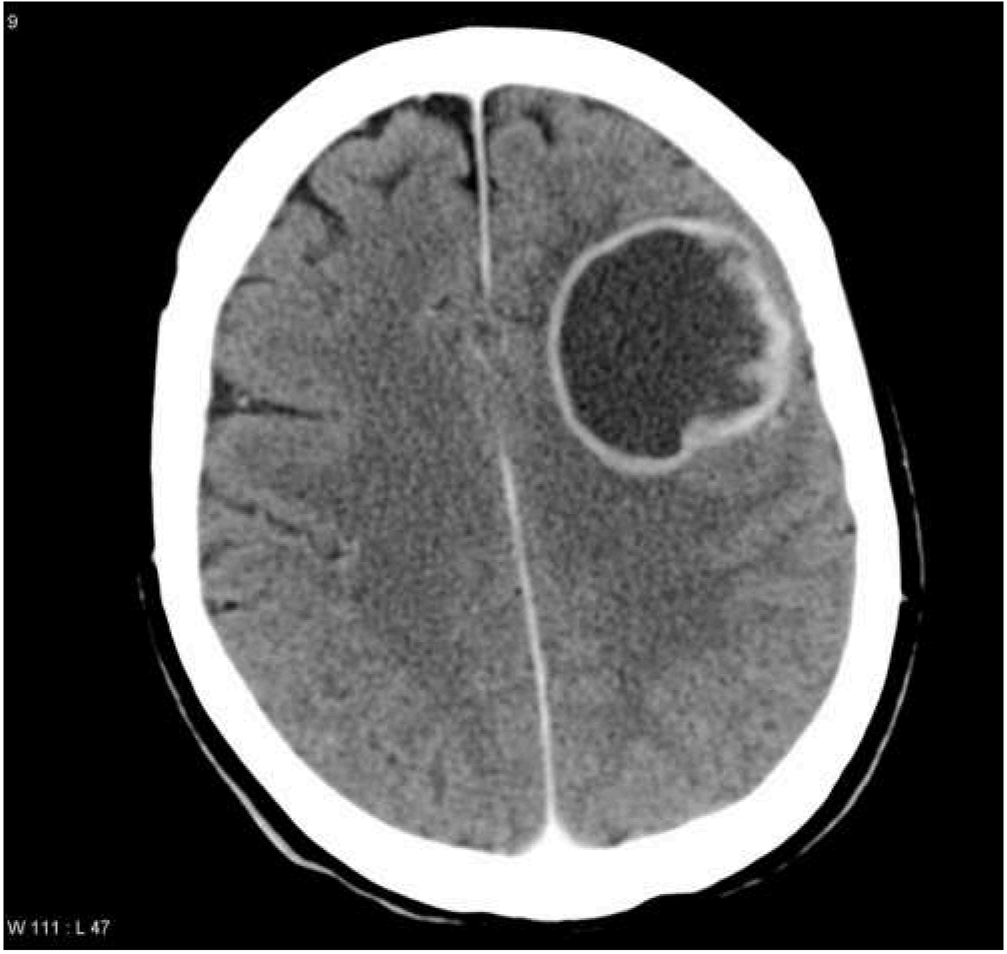
Meningioma - MRI showing the typical well-circumscribed appearance. A dural tail can be where the tumour 'connects' to the dura. It is seen in around 65% of meningiomas.



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Glioblastoma multiforme - CT showing a peripherally enhancing lesion within the left frontal lobe. Note the contrast to the more homogenous meningioma above.

+ Q 123

Next question >

Textbooks

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Extended textbook

Media

ADULT
BRAIN TUMORS
Osmosis.org

Adult brain tumours

Osmosis - YouTube 👍 4 👎 0

PEDIATRIC
BRAIN TUMORS
Osmosis.org

Paediatric brain tumours

Osmosis - YouTube 👍 2 👎 1

GLIOBLASTOMA
Osmosis
KNOWLEDGE SHOT

Glioblastoma

Osmosis - YouTube 👍 2 👎 1

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Score: **28%**

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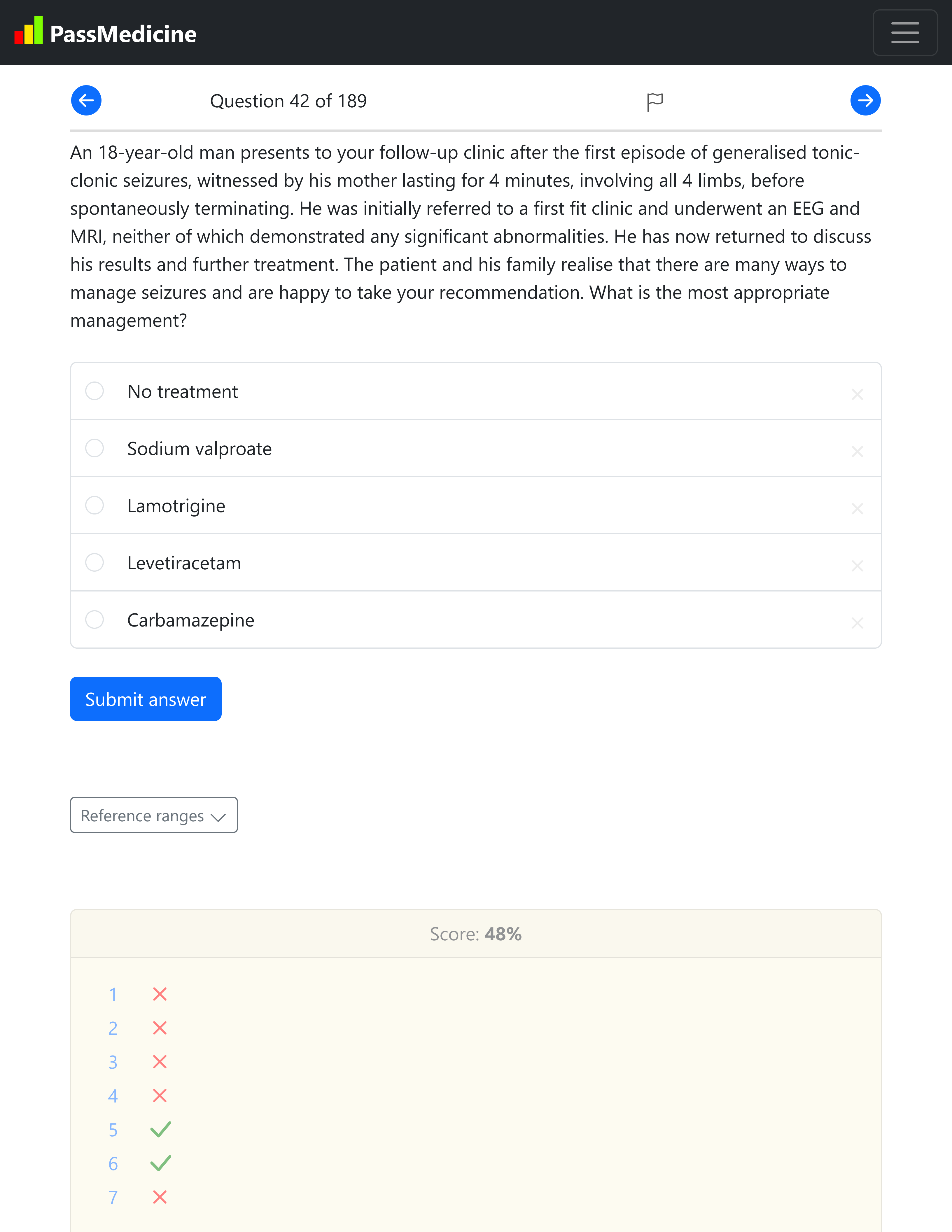

Question 42 of 189

An 18-year-old man presents to your follow-up clinic after the first episode of generalised tonic-clonic seizures, witnessed by his mother lasting for 4 minutes, involving all 4 limbs, before spontaneously terminating. He was initially referred to a first fit clinic and underwent an EEG and MRI, neither of which demonstrated any significant abnormalities. He has now returned to discuss his results and further treatment. The patient and his family realise that there are many ways to manage seizures and are happy to take your recommendation. What is the most appropriate management?

- ○ No treatment
- ○ Sodium valproate
- ○ Lamotrigine
- ○ Levetiracetam
- ○ Carbamazepine

Score: **48%**

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Question 42 of 189 ✕

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No treatment	66%
Sodium valproate	25%
Lamotrigine	2%
Levetiracetam	6%
Carbamazepine	1%

Treatment is not usually recommended after a single seizure, as this may have been a provoked seizure instead of the first presentation of epilepsy. One prospective, but methodologically flawed, study demonstrated a 14% risk of seizure recurrent after a single, unprovoked seizure at 1 year[1]. However, the risks significant increase after a patient has had two seizures, rising to 73% recurrent rate at 4 years[2]. It is therefore accepted that antiepileptic treatments should only be commenced after 2 seizures, except if the risks of a further seizure is unacceptable to the patient, an underlying structural defect is proven to be the likely cause of the seizure or if the EEG demonstrated epileptiform activity.

1. Hausea WA, Rich SS, Annegers JF et al. Seizure recurrent after a 1st unprovoked seizure: an extended follow-up. Neurology 1990; 40(8):1163
2. Hauser WA, Rich SS, Lee JR et al. Risk of recurrent seizures after two unprovoked seizures. NEJM 1998; 338 (7):429

Discuss (2) Improve

Next question ›

Epilepsy: treatment ★

Most neurologists now start antiepileptics following a second epileptic seizure. NICE guidelines suggest starting antiepileptics after the first seizure if any of the following are present:

- the patient has a neurological deficit
- brain imaging shows a structural abnormality
- the EEG shows unequivocal epileptic activity
- the patient or their family or carers consider the risk of having a further seizure unacceptable

It should be remembered that the following are only general guidelines. For example, **maternal use of sodium valproate is associated with a significant risk of neurodevelopmental delay in children**. Guidance is now clear that sodium valproate should not be used during pregnancy and in women of childbearing age unless clearly necessary.

Drug treatment according to the type of epilepsy

Generalised tonic-clonic seizures

- males: sodium valproate
- females: lamotrigine or levetiracetam
- girls aged under 10 years and who are unlikely to need treatment when they are old enough to have children or women who are unable to have children may be offered sodium valproate first-line

Focal seizures

- first line: lamotrigine or levetiracetam
- second line: carbamazepine, oxcarbazepine or zonisamide

Absence seizures (Petit mal)

- first line: ethosuximide
- second line:
  - male: sodium valproate
  - female: lamotrigine or levetiracetam
- carbamazepine may exacerbate absence seizures

Myoclonic seizures

- males: sodium valproate
- females: levetiracetam

Tonic or atonic seizures

- males: sodium valproate
- females: lamotrigine

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 28 31

2022 Epilepsy guidelines

Royal College of Physicians 15 16

2013 Modern management of epilepsy

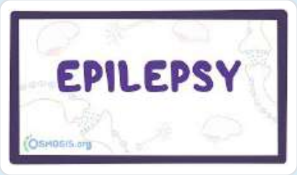
Suggest link

Report broken link

Media

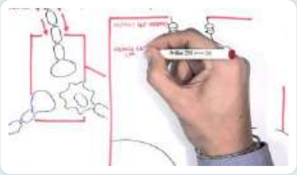
Antiepileptic drugs made easy

Speed Pharmacology - YouTube 👍 3 👎 0



Epilepsy (generalized, focal) - simple, complex, tonic, clonic, causes, symptoms

Osmosis - YouTube 👍 3 👎 0



Pharmacology - Antiepileptics

Armando Hasudungan - YouTube 👍 3 👎 1

Report broken media

Score: **28%**

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Question 43 of 189

A 16-year-old boy is brought into the emergency department by his mother after an episode of unresponsiveness while walking, lasting transiently for seconds before fully resolving. He has no recollection of these episodes. He was diagnosed with absence seizures aged 11 and was previously prescribed anti-epileptics. These had been gradually weaned off two years ago by his neurologists following a 3-year seizure-free period.

Over the past 48 hours, he has now had 4 of his typical absences. You decide to prescribe an anti-epileptic. Which antiepileptic should be avoided?

- ○ Carbamazepine ×
- ○ Sodium valproate ×
- ○ Lamotrigine ×
- ○ Levetiracetam ×
- ○ No contraindications ×

Submit answer

Reference ranges

Score: **48%**

1 ✗
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Over the past 48 hours, he has now had 4 of his typical absences. You decide to prescribe an anti-epileptic. Which antiepileptic should be avoided?

Carbamazepine	64%
Sodium valproate	19%
Lamotrigine	6%
Levetiracetam	4%
No contraindications	7%

Classically, carbamazepine can aggravate juvenile myoclonic epilepsy and absence seizures[1]. In general, carbamazepine should always be avoided in patients where the type of seizure is uncertain. It is otherwise a useful AED in the treatment of partial or secondary generalised seizures.

1. WD Shields, Saslow E. Myoclonic, atonic, absence seizures following institution of carbamazepine therapy in children. Neurology 1983; 33:1487

Discuss (2) Improve

Next question

Absence seizures

Absence seizures (petit mal) are a form of generalised epilepsy that is mostly seen in children. The typical age of onset of 3-10 years old and girls are affected twice as commonly as boys

Features

- absences last a few seconds and are associated with a quick recovery
- seizures may be provoked by hyperventilation or stress
- the child is usually unaware of the seizure
- they may occur many times a day
- EEG: bilateral, symmetrical 3Hz spike and wave pattern

Management

- sodium valproate and ethosuximide are first-line treatment
- good prognosis - 90-95% become seizure free in adolescence

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 9 👎 5

2012 Epilepsy guidelines

Suggest link

Report broken link

Score: **28%**

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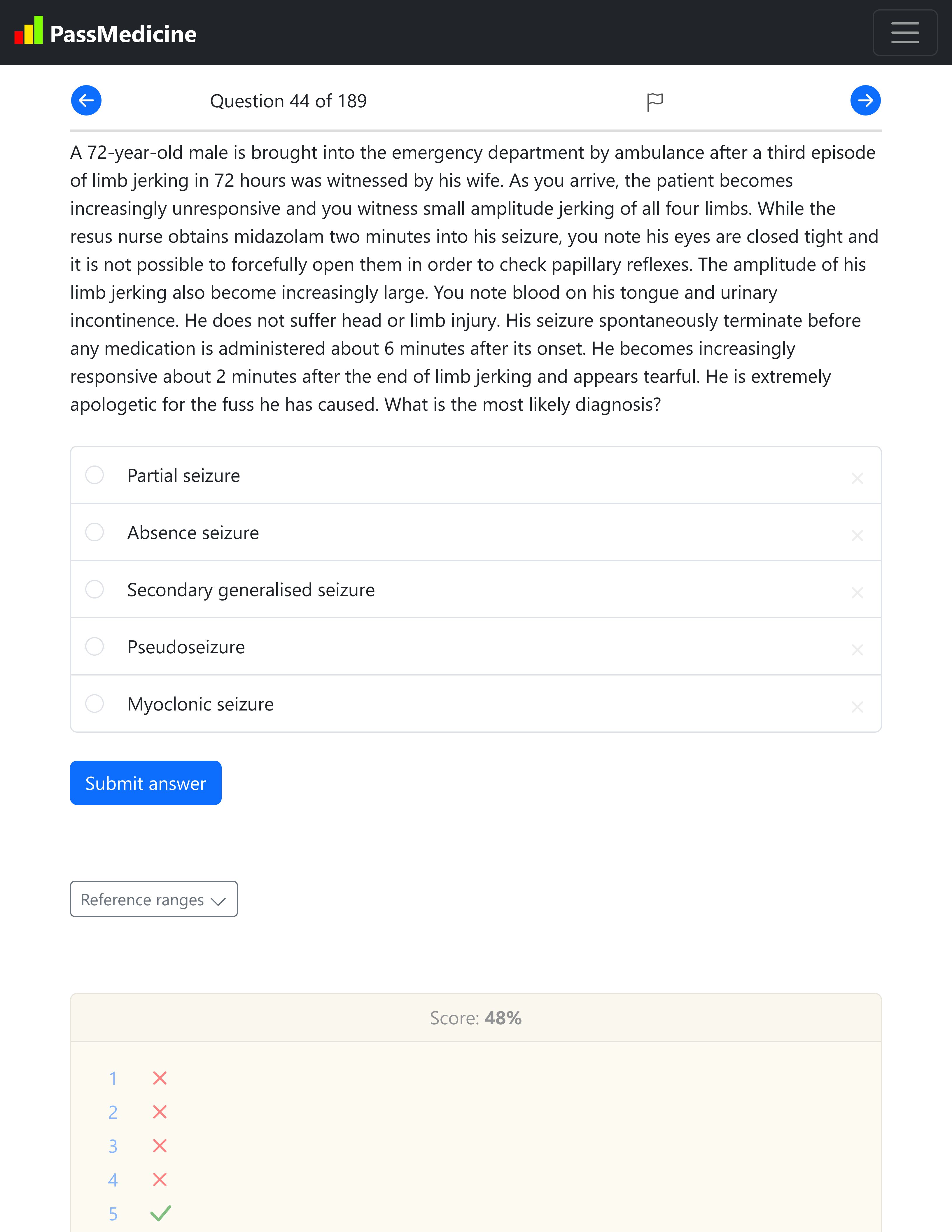

A 72-year-old male is brought into the emergency department by ambulance after a third episode of limb jerking in 72 hours was witnessed by his wife. As you arrive, the patient becomes increasingly unresponsive and you witness small amplitude jerking of all four limbs. While the resus nurse obtains midazolam two minutes into his seizure, you note his eyes are closed tight and it is not possible to forcefully open them in order to check papillary reflexes. The amplitude of his limb jerking also become increasingly large. You note blood on his tongue and urinary incontinence. He does not suffer head or limb injury. His seizure spontaneously terminate before any medication is administered about 6 minutes after its onset. He becomes increasingly responsive about 2 minutes after the end of limb jerking and appears tearful. He is extremely apologetic for the fuss he has caused. What is the most likely diagnosis?

- ○ Partial seizure
- ○ Absence seizure
- ○ Secondary generalised seizure
- ○ Pseudoseizure
- ○ Myoclonic seizure

Score: **48%**

1 ✗
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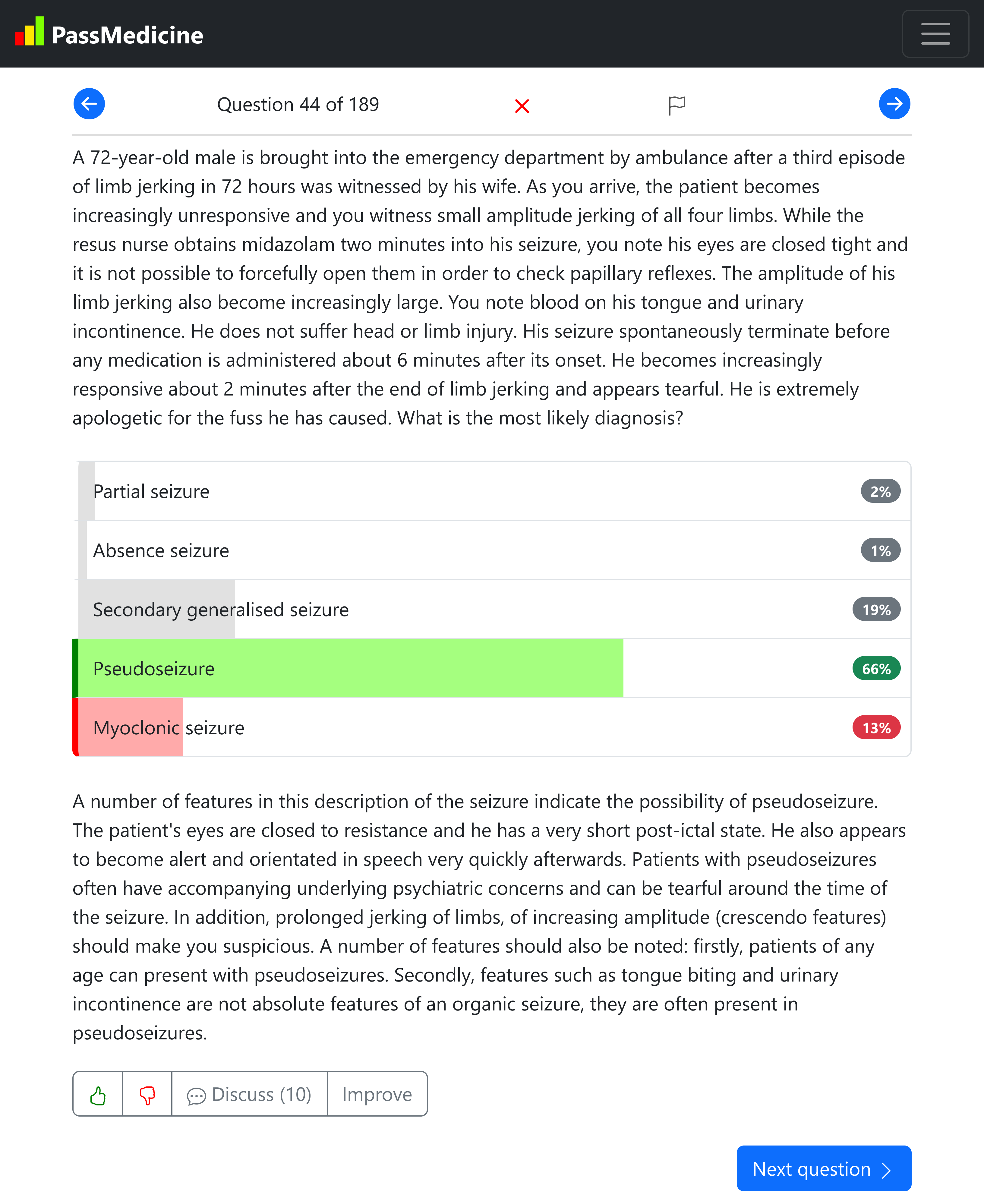

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Partial seizure	2%
Absence seizure	1%
Secondary generalised seizure	19%
Pseudoseizure	66%
Myoclonic seizure	13%

A number of features in this description of the seizure indicate the possibility of pseudoseizure. The patient's eyes are closed to resistance and he has a very short post-ictal state. He also appears to become alert and orientated in speech very quickly afterwards. Patients with pseudoseizures often have accompanying underlying psychiatric concerns and can be tearful around the time of the seizure. In addition, prolonged jerking of limbs, of increasing amplitude (crescendo features) should make you suspicious. A number of features should also be noted: firstly, patients of any age can present with pseudoseizures. Secondly, features such as tongue biting and urinary incontinence are not absolute features of an organic seizure, they are often present in pseudoseizures.

Psychogenic non-epileptic seizures ★

Psychogenic nonepileptic seizures are sometimes referred to as pseudoseizures.

Factors favouring pseudoseizures
- pelvic thrusting
- family member with epilepsy
- much more common in females
- crying after seizure
- don't occur when alone
- gradual onset

Factors favouring true epileptic seizures
- tongue biting
- raised serum prolactin*

Video telemetry is useful for differentiating

*why prolactin is raised following seizures is not fully understood. It is hypothesised that there is spread of electrical activity to the ventromedial hypothalamus, leading to release of a specific prolactin regulator into the hypophyseal portal system

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Sheffield Teaching Hospitals patient information leafelet 👍 3 👎 2

Non-epileptic attacks: A short guide for patients and families

Suggest link Report broken link

Score: **28%**

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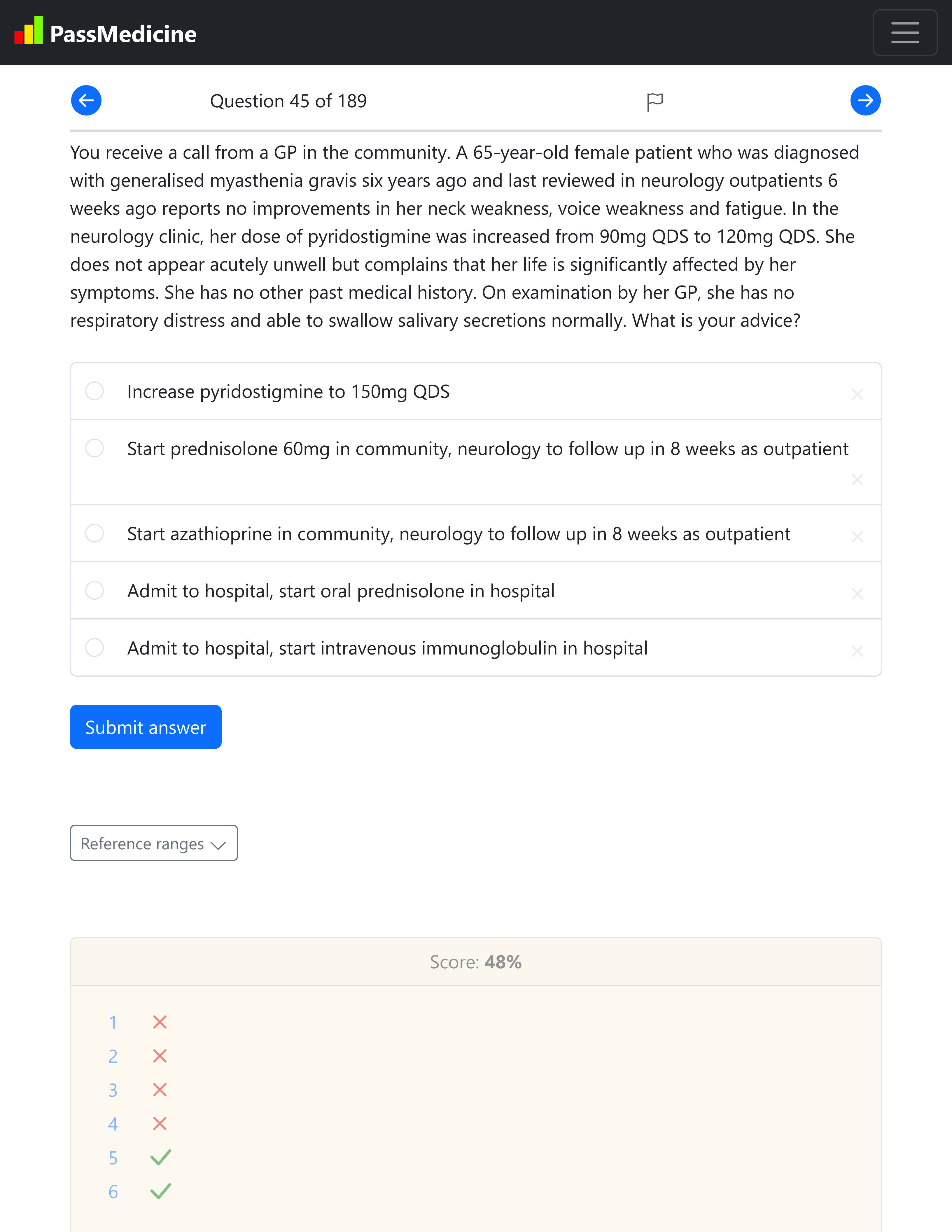

You receive a call from a GP in the community. A 65-year-old female patient who was diagnosed with generalised myasthenia gravis six years ago and last reviewed in neurology outpatients 6 weeks ago reports no improvements in her neck weakness, voice weakness and fatigue. In the neurology clinic, her dose of pyridostigmine was increased from 90mg QDS to 120mg QDS. She does not appear acutely unwell but complains that her life is significantly affected by her symptoms. She has no other past medical history. On examination by her GP, she has no respiratory distress and able to swallow salivary secretions normally. What is your advice?

- ○ Increase pyridostigmine to 150mg QDS
- ○ Start prednisolone 60mg in community, neurology to follow up in 8 weeks as outpatient
- ○ Start azathioprine in community, neurology to follow up in 8 weeks as outpatient
- ○ Admit to hospital, start oral prednisolone in hospital
- ○ Admit to hospital, start intravenous immunoglobulin in hospital

Score: **48%**

1 ✗
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6 ✓

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Question 45 of 189 ✗

You receive a call from a GP in the community. A 65-year-old female patient who was diagnosed with generalised myasthenia gravis six years ago and last reviewed in neurology outpatients 6 weeks ago reports no improvements in her neck weakness, voice weakness and fatigue. In the neurology clinic, her dose of pyridostigmine was increased from 90mg QDS to 120mg QDS. She does not appear acutely unwell but complains that her life is significantly affected by her symptoms. She has no other past medical history. On examination by her GP, she has no respiratory distress and able to swallow salivary secretions normally. What is your advice?

- Increase pyridostigmine to 150mg QDS 13%
- Start prednisolone 60mg in community, neurology to follow up in 8 weeks as outpatient 41%
- Start azathioprine in community, neurology to follow up in 8 weeks as outpatient 16%
- Admit to hospital, start oral prednisolone in hospital 15%
- Admit to hospital, start intravenous immunoglobulin in hospital 16%

The patient remains symptomatic despite being increased onto the maximum dose of pyridostigmine. The second decision is whether the patient is acutely, severely unwell, in which case rapid therapies such as IV Ig or plasmapheresis must be initiated, as per myasthenic crisis. In this case, she has no signs of respiratory distress or dysphagia, her symptoms are more of a case of not improved instead of rapid deterioration. Therefore, the patient does not require rapid therapy. However, she does require immunotherapy initiation. As a rule of thumb, patients who remain symptomatic despite 60mg QDS of pyridostigmine should be considered for immunotherapy.

The modality of immunotherapy must be individualised to the patient, as each drug has their own contraindications. In younger patients with no concerns regarding blood sugars, oral prednisolone is reasonable. Those with liver disease should avoid azathioprine, renal disease patients should avoid ciclosporin while those with haematological abnormalities should avoid mycophenolate and azathioprine.

However, starting of prednisolone, particularly at high doses, have been classically described to cause a paradoxical reaction in half of all patients, resulting in a transient deterioration before improvements are observed. Up to 10% of patients have been reported to have sufficiently severe respiratory failure requiring mechanical ventilation[1]. Initiation of prednisolone should, therefore, take place in a monitored hospital setting.

1. Miller RG, Milner-Brown HS, Mirka A. Prednisone-induced worsening of neuromuscular function in myasthenia gravis. Neurology. 1986;36(5):729.

Discuss (11) Improve

Next question ›

Myasthenia gravis: exacerbating factors

The most common exacerbating factor is exertion resulting in fatigability, which is the hallmark feature of myasthenia gravis.. Symptoms become more marked during the day

The following drugs may exacerbate myasthenia:
- penicillamine
- quinidine, procainamide
- beta-blockers
- lithium
- phenytoin
- antibiotics: gentamicin, macrolides, quinolones, tetracyclines

+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians (Edin) 👍 4 👎 2

Myasthenia gravis review

Myasthenia Gravis Association 👍 9 👎 3

Drugs which may aggravate myasthenia gravis

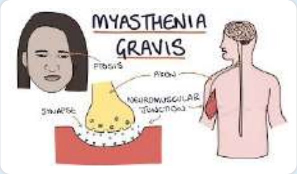
Suggest link Report broken link

Media

MYASTHENIA
GRAVIS

Myasthenia gravis - causes, symptoms, treatment, pathology

Osmosis - YouTube 👍 1 👎 0



Myasthenia gravis

Zero to Finals - YouTube 👍 2 👎 1

Report broken media

Score: **28%**

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Question 46 of 189

A 42-year-old male presents with double vision and weakness in his fingers, which he noticed when he repeatedly dropped his pen whilst trying to write at work. His symptoms appear to have onset over the past few days. He has no past medical history other than a recent episode of diarrhoea and vomiting about two weeks ago. He reports no limb weakness and no sensory loss. He denies any back pain or palpitations. On examination, there is a 3/5 weakness in finger flexion, finger extension and wrist extension in both hands, with no fatigability. No reflexes present in the lower or upper limbs. There is no ptosis or nystagmus but reduced eye movements in all directions. His finger-nose test demonstrates reduced coordination bilaterally and the patient has too little confidence to walk. Which investigation is most likely to be diagnostic?

○ Anti-GM1 antibody ×

○ Anti-GQ1b antibody ×

○ MRI brain and whole spine ×

○ Anti-MUsK (muscle specific kinase) antibody ×

○ Anti-acetylcholine receptor antibody ×

Submit answer

Reference ranges

Score: **48%**

1 ✗

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Anti-GM1 antibody	14%
Anti-GQ1b antibody	72%
MRI brain and whole spine	6%
Anti-MUsK (muscle specific kinase) antibody	5%
Anti-acetylcholine receptor antibody	3%

The patient has presented with a classic triad of ophthalmoplegia, ataxia and areflexia, which should suggest Miller-Fisher syndrome, an acute onset demyelinating peripheral neuropathy recognised as a variant of Guillain-Barre Syndrome. Anti-GQ1b antibody is present in between 85-90% of patients with Miller-Fisher syndrome[1].

Key differentials of MFS include myasthenia gravis, brainstem infarcts and brainstem encephalitis. In this case, the absence of fatigability and nystagmus rule against neuromuscular junction disorders and brainstem events respectively.

1. Chiba A, Kusunoki S, Obata H et al. Serum anti-GQ1b IgG antibody is associated with ophthalmoplegia in Miller Fisher syndrome and Guillain-Barre syndrome: clinical and immunohistochemical studies. Neurology. 1993;43(10):1911.

Discuss (2) Improve

Next question ›

Guillain-Barre syndrome

Guillain-Barre syndrome describes an immune-mediated demyelination of the peripheral nervous system often triggered by an infection (classically *Campylobacter jejuni*)

Pathogenesis
- cross-reaction of antibodies with gangliosides in the peripheral nervous system
- correlation between anti-ganglioside antibody (e.g. anti-GM1) and clinical features has been demonstrated
- anti-GM1 antibodies in 25% of patients

Miller Fisher syndrome
- variant of Guillain-Barre syndrome
- associated with ophthalmoplegia, areflexia and ataxia. The eye muscles are typically affected first
- usually presents as a descending paralysis rather than ascending as seen in other forms of Guillain-Barre syndrome
- anti-GQ1b antibodies are present in 90% of cases

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

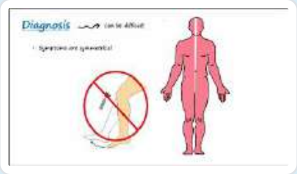
Links

Medcomic 👍 18 👎 4

Guillain-Barre syndrome

Suggest link Report broken link

Media



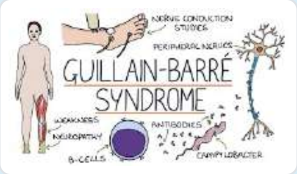
Guillain-Barre Syndrome (GBS) (Described Concisely)

PhysioPathoPharmaco - YouTube 👍 0 👎 0

SYMPTOMS - BASED on NERVES AFFECTED
MUSCLE WEAKNESS & ABSENCE of REFLEXES

Guillain-Barre syndrome

Osmosis - YouTube 👍 0 👎 0


NERVE CONDUCTION STUDIES
PERIPHERAL NERVES
GUILLAIN-BARRÉ SYNDROME
WEAKNESS
NEUROPATHY
ANTIBODIES
B-CELLS
CAMPYLOBACTER

Understanding Guillain-Barre Syndrome

Zero To Finals - YouTube 👍 0 👎 1

Report broken media

Score: **28%**

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Question 47 of 189

A 50-year-old Caucasian female on holiday from Australia presents to the walk-in urgent care centre with four-day history of left-sided temporal headache, which is persistent and of gradual onset. In addition, she complains of double vision as well. She is known to have migraines but is relatively well-controlled. She is normally independent with no significant family history. She takes no regular medications except the oral contraceptive pill. On examination, you note a left sided loss of the afferent papillary reflex. She also has a loss of vertical gaze and is unable to adduct her left eye. She has a reduced sensation to light touch on the left forehead and cheek, not crossing the midline. What is the most likely diagnosis?

- ○ Left space-occupying lesions ×
- ○ Multiple sclerosis ×
- ○ Right MCA territory ischaemic infarct ×
- ○ Cavernous sinus thrombosis ×
- ○ Migraine with aura ×

Submit answer

Reference ranges

Score: **48%**

1 ✗
2 ✗
3 ✗
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A 50-year-old Caucasian female on holiday from Australia presents to the walk-in urgent care centre with four-day history of left-sided temporal headache, which is persistent and of gradual onset. In addition, she complains of double vision as well. She is known to have migraines but is relatively well-controlled. She is normally independent with no significant family history. She takes no regular medications except the oral contraceptive pill. On examination, you note a left sided loss of the afferent papillary reflex. She also has a loss of vertical gaze and is unable to adduct her left eye. She has a reduced sensation to light touch on the left forehead and cheek, not crossing the midline. What is the most likely diagnosis?

Left space-occupying lesions	4%
Multiple sclerosis	8%
Right MCA territory ischaemic infarct	5%
Cavernous sinus thrombosis	79%
Migraine with aura	5%

The patient describes a clear history of loss of her 3rd cranial nerve, with the possible involvement of the left 4th, with V1 and V2 branches of the left trigeminal nerve. The only structure involving all these cranial nerves (and classically with the sympathetic postganglionic neuron) is the cavernous sinus. The patient has a number of risk factors for venous thrombotic disease, including recent long-haul flight and the oral contraceptive pill. The most likely causes are dehydration and VTE disease risk factors, but a carotid artery aneurysm should also be considered.

Discuss (4) | Improve

Next question

Intracranial venous thrombosis

Overview
- can cause cerebral infarction, much lesson common than arterial causes

- 50% of patients have isolated sagittal sinus thromboses - the remainder have coexistent lateral sinus thromboses and cavernous sinus thromboses

General features

Common features

- headache (may be sudden onset)
- nausea & vomiting
- reduced consciousness

Investigation

- MRI venography is the gold standard
    - CT venography is an alternative
- non-contrast CT head is normal in around 70% of patients
- D-dimer levels may be elevated

Management

- anticoagulation
    - typically with low molecular weight heparin acutely
    - warfarin is still generally used for longer term anticoagulation

Specific syndromes

Sagittal sinus thrombosis

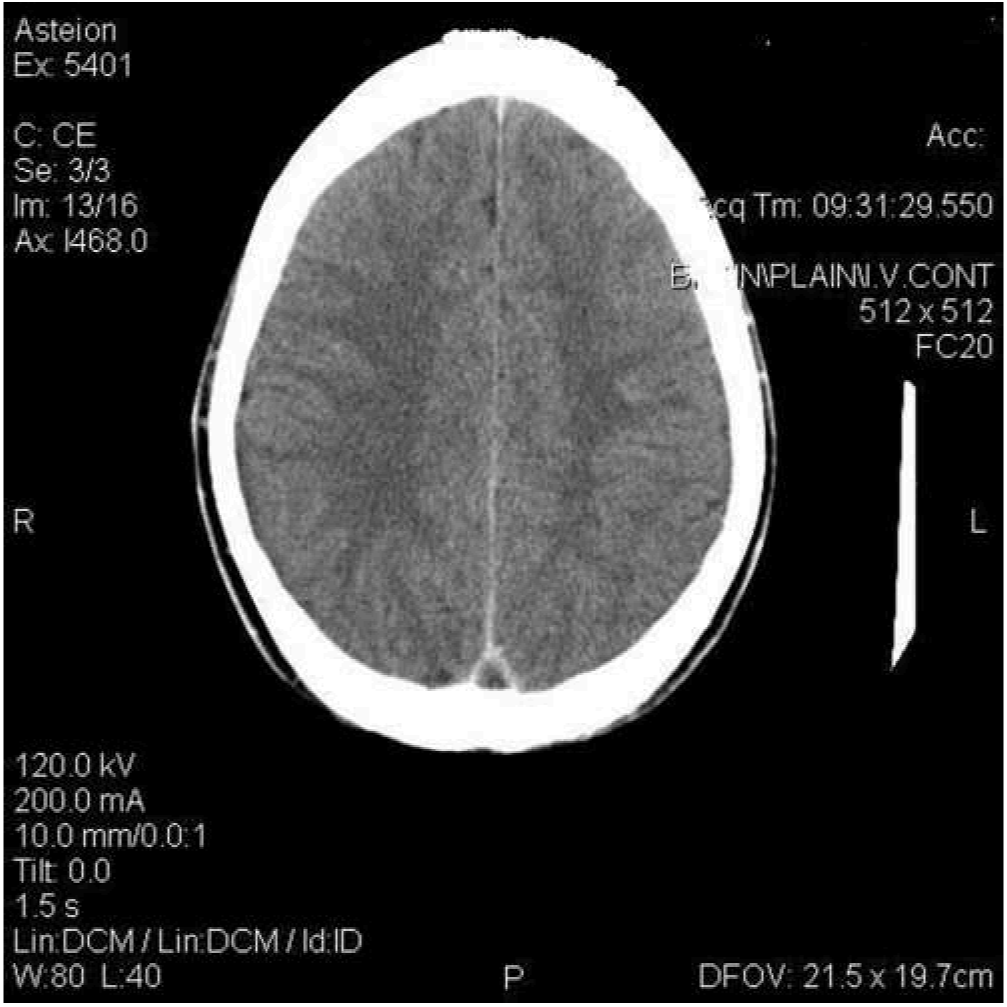
- may present with seizures and hemiplegia
- parasagittal biparietal or bifrontal haemorrhagic infarctions are sometimes seen
- 'empty delta sign' seen on venography

Cavernous sinus thrombosis

- other causes of cavernous sinus syndrome: local infection (e.g. sinusitis), neoplasia, trauma
- periorbital erythema and oedema
- ophthalmoplegia: 6th nerve damage typically occurs before 3rd & 4th
- trigeminal nerve involvement may lead to hyperaesthesia of upper face and eye pain
- central retinal vein thrombosis

Lateral sinus thrombosis

- 6th and 7th cranial nerve palsies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W:80 L:40
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DFOV: 21.5 x 19.7cm

© Image used on license from Radiopaedia

CT with contrast demonstrating a **superior sagittal sinus thrombosis** showing the typical empty delta sign. Look at the 'bottom' of the scan for the triangular shaped dural sinus. This should normally be white due to it being filled with contrast. The empty delta sign occurs when the thrombus fails to enhance within the dural sinus and is outlined by enhanced collateral channels in the falx. This sign is seen in only about 25%-30% of cases but is highly diagnostic for sagittal sinus thrombosis

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **28%**

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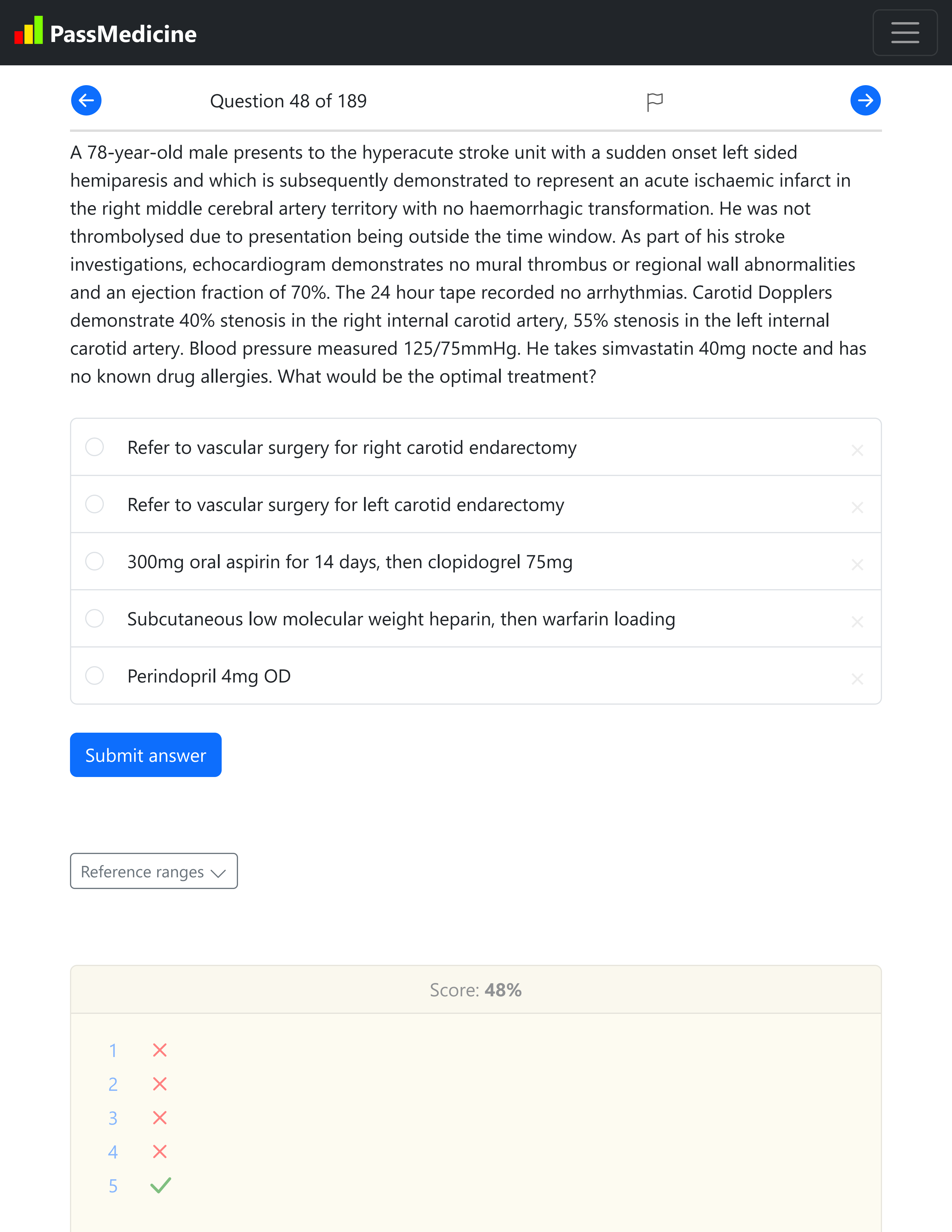

Question 48 of 189

A 78-year-old male presents to the hyperacute stroke unit with a sudden onset left sided hemiparesis and which is subsequently demonstrated to represent an acute ischaemic infarct in the right middle cerebral artery territory with no haemorrhagic transformation. He was not thrombolysed due to presentation being outside the time window. As part of his stroke investigations, echocardiogram demonstrates no mural thrombus or regional wall abnormalities and an ejection fraction of 70%. The 24 hour tape recorded no arrhythmias. Carotid Dopplers demonstrate 40% stenosis in the right internal carotid artery, 55% stenosis in the left internal carotid artery. Blood pressure measured 125/75mmHg. He takes simvastatin 40mg nocte and has no known drug allergies. What would be the optimal treatment?

- ○ Refer to vascular surgery for right carotid endarectomy
- ○ Refer to vascular surgery for left carotid endarectomy
- ○ 300mg oral aspirin for 14 days, then clopidogrel 75mg
- ○ Subcutaneous low molecular weight heparin, then warfarin loading
- ○ Perindopril 4mg OD

Score: **48%**

1 ✗
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Refer to vascular surgery for right carotid endarectomy	5%
Refer to vascular surgery for left carotid endarectomy	15%
300mg oral aspirin for 14 days, then clopidogrel 75mg	77%
Subcutaneous low molecular weight heparin, then warfarin loading	3%
Perindopril 4mg OD	1%

Carotid endarectomy (CEA) is not indicated for either internal carotid artery in this case. The present NICE guideline recommends surgical intervention if the symptomatic side demonstrates greater than 70% stenosis and to be discussed for CEA if 50-69% stenosis[1]. The management of asymptomatic stenosis is controversial: minimal but significant benefit is proven only for patients with greater than 60% stenosis and if the benefits outweigh the risk of stroke or death post CEA, which currently stands at 3%. NICE recommends consideration for asymptomatic CEA if stenosis is greater than 60% only. In this case, neither internal carotid stenosis fits the criteria for surgical intervention. Instead, best medical management with antiplatelet therapy is the treatment of choice. The use of anticoagulation beyond cardioembolic disease is controversial and not routinely used. The patients blood pressure is within the target range. The patient is already on a statin.

Beware when interpreting carotid stenosis measurements: two major trials of CEA for carotid endarectomy measured the degree of stenosis differently: ECST compared the stenotic diameter to the probable original diameter at the carotid bulb, the clearest benefit was for patients with greater than 80% stenosis[2]. NASCET compared residual diameter to the lumen directly distal to the stenosis, the greatest benefit of CEA was for patients with greater than 70% stenosis[3]. Simplistically, any patient with greater than 70% to 99% symptomatic stenosis would benefit significantly from CEA, patients with 50 to 69% stenosis would also benefit from CEA but to a lesser degree and only if performed within 2 weeks of the last episode.

1. Carotid imaging and carotid endarterectomy for people with TIA or non-disabling stroke. NICE guidelines and pathway May 2011
2. North American Symptomatic Carotid Endarterectomy Trial. Methods, patient characteristics, and progress. Stroke. 1991;22(6):711
3. MRC European Carotid Surgery Trial: interim results for symptomatic patients with severe (70-99%) or with mild (0-29%) carotid stenosis. European Carotid Surgery Trialists' Collaborative Group. Lancet. 1991;337(8752):1235

👍 👎 Discuss (5) Improve

Next question >

Stroke: management of acute ischaemic stroke ★

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:
- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
  - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
  - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:
- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.
- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
  - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
  - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Absolute	Relative
- Oesophageal varices - Uncontrolled hypertension >200/120mmHg	

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

+ Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 11 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 16 15

Stroke and TIA guidelines

Age and Ageing 👍 20 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

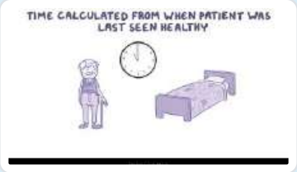
2023 National Clinical Guideline for Stroke

NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia.org
Acute Ischaemic Stroke

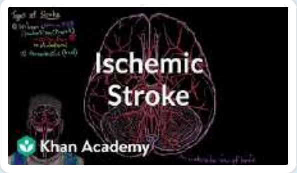
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

Khan Academy - YouTube 👍 1 👎 1

Report broken media

Score: **28%**

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A 64-year-old gentleman presented to the neurology clinic for review. He had a history of motor neuron disease which had been progressing over the past five years. He had significant weakness in all of his limbs and was dependent on a wheelchair. He took baclofen for relief of muscle spasms, and amitriptyline to reduce salivation but was otherwise not on any medication. He mentions that in addition to his weakness continuing to progress he has been suffering from headaches in the morning. These were generalised and described as throbbing in nature. He also reported not feeling refreshed after sleep.

On examination, there is generalised muscle wasting, with fasciculations in all limbs. Reflexes were absent, other than bilateral extensor plantar responses. Eye movements and fundoscopy are normal. Palatal movements were normal, and there was no significant dysphagia on swallow test.

Which intervention is most likely to confer the greatest survival benefit in this patient?

- ○ Riluzole
- ○ Feeding via percutaneous endoscopic gastrostomy
- ○ Non-invasive ventilation
- ○ Home oxygen therapy
- ○ Prophylactic antibiotics

Submit answer

Reference ranges

Score: **48%**

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Which intervention is most likely to confer the greatest survival benefit in this patient?

Riluzole	27%
Feeding via percutaneous endoscopic gastrostomy	3%
Non-invasive ventilation	68%
Home oxygen therapy	2%
Prophylactic antibiotics	0%

The morning headaches and lack of feeling refreshed after sleep are suggestive of carbon dioxide retention due to respiratory impairment in patients with motor neuron disease. Non-invasive ventilation improves quality of life and confers a survival benefit in patients with respiratory impairment in motor neuron disease.

Riluzole also offers a modest survival benefit in motor neuron disease (it is the only drug known to do so), prolonging life expectancy by on average two to four months. Given there is no evidence of dysphagia feeding via a percutaneous endoscopic gastrostomy is not indicated in this patient. Home oxygen therapy and prophylactic antibiotics are not routinely used in motor neuron disease.

Discuss (1) Improve

Next question >

Motor neuron disease: management ★

Motor neuron disease is a neurological condition of unknown cause which can present with both upper and lower motor neuron signs. It rarely presents before 40 years and various patterns of disease are recognised including amyotrophic lateral sclerosis, progressive muscular atrophy and bulbar palsy

Riluzole
- prevents stimulation of glutamate receptors
- used mainly in amyotrophic lateral sclerosis
- prolongs life by about 3 months

Respiratory care
- non-invasive ventilation (usually BIPAP) is used at night
- studies have shown a survival benefit of around 7 months

Nutrition
- percutaneous gastrostomy tube (PEG) is the preferred way to support nutrition and has been associated with prolonged survival

Prognosis
- poor: 50% of patients die within 3 years

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 6 👎 5

2016 Motor neurone disease: assessment and management

Suggest link Report broken link

Media

MOTOR NEURONE DISEASE
SPEECH
TRUNK
MOTOR NEURONE DEGENERATION
VENTILATION
LIMBS
TONGUE WASTING
BRISK REFLEXES

Motor neuron disease

Zero To Finals - YouTube 👍 0 👎 0

Report broken media

Score: **28%**

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A 42-year-old female presented with a 6-month history of weakness affecting her upper limbs. She found that this was worse at the end of the day, particularly if she had been doing a lot of lifting. She also reported occasional difficulty in swallowing at the end of the day, with drinks causing her to cough. She had been prescribed pyridostigmine, but she stopped taking this as she had not noticed any benefit.

On examination, there was a mild partial ptosis, and a slight facial droop bilaterally. Extra-ocular movements were normal. Power in shoulder abduction was grade 4/5 bilaterally, but was reduced to 3/5 after repeatedly abducting and adducting the shoulder 20 times. Tone and sensation were normal.

Initial investigation results are shown below:

Nerve conduction studies: Decremental response to repetitive nerve stimulation
Single-fibre electromyography: Increased jitter
Anti-Jo antibodies: Negative
Anti-Mi2 antibodies: Negative
Anti-acetylcholine receptor antibodies: Negative

Which of the following immunological tests is most likely to contribute to the diagnosis?

- ○ Anti-muscle-specific tyrosine kinase(MuSK) antibodies
- ○ Anti-N-methyl D-aspartate (NMDA) receptor antibodies
- ○ Anti-glutamic acid decarboxylase (GAD) antibodies
- ○ Anti-acetylcholine receptor antibody
- ○ Anti-Hu antibodies

Submit answer

Reference ranges

Score: **48%**

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Single-fibre electromyography: Increased jitter
Anti-Jo antibodies: Negative
Anti-Mi2 antibodies: Negative
Anti-acetylcholine receptor antibodies: Negative

Which of the following immunological tests is most likely to contribute to the diagnosis?

Anti-muscle-specific tyrosine kinase(MuSK) antibodies	64%
Anti-N-methyl D-aspartate (NMDA) receptor antibodies	4%
Anti-glutamic acid decarboxylase (GAD) antibodies	3%
Anti-acetylcholine receptor antibody	25%
Anti-Hu antibodies	3%

This patient presents with weakness affecting the upper limbs, eyelids, facial muscles and pharynx, which demonstrates fatiguability. This is a characteristic description of myasthenia gravis, and this is supported by the electrophysiological tests. The majority of patients with this condition are positive for anti-acetylcholine receptor antibodies, which are present in about 85%. In the remaining patients, about 40% are positive for anti-muscle-specific tyrosine kinase antibodies. In anti-acetylcholine receptor negative cases, response to acetylcholinesterase inhibitors (such as pyridostigmine) tends to be worse.

Anti-aquaporin antibodies are associated with neuromyelitis optica, anti-N-methyl D-aspartate receptor antibodies are associated with autoimmune encephalitis, anti-glutamic acid decarboxylase antibodies are associated with stiff man syndrome, and anti-Hu antibodies are associated with a paraneoplastic sensory neuropathy and encephalitis.

👍 👎 Discuss (4) Improve

Next question >

Myasthenia gravis

Myasthenia gravis is an autoimmune disorder resulting in insufficient functioning acetylcholine receptors. Antibodies to acetylcholine receptors are seen in 85-90% of cases*. Myasthenia is more common in women (2:1)

The key feature is muscle fatigability - muscles become progressively weaker during periods of activity and slowly improve after periods of rest:
- extraocular muscle weakness: diplopia
- proximal muscle weakness: face, neck, limb girdle
- ptosis
- dysphagia

Associations
- thymomas in 15%
- autoimmune disorders: pernicious anaemia, autoimmune thyroid disorders, rheumatoid, SLE
- thymic hyperplasia in 50-70%

Investigations
- single fibre electromyography: high sensitivity (92-100%)
- CT thorax to exclude thymoma
- CK normal
- antibodies to acetylcholine receptors
  - positive in around 85-90% of patients
  - n the remaining patients, about about 40% are positive for anti-muscle-specific tyrosine kinase antibodies
- Tensilon test: IV edrophonium reduces muscle weakness temporarily - not commonly used any more due to the risk of cardiac arrhythmia

Management

- long-acting acetylcholinesterase inhibitors
  - pyridostigmine is first-line
- immunosuppression is usually not started at diagnosis, but the majority of patients eventually require it in addition to long-acting acetylcholinesterase inhibitors:
  - prednisolone initially
  - azathioprine, cyclosporine, mycophenolate mofetil may also be used
- thymectomy

Management of myasthenic crisis

- plasmapheresis
- intravenous immunoglobulins

*antibodies are less commonly seen in disease limited to the ocular muscles

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

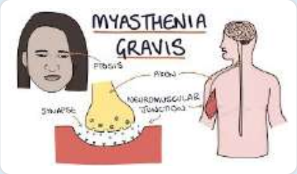
Postgraduate Medical Journal 👍 13 👎 3

Review of myasthenia gravis

Suggest link

Report broken link

Media


MYASTHENIA GRAVIS
PTOSIS
AXON
SYNAPSE
NEUROMUSCULAR JUNCTION

Myasthenia gravis

Zero to Finals - YouTube 👍 12 👎 1

NEUROLOGY
MYASTHENIA GRAVIS

Myasthenia gravis

Armando Hasudungan - YouTube 👍 1 👎 1

MYASTHENIA GRAVIS

Myasthenia gravis - causes, symptoms, treatment, pathology

Osmosis - YouTube 👍 2 👎 2

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Score: **28%**

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You are called to a general medical ward during the night to review 48 year female who has become acutely agitated and confused. She came into hospital two days ago with a suspected urinary tract infection and has been making uneventful progress. She has a past history of traumatic spinal injury at level C5 three years ago with resulting spastic quadraparesis. On your arrival her blood pressure is 220/105 mmHg, heart rate is 55/min, and she is flushed and diaphoretic.

Of the following, what additional feature might you expect to find?

- ○ Palpable bladder
- ○ Ejection systolic murmur in the pulmonary area
- ○ Flushing of the skin in the trunk and legs
- ○ Relative afferent pupillary defect
- ○ Complete heart block

Submit answer

Reference ranges

Score: **28%**

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Of the following, what additional feature might you expect to find?

Palpable bladder	71%
Ejection systolic murmur in the pulmonary area	5%
Flushing of the skin in the trunk and legs	11%
Relative afferent pupillary defect	9%
Complete heart block	4%

This question is describing a case of autonomic dysreflexia. Of the options given, palpation of a distended bladder is likely and also diagnostic. Pulmonary murmurs can be a feature of the carcinoid syndrome. A relative afferent pupillary defect is seen with optic nerve pathology such as optic neuritis. Flushing of skin above the cord lesion is common due to parasympathetic feedback causing vasodilation but of course anything below the cord lesion remains vasoconstricted.

Discuss (8) Improve

Next question ›

Autonomic dysreflexia

This clinical syndrome occurs in patients who have had a spinal cord injury at, or above T6 spinal level. Briefly, afferent signals, most commonly triggered by faecal impaction or urinary retention (but many other triggers have been reported) cause a sympathetic spinal reflex via thoracolumbar outflow. The usual, centrally mediated, parasympathetic response however is prevented by the cord lesion.

Features
- the result is an unbalanced physiological response, characterised by extreme hypertension
- flushing and sweating above the level of the cord lesion
- agitation
- in untreated cases, severe consequences of extreme hypertension have been reported, e.g. haemorrhagic stroke.

Management of autonomic dysreflexia involves removal/control of the stimulus and treatment of any life-threatening hypertension and/or bradycardia.

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal National Orthopaedic Hospital 4 1

Autonomic Dysreflexia

Suggest link

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Media

Autonomic
Dysreflexia
with
Dr Mike

Autonomic Dysreflexia

Dr Matt & Dr Mike - YouTube 👍 36 👎 2

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Score: **24.8%**

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A 53-year-old accountant presents with two weeks of a constant headache with two seizures in the last 24 hours. His family say that he has changed in character and seems more aggressive. They also say that he appears weaker, is eating less and has 2 lbs of weight over the past week.

He is known to be HIV positive and had an anterior resection for rectal carcinoma 3 years ago and a recent endoscopy shows gastric polyps. He is currently well but has suffered from multiple episodes of sinusitis and is taking a four-week course of oral antibiotics.

On examination, he has reduced power in the left upper limb across all muscular groups but preserved sensation. Reflexes are reduced on the left side. In the left lower limb, power is mildly reduced and there is an upgoing plantar with normal sensation. He has a Glasgow Coma Score of 13 and he is drowsy. Pupils are equal and reactive but there is papilloedema. His observations show a heart rate of 96 beats per minute, blood pressure 110/98 mmHg, temperature 38.0'C and respiratory rate 18 per minute with saturations of 98%.

Na^+	137 mmol/l
K^+	4.3 mmol/l
Urea	3.5 mmol/l
Creatinine	40 µmol/l

Hb	100 g/l
Platelets	$378* 10^9$/l
WBC	$12.3* 10^9$/l

Blood cultures	pending
CT head	requested

What is the likely diagnosis?

○ Tertiary syphilis ×

○ Lymphoma ×

○ Toxoplasmosis ×

○ Cerebral abscess ×

○ Glioma ×

Submit answer

Reference ranges ⌵

Score: **28%**

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Question 52 of 189

A 53-year-old accountant presents with two weeks of a constant headache with two seizures in the last 24 hours. His family say that he has changed in character and seems more aggressive. They also say that he appears weaker, is eating less and has 2 lbs of weight over the past week.

He is known to be HIV positive and had an anterior resection for rectal carcinoma 3 years ago and a recent endoscopy shows gastric polyps. He is currently well but has suffered from multiple episodes of sinusitis and is taking a four-week course of oral antibiotics.

On examination, he has reduced power in the left upper limb across all muscular groups but preserved sensation. Reflexes are reduced on the left side. In the left lower limb, power is mildly reduced and there is an upgoing plantar with normal sensation. He has a Glasgow Coma Score of 13 and he is drowsy. Pupils are equal and reactive but there is papilloedema. His observations show a heart rate of 96 beats per minute, blood pressure 110/98 mmHg, temperature 38.0'C and respiratory rate 18 per minute with saturations of 98%.

Na^+	137 mmol/l
K^+	4.3 mmol/l
Urea	3.5 mmol/l
Creatinine	40 µmol/l

Hb	100 g/l
Platelets	$378* 10^9/l$
WBC	$12.3* 10^9/l$

Blood cultures	pending
CT head	requested

What is the likely diagnosis?

Tertiary syphilis	5%
Lymphoma	8%
Toxoplasmosis	19%

Cerebral abscess 65%

Glioma 3%

Features of raised intracranial pressure and fever points towards a cerebral abscess

Important for me Less important

There is evidence of raised intracranial pressure that is gradual in onset. Subarachnoid presents with a severe headache which has not occurred here. Tertiary syphilis is rare and there are no specific signs pointing towards this diagnosis. Toxoplasmosis is possible given he is HIV positive but there are no additional features pointing towards this. Of cerebral abscess and glioma, the presence of fever makes the former likely and the abscess is likely secondary bacterial spread from sinusitis. A glioma is possible given the weight loss and previous malignancy but would usually present over a longer period of time.

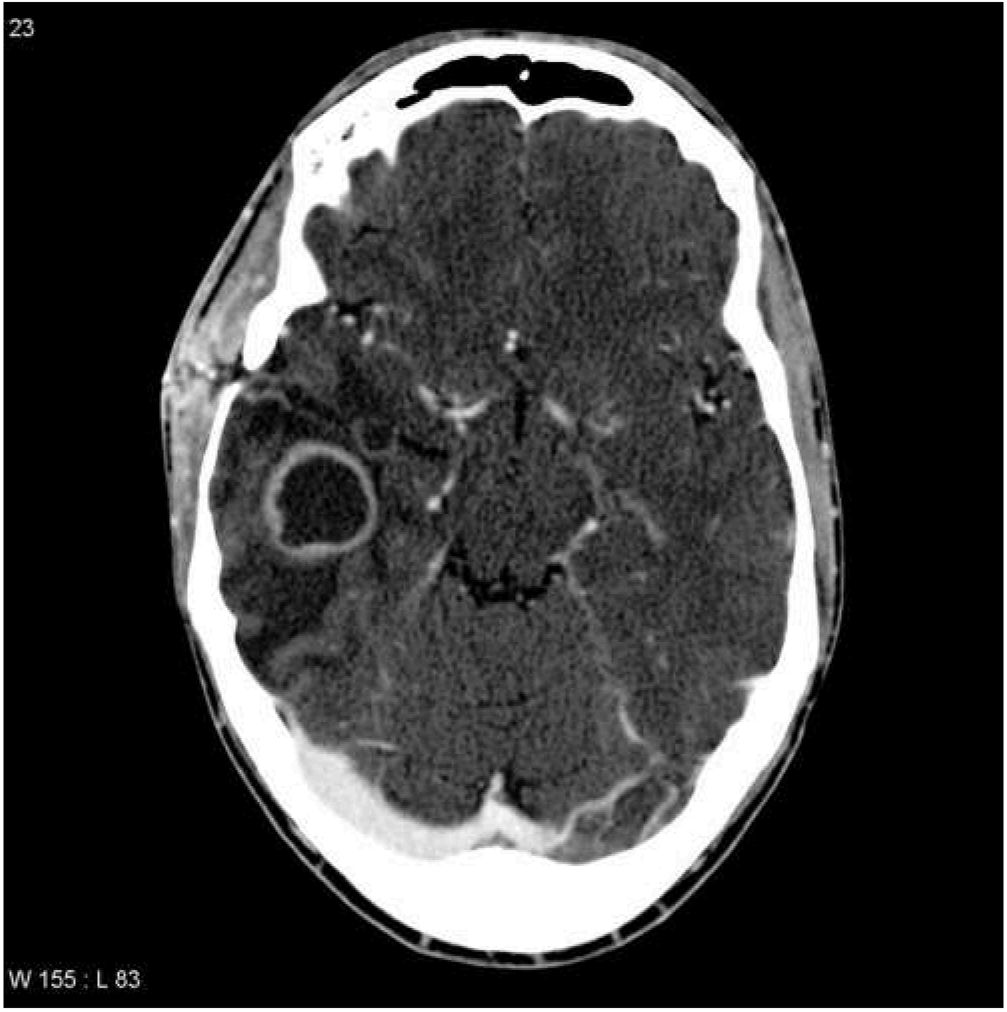
👍 👎 Discuss (7) Improve

Next question ›

Intracerebral abscess ★

Features
- fever
- headache
- seizures
- signs of raised intracranial pressure
- focal neurological deficits

Management
- surgical drainage: supratentorial abscesses may be drained via a burr hole
- antibiotic therapy



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CT demonstrating a cerebral abscess. CT demonstrates a rounded lesions with vast amounts of surrounding vasogenic oedema resulting in mass effect and distortion of the cerebral peduncle. Contrast confirms the presence of a smooth ring of enhancement, characteristic of an abscess.

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **24.8%**

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Question 53 of 189

A 23-year-old man with known Von Hippel Lindau syndrome sees his GP to routinely review his mood. He has been well previously but has known retinal haemangiomas. He is taking venlafaxine and has improved in his mood which was damaged from a recent relationship break down. He does continue to get panic attacks where he develops palpitations and feels clammy and is using mindfulness to control these.

On examination, he is well and alert. His blood pressure is 170/110mmHg. He has 6/6 visual acuity bilaterally and there are no haemorrhages or cotton wool spots on fundoscopy. His abdomen is soft and there are no palpable masses.

Na^+	134 mmol/l
K^+	4.1 mmol/l
HCO3	25mmol/l
Urea	6.7 mmol/l
Creatinine	87 µmol/l
TSH	3.2 mU/l (range 0-4)
ECG	large QRS complexes consistent with left ventricular hypertrophy

What is the likely cause of his presentation?

- ○ Venlafaxine
- ○ Phaeochromocytoma
- ○ Renal artery stenosis
- ○ Depression/anxiety
- ○ Liddle's syndrome

Submit answer

Reference ranges ∨

Score: **28%**

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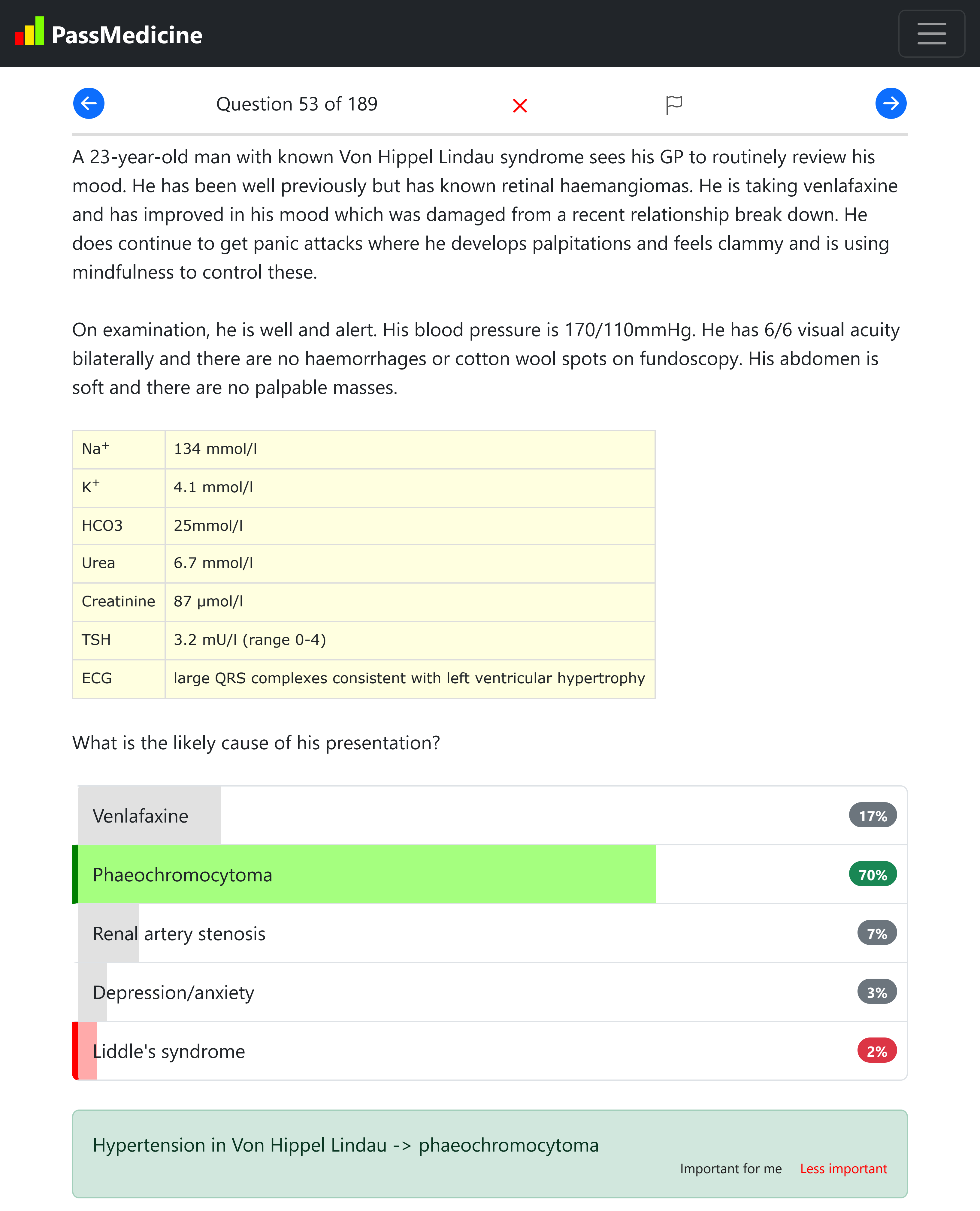

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Na^+	134 mmol/l
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HCO3	25mmol/l
Urea	6.7 mmol/l
Creatinine	87 µmol/l
TSH	3.2 mU/l (range 0-4)
ECG	large QRS complexes consistent with left ventricular hypertrophy

What is the likely cause of his presentation?

Venlafaxine	17%
Phaeochromocytoma	70%
Renal artery stenosis	7%
Depression/anxiety	3%
Liddle's syndrome	2%

Hypertension in Von Hippel Lindau -> phaeochromocytoma

Important for me Less important

This gentleman has presented with hypertension and episodes of sweating and palpitations. He is usually well and is on venlafaxine which can cause hypertension but not the other symptoms. Renal artery stenosis would usually present with deranged urea and electrolytes and is rare in this age group. Liddle's syndrome presents with a hypernatraemia, hypokalaemia metabolic alkalosis which is not seen here. Depression/anxiety can cause BP fluctuations but is a diagnosis of exclusion. Phaeochromocytoma would explain these symptoms and also is more common in Von Hippel Lindau so is the answer.

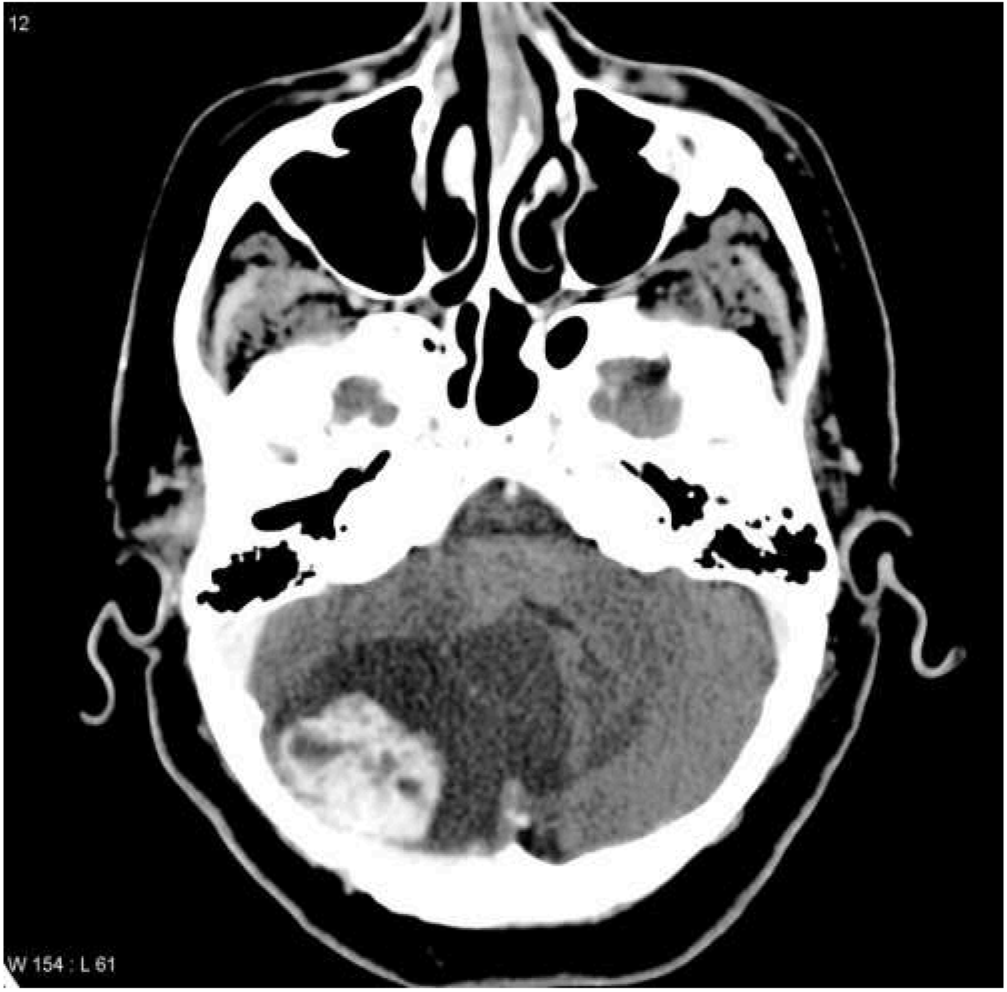
👍 👎 Discuss (2) Improve

Next question ›

Von Hippel-Lindau syndrome ★

Von Hippel-Lindau (VHL) syndrome is an autosomal dominant condition predisposing to neoplasia. It is due to an abnormality in the VHL gene located on short arm of chromosome 3

Features
- cerebellar haemangiomas: these can cause subarachnoid haemorrhages
- retinal haemangiomas: vitreous haemorrhage
- renal cysts (premalignant)
- phaeochromocytoma
- extra-renal cysts: epididymal, pancreatic, hepatic
- endolymphatic sac tumours
- clear-cell renal cell carcinoma



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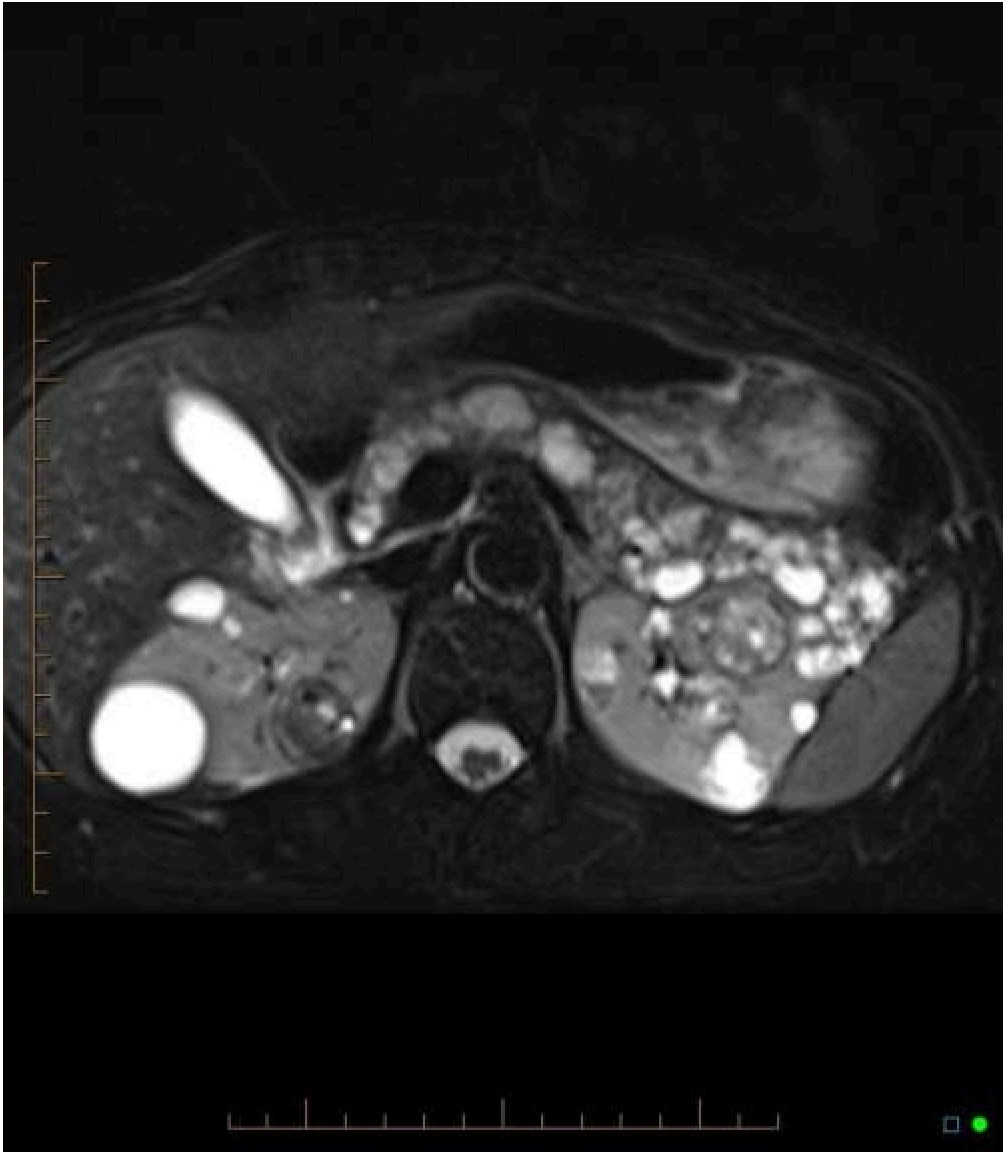
CT scan showing a cerebellar haemangioma in a patient with Von Hippel-Lindau syndrome.



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MRI showing renal cysts in patient with known Von Hippel-Lindau syndrome.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **24.8%**

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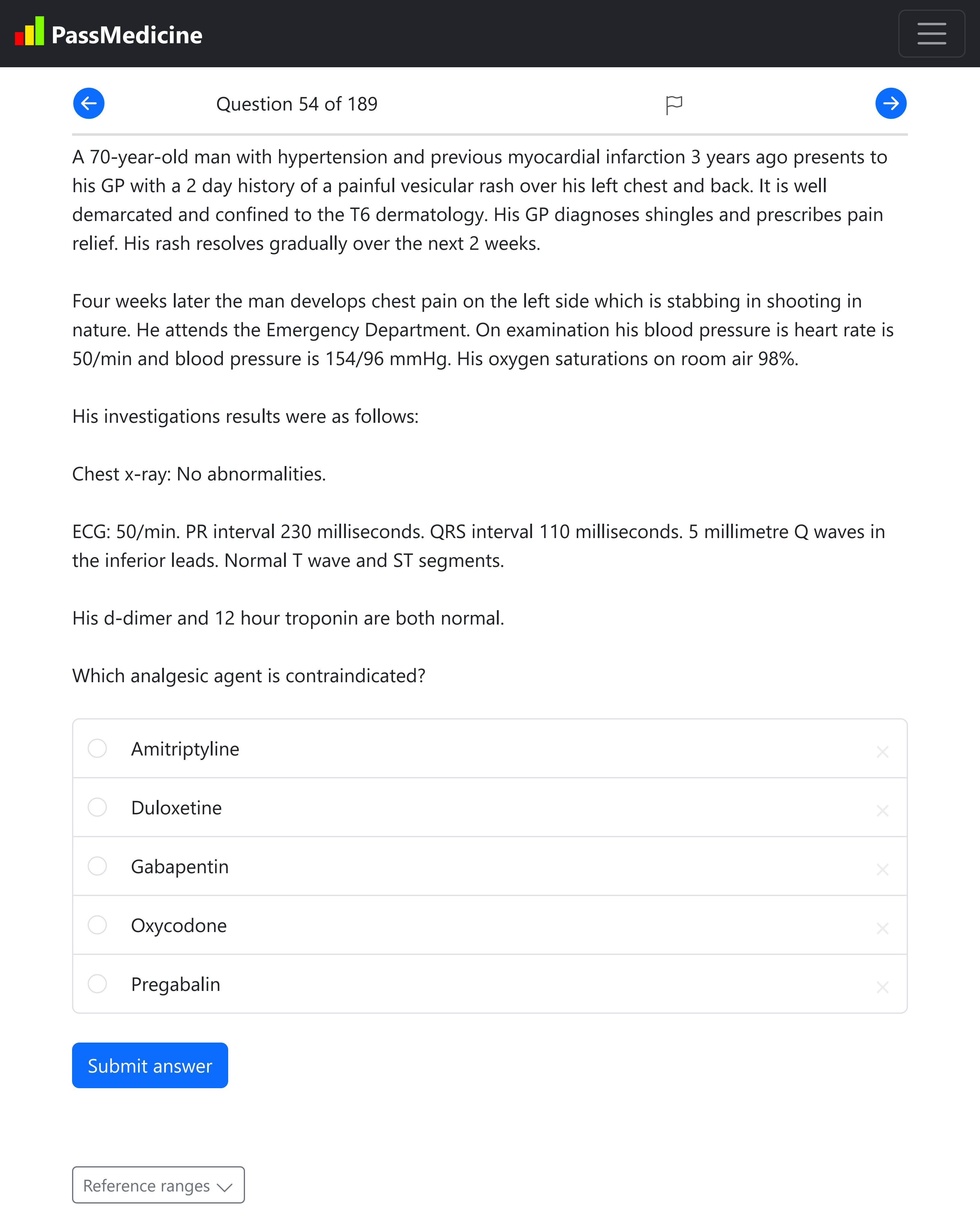

A 70-year-old man with hypertension and previous myocardial infarction 3 years ago presents to his GP with a 2 day history of a painful vesicular rash over his left chest and back. It is well demarcated and confined to the T6 dermatology. His GP diagnoses shingles and prescribes pain relief. His rash resolves gradually over the next 2 weeks.

Four weeks later the man develops chest pain on the left side which is stabbing in shooting in nature. He attends the Emergency Department. On examination his blood pressure is heart rate is 50/min and blood pressure is 154/96 mmHg. His oxygen saturations on room air 98%.

His investigations results were as follows:

Chest x-ray: No abnormalities.

ECG: 50/min. PR interval 230 milliseconds. QRS interval 110 milliseconds. 5 millimetre Q waves in the inferior leads. Normal T wave and ST segments.

His d-dimer and 12 hour troponin are both normal.

Which analgesic agent is contraindicated?

- ○ Amitriptyline
- ○ Duloxetine
- ○ Gabapentin
- ○ Oxycodone
- ○ Pregabalin

Score: **28%**

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His d-dimer and 12 hour troponin are both normal.

Which analgesic agent is contraindicated?

Amitriptyline	61%
Duloxetine	18%
Gabapentin	5%
Oxycodone	12%
Pregabalin	4%

This gentleman has post-herpetic neuralgia causing his chest pain.

He has a history of heart attack and his ECG shows evidence of inferior myocardial infarction in the territory of the right coronary artery. This has resulted in a first degree heart block as evidenced by the long PR interval.

NICE Guidance on Neuropathic Pain (CG173) advocates a choice of amitriptyline, duloxetine, gabapentin or pregabalin. According to the BNF amitriptyline is contraindicated in arrhythmias particularly heart block, as in this case. It is also contraindicated in the recovery period following myocardial infarction, though this gentleman is outside of this.

👍 👎 Discuss (4) Improve

Next question >

Tricyclic antidepressants ★

Tricyclic antidepressants (TCAs) are used less commonly now for depression due to their side-effects and toxicity in overdose. They are however used widely in the treatment of neuropathic pain, where smaller doses are typically required.

The primary mechanism by which TCAs exert their antidepressant effects is through the inhibition of the reuptake of neurotransmitters

- Serotonin (5-HT): This neurotransmitter has a pivotal role in mood regulation. Inhibition of its reuptake leads to increased concentrations in the synaptic cleft, enhancing serotonergic neurotransmission.
- Noradrenaline (NA): Similar to 5-HT, blocking the reuptake of NA increases its synaptic cleft concentration, intensifying noradrenergic neurotransmission.

As well as 5-HT and NA, tricyclics interact with number of other receptors that contribute to their side-effect profile:

- antagonism of histamine receptors
  - drowsiness
- antagonism of muscarinic receptors
  - dry mouth
  - blurred vision
  - constipation
  - urinary retention
- antagonism of adrenergic receptors
  - postural hypotension
- lengthening of QT interval

Choice of tricyclic

- low-dose amitriptyline is commonly used in the management of neuropathic pain and the prophylaxis of headache (both tension and migraine)
- lofepramine has a lower incidence of toxicity in overdose
- amitriptyline and dosulepin (dothiepin) are considered the most dangerous in overdose

More sedative	Less sedative
Amitriptyline Clomipramine Dosulepin Trazodone*	Imipramine Lofepramine Nortriptyline

*trazodone is technically a 'tricyclic-related antidepressant'

+ Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Media

ANTIDEPRESSANTS
PHARMACOLOGY

Antidepressants Pharmacology

Soton Brain Hub - YouTube 0 0

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Report broken media

Score: **24.8%**

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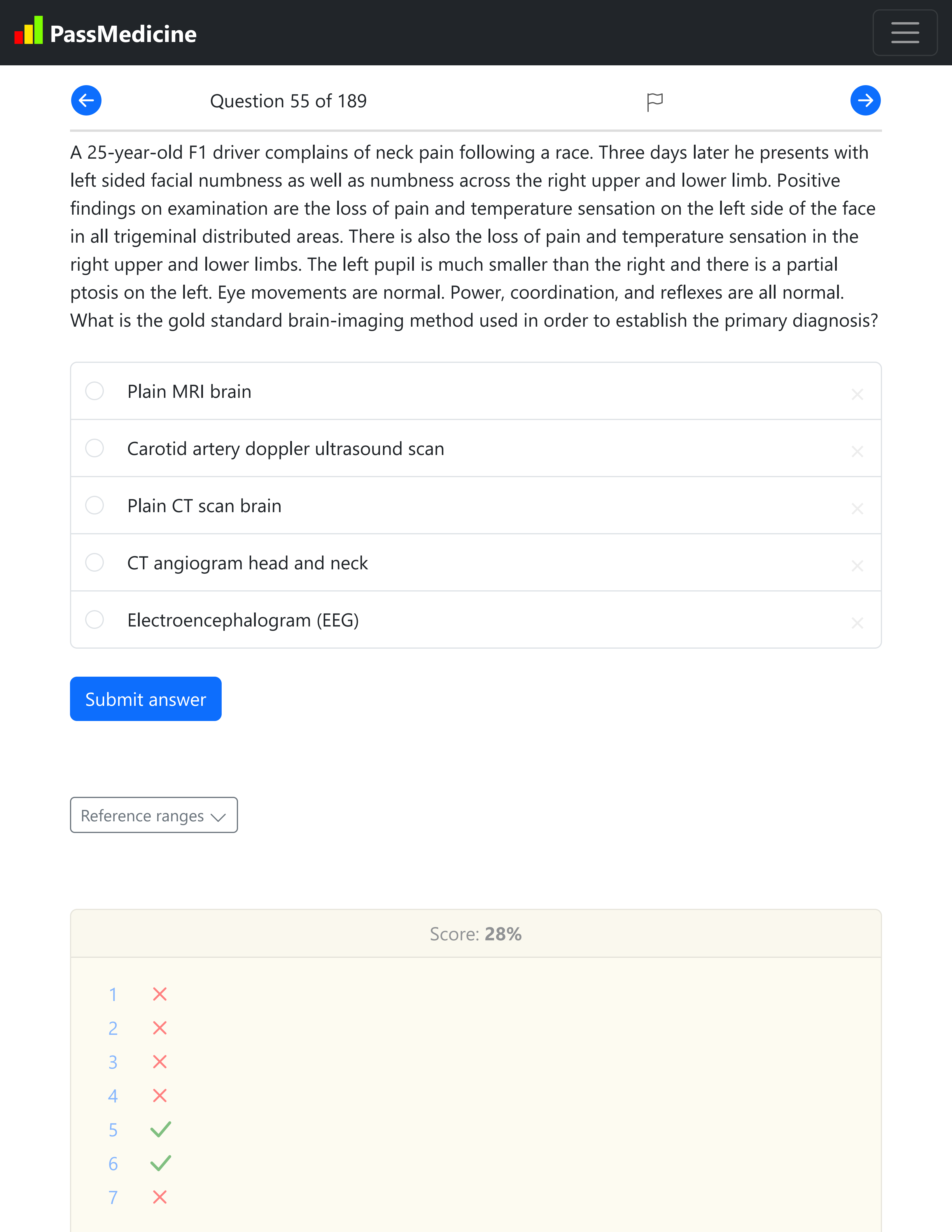

Question 55 of 189

A 25-year-old F1 driver complains of neck pain following a race. Three days later he presents with left sided facial numbness as well as numbness across the right upper and lower limb. Positive findings on examination are the loss of pain and temperature sensation on the left side of the face in all trigeminal distributed areas. There is also the loss of pain and temperature sensation in the right upper and lower limbs. The left pupil is much smaller than the right and there is a partial ptosis on the left. Eye movements are normal. Power, coordination, and reflexes are all normal. What is the gold standard brain-imaging method used in order to establish the primary diagnosis?

- ○ Plain MRI brain ×
- ○ Carotid artery doppler ultrasound scan ×
- ○ Plain CT scan brain ×
- ○ CT angiogram head and neck ×
- ○ Electroencephalogram (EEG) ×

Submit answer

Reference ranges

Score: **28%**

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A 25-year-old F1 driver complains of neck pain following a race. Three days later he presents with left sided facial numbness as well as numbness across the right upper and lower limb. Positive findings on examination are the loss of pain and temperature sensation on the left side of the face in all trigeminal distributed areas. There is also the loss of pain and temperature sensation in the right upper and lower limbs. The left pupil is much smaller than the right and there is a partial ptosis on the left. Eye movements are normal. Power, coordination, and reflexes are all normal. What is the gold standard brain-imaging method used in order to establish the primary diagnosis?

Plain MRI brain	20%
Carotid artery doppler ultrasound scan	7%
Plain CT scan brain	3%
CT angiogram head and neck	69%
Electroencephalogram (EEG)	0%

This gentleman has a vertebral artery dissection following neck hyperflexion during the F1 car race. It is not uncommon for young people to get these sorts of strokes, particularly young sporting people such as in the sad case of the cricketer Phil Hughes.

The vertebral artery has a tributary (the posterior inferior cerebellar artery -PICA) to the medulla and in this case, the F1 driver has therefore developed a left sided lateral medullary syndrome (aka Wallenberg syndrome). Below are the features of this syndrome:

Cerebellar features: ipsilateral limb ataxia, vertigo, nystagmus to the side of the lesion. These are due to taking out the ipsilateral spinocerebellar tracts within the medulla.

Brain stem features:

dizziness and vomiting (taken out the vestibular and vagal nuclei)
dysphagia and dysarthria (again this represents the vagal nuclei being taken out)
ipsilateral Horner's syndrome (the sympathetic tract has been taken out on that side)
ipsilateral facial pain and temperature sensory loss (ipsilateral trigeminal tract taken out)

ipsilateral pharyngeal and laryngeal paralysis from cranial glossopharyngeal and vagus palsies

IMPORTANTLY, contralateral pain and temperature sensory loss (spinothalamic tract taken out. Remember, this crossed over well before the medulla, down at the level of the cord itself).

With all brainstem strokes, the side of the lesion is the side of the cranial nerve sign. Ie...loss of sensation on the left side of the face, for example, but with a loss of sensation of the right side of the body...still tells you that the lesion is on the left because that is the side of the cranial nerve sign. In a left-sided 'ventral pontine syndrome' (Millard Gubler), for example, you get ipsilateral facial weakness (the left facial nerve has gone -ie the cranial nerve sign) with contralateral body hemiparesis (the right corticospinal tract has been taken out).

The management of vertebral artery dissection is actually anticoagulation (once bleed excluded by scans). This is intended to prevent further thromboembolic complications (here, for example, our patient has had a subsequent brainstem stroke). Note that there are actually no randomised controlled trials to support the use of anticoagulation here, but its what people do. Antiplatelet therapy is a reasonable option to consider in patients who are suspected of suffering from a vertebral artery dissection while awaiting definitive investigations. So here, if a plain CT excluded bleed, then you clearly have a stroke and aspirin would be a reasonable choice whilst you wait for you CT angiogram. Note plain CT would not establish the diagnosis. In order to visualise the dissected vessel, you need an angiogram.

👍 👎 Discuss (7) Improve

Next question >

Stroke by anatomy ★

Site of the lesion	Associated effects
Anterior cerebral artery	Contralateral hemiparesis and sensory loss, lower extremity > upper
Middle cerebral artery	Contralateral hemiparesis and sensory loss, upper extremity > lower Contralateral homonymous hemianopia Aphasia
Posterior cerebral artery	Contralateral homonymous hemianopia with macular sparing Visual agnosia

Site of the lesion	**Associated effects**
Weber's syndrome (branches of the posterior cerebral artery that supply the midbrain)	Ipsilateral CN III palsy Contralateral weakness of upper and lower extremity
Posterior inferior cerebellar artery (lateral medullary syndrome, Wallenberg syndrome)	Ipsilateral: facial pain and temperature loss Contralateral: limb/torso pain and temperature loss Ataxia, nystagmus
Anterior inferior cerebellar artery (lateral pontine syndrome)	Symptoms are similar to Wallenberg's (see above), but: Ipsilateral: facial paralysis and deafness
Retinal/ophthalmic artery	Amaurosis fugax
Basilar artery	'Locked-in' syndrome

Lacunar strokes

- present with either isolated hemiparesis, hemisensory loss or hemiparesis with limb ataxia
- strong association with hypertension
- common sites include the basal ganglia, thalamus and internal capsule

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 👍 75 👎 10

Brainstem Rules of 4

Suggest link Report broken link

Media

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 0 👎 0

Brainstem Stroke

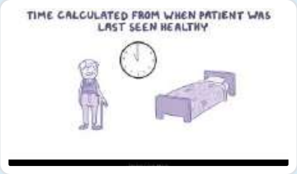
Brainstem stroke syndromes

Soton Brain Hub - YouTube 👍 14 👎 1

NEUROLOGY
MIDBRAIN SYNDROMES

Midbrain Syndromes - Weberâ€™s Syndrome, Benediktâ€™s Syndrome and Parinaud Syndrome

Armando Hasudungan - YouTube 👍 2 👎 1


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 1 👎 1

Report broken media

Score: **24.8%**

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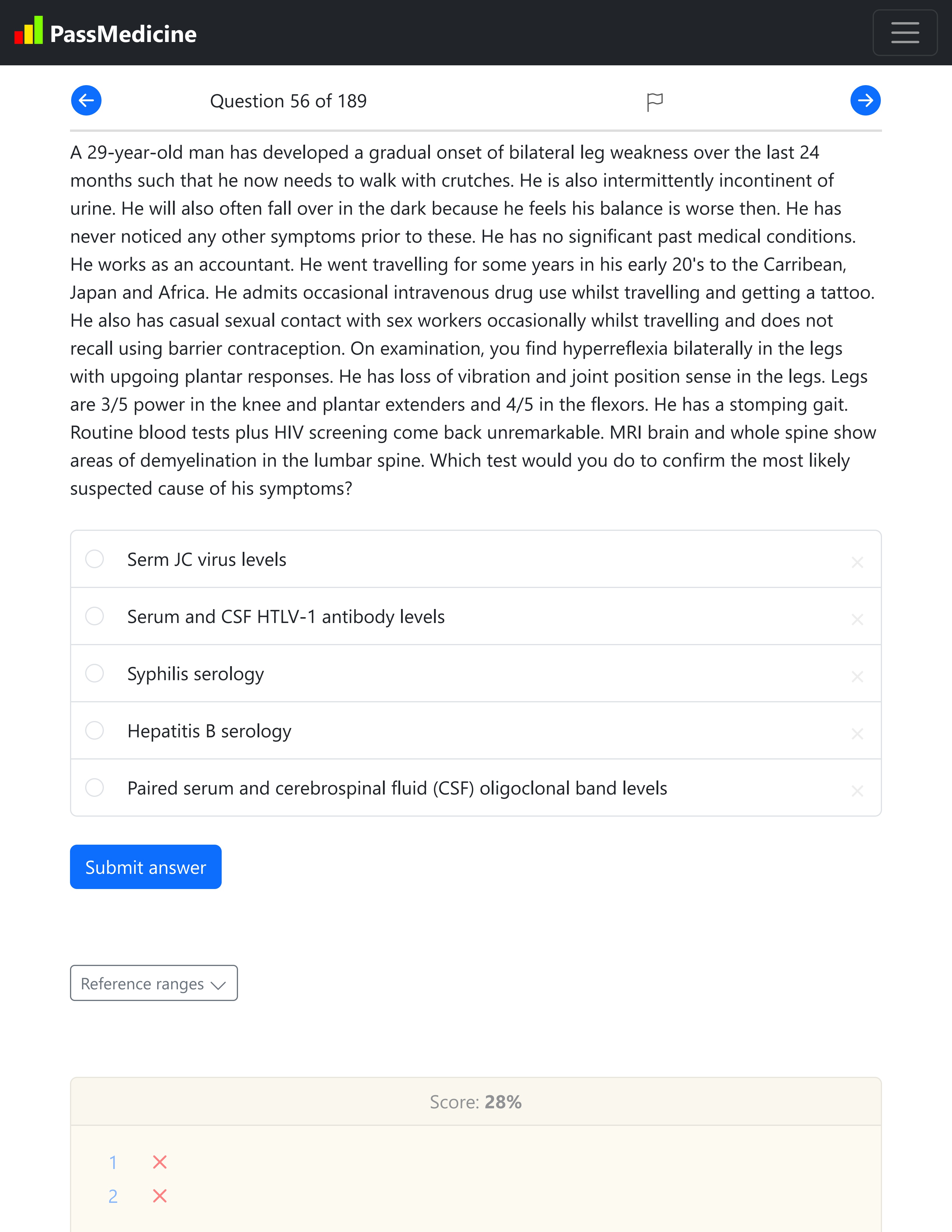

← Question 56 of 189 →

A 29-year-old man has developed a gradual onset of bilateral leg weakness over the last 24 months such that he now needs to walk with crutches. He is also intermittently incontinent of urine. He will also often fall over in the dark because he feels his balance is worse then. He has never noticed any other symptoms prior to these. He has no significant past medical conditions. He works as an accountant. He went travelling for some years in his early 20's to the Carribean, Japan and Africa. He admits occasional intravenous drug use whilst travelling and getting a tattoo. He also has casual sexual contact with sex workers occasionally whilst travelling and does not recall using barrier contraception. On examination, you find hyperreflexia bilaterally in the legs with upgoing plantar responses. He has loss of vibration and joint position sense in the legs. Legs are 3/5 power in the knee and plantar extenders and 4/5 in the flexors. He has a stomping gait. Routine blood tests plus HIV screening come back unremarkable. MRI brain and whole spine show areas of demyelination in the lumbar spine. Which test would you do to confirm the most likely suspected cause of his symptoms?

- ○ Serm JC virus levels
- ○ Serum and CSF HTLV-1 antibody levels
- ○ Syphilis serology
- ○ Hepatitis B serology
- ○ Paired serum and cerebrospinal fluid (CSF) oligoclonal band levels

Score: **28%**

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A 29-year-old man has developed a gradual onset of bilateral leg weakness over the last 24 months such that he now needs to walk with crutches. He is also intermittently incontinent of urine. He will also often fall over in the dark because he feels his balance is worse then. He has never noticed any other symptoms prior to these. He has no significant past medical conditions. He works as an accountant. He went travelling for some years in his early 20's to the Carribean, Japan and Africa. He admits occasional intravenous drug use whilst travelling and getting a tattoo. He also has casual sexual contact with sex workers occasionally whilst travelling and does not recall using barrier contraception. On examination, you find hyperreflexia bilaterally in the legs with upgoing plantar responses. He has loss of vibration and joint position sense in the legs. Legs are 3/5 power in the knee and plantar extenders and 4/5 in the flexors. He has a stomping gait. Routine blood tests plus HIV screening come back unremarkable. MRI brain and whole spine show areas of demyelination in the lumbar spine. Which test would you do to confirm the most likely suspected cause of his symptoms?

Serm JC virus levels	16%
Serum and CSF HTLV-1 antibody levels	29%
Syphilis serology	37%
Hepatitis B serology	1%
Paired serum and cerebrospinal fluid (CSF) oligoclonal band levels	16%

The diagnosis is tropical spastic paraparesis (TSP).

This is otherwise called HTLV-1 Associated Myelopathy (HAM), describing how this demyelinating condition is caused by infection with HTLV-1. HTLV-I is a retrovirus endemic in southern Japan, equatorial Africa and South America. Transmission occurs through sexual or other intimate contacts, intrauterine, breastfeeding, sharing of needles by drug users, or blood transfusion from infected persons. This patient has many of these risk factors from his time travelling.

The incubation period between infection and symptomatic disease may be a few months up to decades and actually, the majority of infected individuals remain lifelong asymptomatic carriers. It is not clear what determines whether or not one becomes symptomatic but the key is suspected to be bigger viral loads resulting in progression to symptoms.

The presentation is with progressive upper motor neurone symptoms and signs particularly confined to the lower limbs (see below for differentials of such a presentation.

The key to knowing which of these differentials is the likely cause here is understanding the risk factors and ordering the correct tests (e.g. not missing out an HIV test); as the signs and symptoms are fairly similar in all of these upper motor neurone conditions.

Discuss (6) | Improve

Next question

Spastic paraparesis

Spastic paraparesis describes a upper motor neuron pattern of weakness in the lower limbs

Causes
- demyelination e.g. multiple sclerosis
- cord compression: trauma, tumour
- parasagittal meningioma
- tropical spastic paraparesis
- transverse myelitis e.g. HIV
- syringomyelia
- hereditary spastic paraplegia
- osteoarthritis of the cervical spine

+ Q 123

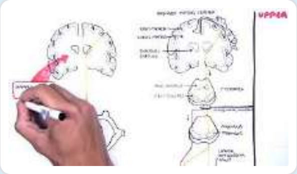
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Introduction to Upper and Lower Motor Neuron Lesions

Armando Hasudungan - YouTube 👍 2 👎 0

Report broken media

Score: **24.8%**

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Question 57 of 189

A 35-year-old lady with no significant past medical history presents to you with lethargy and fatigue over several years, often finding she does not sleep well at night. She complains of a sensation of discomfort in her lower extremities at rest, particularly worse when she is trying to fall asleep. She also describes an abnormal crawling and itching sensation below the knees and often walking will relieve her symptoms. There is no history of pain or nigh time snoring. Clinical examination and routine blood investigations are unremarkable. She tells you that over the years her general practitioner has ordered several tests on her that have all been normal, including brain imaging, thyroid function, and monitoring of her haemoglobin levels. She is not on any medications. She is now struggling to do her job as a teacher and is now working only part time because of the symptoms. Considering the likely underlying diagnosis, which of the following would you use to try and alleviate her symptoms?

- ○ Lithium ×
- ○ Quinine ×
- ○ Pramipexole ×
- ○ Amitriptyline ×
- ○ Acetazolamide ×

Submit answer

Reference ranges

Score: **28%**

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Lithium	1%
Quinine	14%
Pramipexole	61%
Amitriptyline	22%
Acetazolamide	1%

The diagnosis is restless leg syndrome (RLS). This is a neurologic movement disorder of the limbs that is often associated with a sleep complaint. This will often end up presenting in neurology clinics and it is important to know how to help such patients. RLS can lead to significant physical and emotional disability.

Specific DSM-5 criteria for RLS are as follows:

- an urge to move the legs that is usually accompanied by or occurs in response to uncomfortable and unpleasant sensations in the legs, characterized by all of the following: (1) the urge to move the legs begins or worsens during periods of rest or inactivity; (2) the urge is partially or totally relieved by movement; and (3) the urge to move legs is worse in the evening or at night than during the day or occurs only in the evening or at night.
- Symptoms occur at least 3 times per week and have persisted for at least 3 months.
- Symptoms cause significant distress or impairment in social, occupational, educational, academic, behavioural or other areas of functioning.

- The symptoms cannot be attributed to another mental disorder or medical condition (e.g., leg oedema, arthritis, leg cramps) or behavioural condition (e.g. positional discomfort, habitual foot tapping)
- The disturbance cannot be explained by the effects of a drug of abuse or medication

85% of patients with RLS have periodic movements of sleep, usually involving the legs (periodic leg movements of sleep [PLMS]). This is characterised by involuntary, forceful dorsiflexion of the foot lasting 0.5-5 seconds and occurring every 20-40 seconds throughout sleep. It is, therefore, worth taking a collateral history from a partner if available.

Investigations should include excluding iron deficiency (which may potentiate RLS) and you should order sleep studies to characterise the extent of sleep disturbance.

First line drug treatments are dopaminergic agents (eg, pramipexole, ropinirole, bromocriptine, levodopa-carbidopa, and rotigotine) or gabapentin/pregabalin. Nonpharmacologic approaches include exercise, avoidance of caffeine, alcohol, and nicotine. Also important to try and stop medications that exacerbate RLS if possible, eg selective serotonin reuptake inhibitors (SSRIs), serotonin-norepinephrine reuptake inhibitors (SNRIs), diphenhydramine, and dopamine antagonists.

Discuss (2) Improve

Next question ›

Restless legs syndrome ★

Restless legs syndrome (RLS) is a syndrome of spontaneous, continuous lower limb movements that may be associated with paraesthesia. It is extremely common, affecting between 2-10% of the general population. Males and females are equally affected and a family history may be present

Clinical features
- uncontrollable urge to move legs (akathisia). Symptoms initially occur at night but as condition progresses may occur during the day. Symptoms are worse at rest
- paraesthesias e.g. 'crawling' or 'throbbing' sensations
- movements during sleep may be noted by the partner - periodic limb movements of sleeps (PLMS)

Causes and associations
- there is a positive family history in 50% of patients with idiopathic RLS

- iron deficiency anaemia
- uraemia
- diabetes mellitus
- pregnancy

The diagnosis is clinical although bloods such as ferritin to exclude iron deficiency anaemia may be appropriate

Management
- simple measures: walking, stretching, massaging affected limbs
- treat any iron deficiency
- dopamine agonists are first-line treatment (e.g. Pramipexole, ropinirole)
- benzodiazepines
- gabapentin

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Sumarries 👍 8 👎 5

Restless legs syndrome

Suggest link

Report broken link

Media

IRON STUDIES
50% HAEMODIALYSIS
RESTLESS LEGS SYNDROME
WORSE AT NIGHT
1/3 PREGNANCIES

Restless legs syndrome

Rhesus Medicine - YouTube 👍 0 👎 1

Report broken media

Score: **24.8%**

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Question 58 of 189

A 75-year-old woman presents with a 24-hour history of gradual onset, severe 10/10, frontal headache associated with double vision. The history is otherwise unremarkable. On examination, she has a partial ptosis of the right eye and a pupil resting outwards and downwards with a dilated sluggishly reacting pupil. There is a failure of intorsion of the right eye. Neurological and physical examination are otherwise unremarkable. She has normal routine blood tests and a normal plain CT head. What is the most important next investigation?

- ○ Lumbar puncture
- ○ CT angiogram cerebral vessels.
- ○ Carotid artery biopsy
- ○ Indomethacin trial
- ○ Single fibre EMG of the ocular muscles

Submit answer

Reference ranges

Score: **28%**

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Lumbar puncture	9%
CT angiogram cerebral vessels.	76%
Carotid artery biopsy	2%
Indomethacin trial	6%
Single fibre EMG of the ocular muscles	7%

Presentation with a painful third nerve palsy is a neurosurgical emergency that normally presents to the medics as it may indicate an expanding posterior communicating artery aneurysm. Urgent CT angiogram of the cerebral vessels is required for diagnosis. Management is with surgical clipping. If this does not happen quickly, there is a risk of death from rupture or there may be permanent ptosis and eye movement deficit.

It is important to be able to differentiate between surgical causes of third nerve palsy (eg the above, or a tumour), and medical causes, eg diabetes or vasculitis. The surgical causes indicate an emergency whilst the medical causes are likely chronic conditions requiring optimisation of medical management. The parasympathetic fibres of this nerve are on the periphery of the nerve and the sympathetic fibres are in its centre. Therefore, a fixed dilated or sluggish pupil indicates that the parasympathetic fibres have been wiped out (eg by external compression from an aneurysm or tumour), leaving unopposed sympathetic tract flow within this nerve (dilating the pupil). The blood supply to the nerve is within its internal portion, meaning medical causes like diabetes or vasculitis wipe out the sympathetic portion of the nerve, leaving unopposed parasympathetic flow and consequently a constricted pupil.

The trochlear nerve sits right next to the oculomotor nerve, meaning if a structural cause is big enough, it will take the trochlear nerve out too. The trochlear nerve is responsible for intorsion of the eye (ie internal rotation in the plane of the face). So when you test for a 3rd nerve palsy, you should also test for a 4th nerve palsy by asking the patient to follow your finger as you draw a half-circle from out to in (anti-clockwise): ie your 9'oclock to 3 o'clock. If the eye fails to follow, this is a failure of intorsion.

Discuss (5) Improve

Next question

Third nerve palsy

Features

- eye is deviated 'down and out'
- ptosis
- pupil may be dilated (sometimes called a 'surgical' third nerve palsy)

Causes

- diabetes mellitus
- vasculitis e.g. temporal arteritis, SLE
- false localizing sign* due to uncal herniation through tentorium if raised ICP
- posterior communicating artery aneurysm
  - pupil dilated
  - often associated pain
- cavernous sinus thrombosis
- Weber's syndrome: ipsilateral third nerve palsy with contralateral hemiplegia -caused by midbrain strokes
- other possible causes: amyloid, multiple sclerosis

*this term is usually associated with sixth nerve palsies but it may be used for a variety of neurological presentations

AI + Q 123

Next question

B I A

Textbooks

High-yield textbook

Extended textbook

Media

Third Cranial Nerve Palsy

U-M Kellogg Eye Center - YouTube 👍 6 👎 0

3rd nerve palsy

Individual - YouTube 👍 6 👎 1

OphthoBook.com

Cranial nerve palsies

Tim Root - YouTube 👍 5 👎 2

Report broken media

Score: **24.8%**

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Question 59 of 189

A 75-year-old man presents with slowly progressive dyspnoea. There is no cough or chest pain. He is a non-smoker with a background of Parkinson's disease, rheumatoid arthritis, type 2 diabetes mellitus and atrial fibrillation. Peak flow investigations demonstrate a reduced Forced Vital Capacity (FVC), a Forced Expiratory Volume (FEV1):FVC ratio of 90%, and a low transfer factor for carbon monoxide (TLCO). High-resolution CT confirms ground-glass changes. Which of his following medications is the most likely contributor to his lung-changes?

- ○ Azathioprine
- ○ Levodopa
- ○ Digoxin
- ○ Cabergoline
- ○ Exenatide

Submit answer

Reference ranges

Score: **28%**

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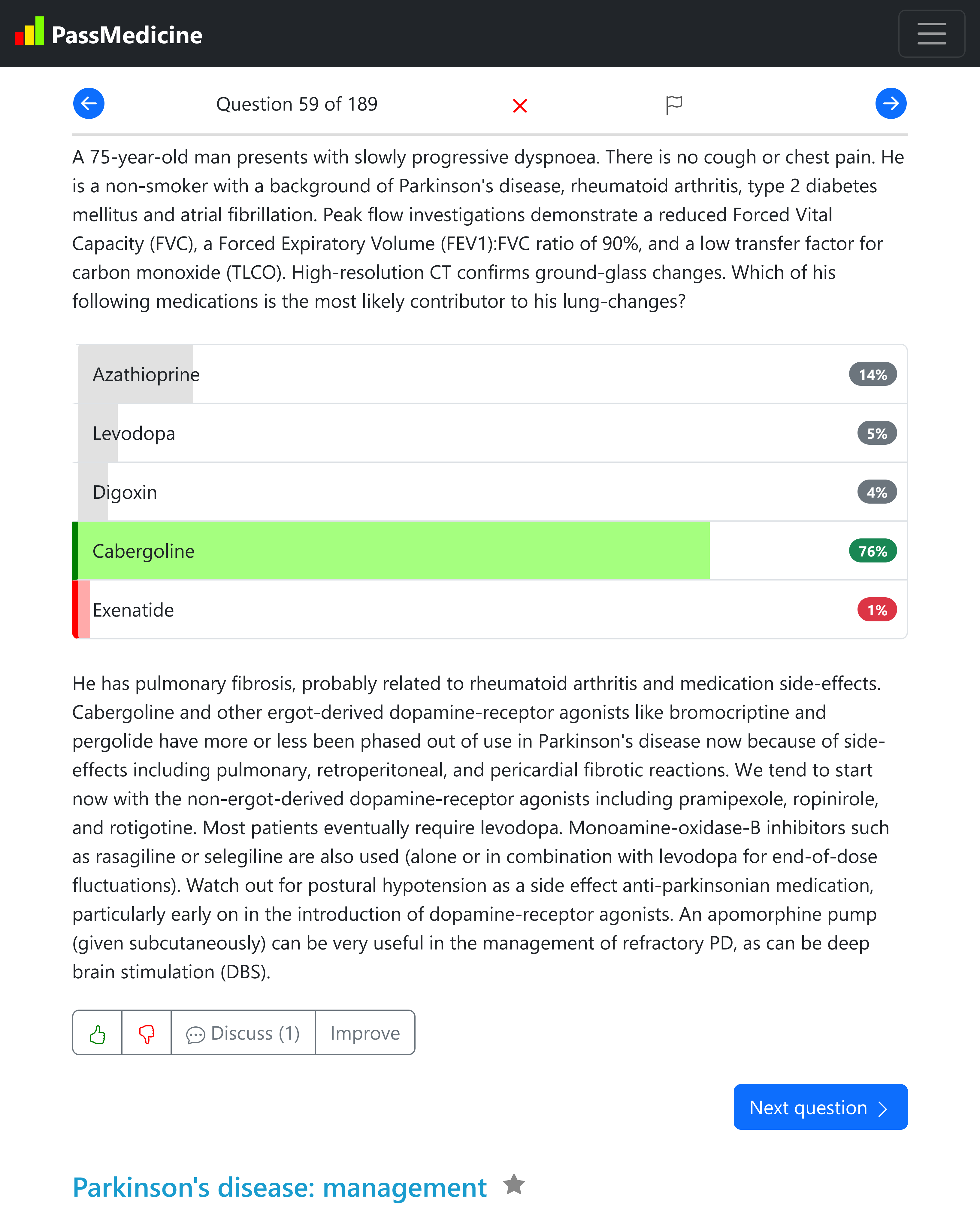


A 75-year-old man presents with slowly progressive dyspnoea. There is no cough or chest pain. He is a non-smoker with a background of Parkinson's disease, rheumatoid arthritis, type 2 diabetes mellitus and atrial fibrillation. Peak flow investigations demonstrate a reduced Forced Vital Capacity (FVC), a Forced Expiratory Volume (FEV1):FVC ratio of 90%, and a low transfer factor for carbon monoxide (TLCO). High-resolution CT confirms ground-glass changes. Which of his following medications is the most likely contributor to his lung-changes?

Azathioprine	14%
Levodopa	5%
Digoxin	4%
Cabergoline	76%
Exenatide	1%

He has pulmonary fibrosis, probably related to rheumatoid arthritis and medication side-effects. Cabergoline and other ergot-derived dopamine-receptor agonists like bromocriptine and pergolide have more or less been phased out of use in Parkinson's disease now because of side-effects including pulmonary, retroperitoneal, and pericardial fibrotic reactions. We tend to start now with the non-ergot-derived dopamine-receptor agonists including pramipexole, ropinirole, and rotigotine. Most patients eventually require levodopa. Monoamine-oxidase-B inhibitors such as rasagiline or selegiline are also used (alone or in combination with levodopa for end-of-dose fluctuations). Watch out for postural hypotension as a side effect anti-parkinsonian medication, particularly early on in the introduction of dopamine-receptor agonists. An apomorphine pump (given subcutaneously) can be very useful in the management of refractory PD, as can be deep brain stimulation (DBS).

Parkinson's disease: management

Parkinsons disease should only be diagnosed, and management initiated, by a specialist with expertise in movement disorders. However, it is important for all doctors to be aware of the medications used in Parkinson's given the prevalence of this condition. NICE published guidelines in 2017 regarding the management of Parkinson's disease.

For first-line treatment:

- if the motor symptoms are affecting the patient's quality of life: levodopa
- if the motor symptoms are not affecting the patient's quality of life: dopamine agonist (non-ergot derived), levodopa or monoamine oxidase B (MAO-B) inhibitor

Whilst all drugs used to treat Parkinson's can cause a wide variety of side-effects NICE produced tables to help with decision making:

	Levodopa	**Dopamine agonists**	**MAO-B inhibitors**
Motor symptoms	More improvement in motor symptoms	Less improvement in motor symptoms	Less improvement in motor symptoms
Activities of daily living	More improvement in activities of daily living	Less improvement in activities of daily living	Less improvement in activities of daily living
Motor complications	More motor complications	Fewer motor complications	Fewer motor complications
Adverse events	Fewer specified adverse events*	More specified adverse events*	Fewer specified adverse events*

* excessive sleepiness, hallucinations and impulse control disorders

If a patient continues to have symptoms despite optimal levodopa treatment or has developed dyskinesia then NICE recommend the addition of a dopamine agonist, MAO-B inhibitor or catechol-O-methyl transferase (COMT) inhibitor as an adjunct. Again, NICE summarise the main points in terms of decision making:

	Dopamine agonists	**MAO-B inhibitors**	**COMT inhibitors**	**Amantadine**
Motor symptoms	Improvement in motor symptoms	Improvement in motor symptoms	Improvement in motor symptoms	No evidence of improvement in motor symptoms

	Dopamine agonists	MAO-B inhibitors	COMT inhibitors	Amantadine
Activities of daily living	Improvement in activities of daily living	Improvement in activities of daily living	Improvement in activities of daily living	No evidence of improvement in activities of daily living
Off time	More off-time reduction	Off-time reduction	Off-time reduction	No studies reporting this outcome
Adverse events	Intermediate risk of adverse events	Fewer adverse events	More adverse events	No studies reporting this outcome
Hallucinations	More risk of hallucinations	Lower risk of hallucinations	Lower risk of hallucinations	No studies reporting this outcome

Specific points regarding Parkinson's medication

NICE reminds us of the risk of acute akinesia or neuroleptic malignant syndrome if medication is not taken/absorbed (for example due to gastroenteritis) and advise against giving patients a 'drug holiday' for the same reason.

Impulse control disorders have become a significant issue in recent years. These can occur with any dopaminergic therapy but are more common with:

- dopamine agonist therapy
- a history of previous impulsive behaviours
- a history of alcohol consumption and/or smoking

If excessive daytime sleepiness develops then patients should not drive. Medication should be adjusted to control symptoms. Modafinil can be considered if alternative strategies fail.

If orthostatic hypotension develops then a medication review looking at potential causes should be done. If symptoms persist then midodrine (acts on peripheral alpha-adrenergic receptors to increase arterial resistance) can be considered.

Consider glycopyrronium bromide to manage drooling of saliva in people with Parkinson's

disease.

Further information regarding specific anti-Parkinson's medication

Levodopa

- nearly always combined with a decarboxylase inhibitor (e.g. carbidopa or benserazide)
  - this prevents the peripheral metabolism of levodopa to dopamine outside of the brain and hence can reduce side effects
- common adverse effects:
  - dry mouth
  - anorexia
  - palpitations
  - postural hypotension
  - psychosis
- some adverse effects are due to the difficulty in achieving a steady dose of levodopa
  - end-of-dose wearing off: symptoms often worsen towards the end of dosage interval. This results in a decline of motor activity
  - 'on-off' phenomenon: large variations in motor performance, with normal function during the 'on' period, and weakness and restricted mobility during the 'off' period
  - dyskinesias at peak dose: dystonia, chorea and athetosis (involuntary writhing movements)
  - these effects may worsen over time with - clinicians therefore may limit doses until necessary
- it is important not to acutely stop levodopa, for example, if a patient is admitted to hospital
  - if a patient with Parkinson's disease cannot take levodopa orally, they can be given a dopamine agonist patch as rescue medication to prevent acute dystonia

Dopamine receptor agonists

- e.g. bromocriptine, ropinirole, cabergoline, apomorphine
- ergot-derived dopamine receptor agonists (bromocriptine, cabergoline) have been associated with pulmonary, retroperitoneal and cardiac fibrosis. The Committee on Safety of Medicines advice that an echocardiogram, ESR, creatinine and chest x-ray should be obtained prior to treatment and patients should be closely monitored
- patients should be warned about the potential for dopamine receptor agonists to cause impulse control disorders and excessive daytime somnolence
- more likely than levodopa to cause hallucinations in older patients. Nasal congestion and postural hypotension are also seen in some patients

MAO-B (Monoamine Oxidase-B) inhibitors

- e.g. selegiline
- inhibits the breakdown of dopamine secreted by the dopaminergic neurons

Amantadine

- mechanism is not fully understood, probably increases dopamine release and inhibits its uptake at dopaminergic synapses
- side-effects include ataxia, slurred speech, confusion, dizziness and livedo reticularis

COMT (Catechol-O-Methyl Transferase) inhibitors

- e.g. entacapone, tolcapone
- COMT is an enzyme involved in the breakdown of dopamine, and hence may be used as an adjunct to levodopa therapy
- used in conjunction with levodopa in patients with established PD

Antimuscarinics

- block cholinergic receptors
- now used more to treat drug-induced parkinsonism rather than idiopathic Parkinson's disease
- help tremor and rigidity
- e.g. procyclidine, benzotropine, trihexyphenidyl (benzhexol)

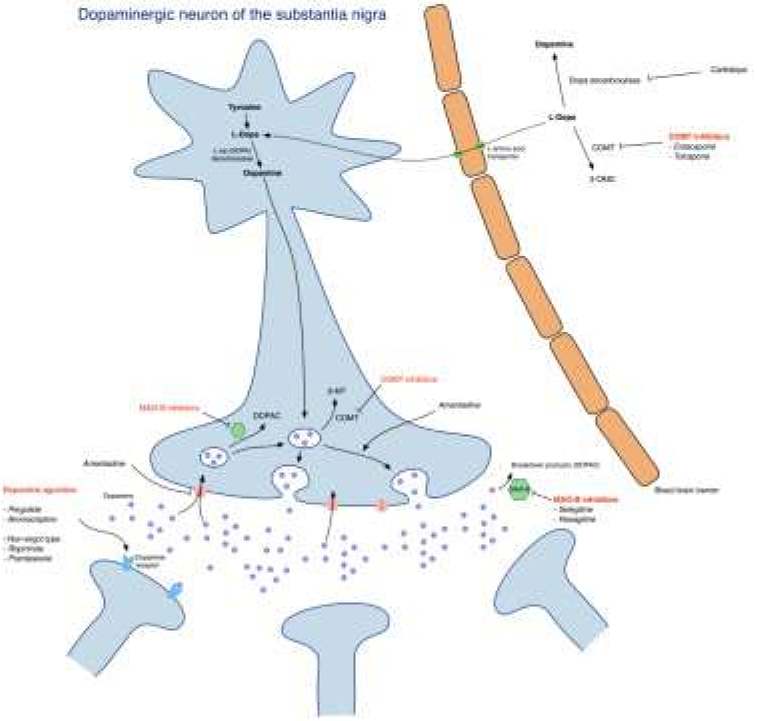

Dopaminergic neuron of the substantia nigra
Tyrosine
L-Dopa
Dopamine
Dopa decarboxylase
Carbidopa
L-Dopa
COMT
COMT inhibitors
- Entacapone
- Tolcapone
3-OMD
COMT inhibitors
3-MT
COMT
DOPAC
MAO-B inhibitors
Amantadine
Amantadine
Dopamine agonists
Dopamine
Breakdown products (DOPAC)
MAO-B
MAO-B inhibitors
- Selegiline
- Rasagiline
Blood brain barrier

Image sourced from Wikipedia

Diagram showing the mechanism of action of Parkinson's drugs

123

Next question

Textbooks

High-yield textbook

Extended textbook

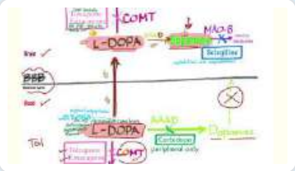
Links

NICE 👍 23 👎 12

2017 Parkinson's disease guidelines

Suggest link Report broken link

Media


COMT
MAO-B
L-DOPA
BBB
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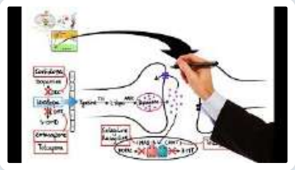
The use of Levodopa/ Carbidopa in the treatment of Parkinson's disease

Medicosis Perfectionalis - YouTube 👍 13 👎 0

PARKINSON'S
DISEASE
PHARMACOLOGY

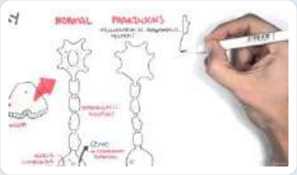
Parkinson's disease pharmacology

Soton Brain Hub - YouTube 👍 4 👎 0



Pharmacology - DRUGS FOR PARKINSON'S DISEASE

Speed Pharmacology - YouTube 👍 8 👎 1


NORMAL
PARKINSON'S

Pharmacology - Parkinson's Disease

Armando Hasudungan - YouTube 👍 16 👎 2

PARKINSON'S DISEASE
Osmosis.org

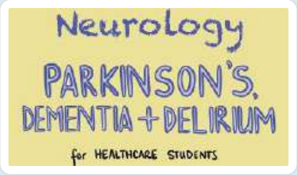
Parkinson's disease

Osmosis - YouTube 👍 4 👎 1

PARKINSON'S DISEASE
SHUFFLING GAIT
PILL ROLLING TREMOR
COG-WHEEL RIGIDITY
DaT SCAN

Understanding Parkinson's Disease

Rhesus Medicine - YouTube 👍 2 👎 1


Neurology
PARKINSON'S, DEMENTIA + DELIRIUM
for HEALTHCARE STUDENTS

Parkinson's disease

MedFlix - YouTube 👍 1 👎 2

Report broken media

Score: **24.8%**

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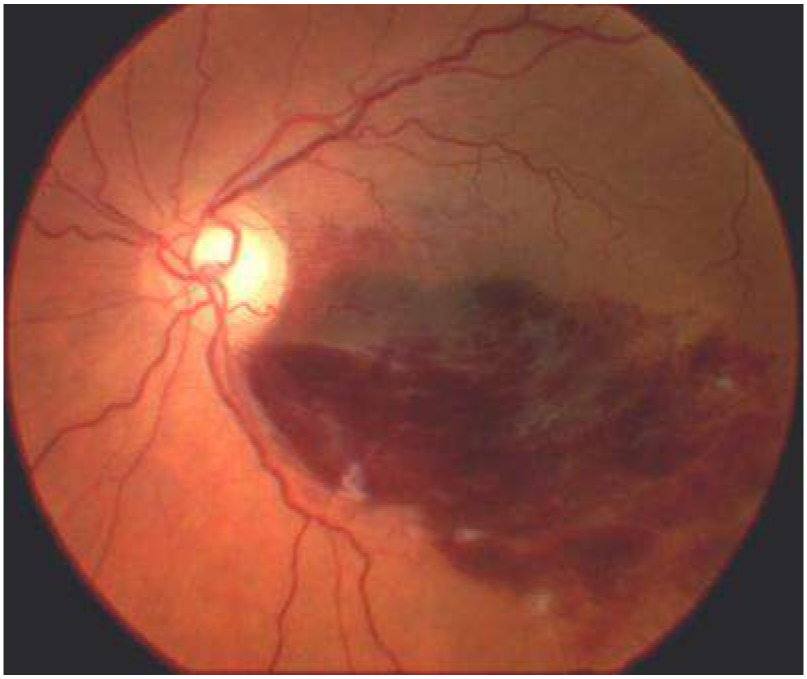
Question 60 of 189

A 62-year-old woman presents to the emergency eye unit with a 1-day history of sudden loss of vision in her left eye. She denies pain or previous concerns with her vision. Her past medical history includes hypertension and type 2 diabetes for which she takes ramipril and metformin, respectively.

On examination, there is reduced visual acuity in her left eye and she is only able to make out hand movements. The visual acuity in her right arm is unchanged from 6/9.

Fundoscopic examination of her left eye is shown below:



What is the most likely diagnosis?

- ○ Diabetic retinopathy
- ○ Hypertensive retinopathy
- ○ Retinal artery occlusion
- ○ Retinal vein occlusion
- ○ Retinitis pigmentosa

Submit answer

Reference ranges ∨

Score: **28%**

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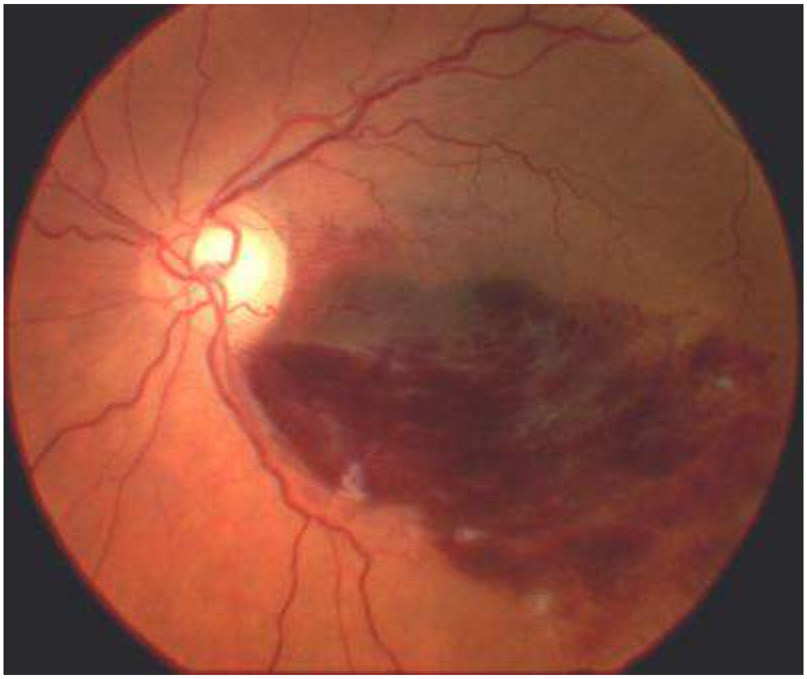

What is the most likely diagnosis?

Diabetic retinopathy	7%
Hypertensive retinopathy	11%
Retinal artery occlusion	6%
Retinal vein occlusion	72%
Retinitis pigmentosa	4%

This patient has a **retinal vein occlusion** characterised by a sudden onset of painless loss of vision. Risk factors for retinal vein occlusion include hypertension and type 2 diabetes and are more common in the middle-aged. The fundoscopic examination shows dilated tortuous veins and intraretinal haemorrhage secondary to occlusion of the retinal vein. Other features that can be seen include cotton wool spots, dot and blot haemorrhages and macular oedema.

Hypertensive retinopathy is incorrect. Hypertensive retinopathy is typically associated with arteriolar narrowing, cotton wool spots, retinal haemorrhages and hard exudates. These are not seen in this fundoscopic examination, making the diagnosis unlikely.

Diabetic retinopathy is incorrect. Diabetic retinopathy is a reasonable differential diagnosis given the past medical history of type 2 diabetes. However, it is unlikely to cause sudden onset visual loss. Rather, diabetic retinopathy causes gradual visual loss and can be staged from pre-proliferative, proliferative and maculopathy. Fundoscopic features include cotton wool spots, microaneurysms ane neovascularisation which, in turn, can lead to retinal detachment and vitreous haemorrhage.

Retinal artery occlusion is incorrect. Retinal artery occlusion is another common cause of sudden onset painless vision loss, frequently from embolic phenomenon. However, fundoscopic findings of retinal artery occlusion include a pale retina with a classic 'cherry-red spot' (the unaffected vascular choroid at the centre of the macula). Cotton wool spots may also be seen.

Retinitis pigmentosa is incorrect. Retinitis pigmentosa is an inherited disorder of the retina that is characterised by degeneration of the rod cells (responsible for night vision). In retinitis pigmentosa, there is bone-spicule deposition within the periphery of the retina. This is not seen here, nor is there a history of night blindness or reduced peripheral vision.

👍 👎 💬 Discuss (3) Improve

Next question >

Sudden loss of vision ★

Sudden loss is a frightening symptom for patients. It may represent an ongoing issue or only be temporary. The term transient monocular visual loss (TMVL) describes a sudden, transient loss of vision that lasts less than 24 hours.

The most common causes of a sudden painless loss of vision are as follows:

- ischaemic/vascular (e.g. thrombosis, embolism, temporal arteritis etc). This includes recognised syndromes e.g. occlusion of central retinal vein and occlusion of central retinal

artery
- vitreous haemorrhage
- retinal detachment
- retinal migraine

Ischaemic/vascular
- often referred to as 'amaurosis fugax'
- wide differential including large artery disease (atherothrombosis, embolus, dissection), small artery occlusive disease (anterior ischemic optic neuropathy, vasculitis e.g. temporal arteritis), venous disease and hypoperfusion
- may represent a form of transient ischaemic attack (TIA). It should therefore be treated in a similar fashion, with aspirin 300mg being given
- altitudinal field defects are often seen: 'curtain coming down'
- ischaemic optic neuropathy is due to occlusion of the short posterior ciliary arteries, causing damage to the optic nerve

Central retinal vein occlusion
- incidence increases with age, more common than arterial occlusion
- causes: glaucoma, polycythaemia, hypertension
- severe retinal haemorrhages are usually seen on fundoscopy

Central retinal artery occlusion
- due to thromboembolism (from atherosclerosis) or arteritis (e.g. temporal arteritis)
- features include afferent pupillary defect, 'cherry red' spot on a pale retina

Vitreous haemorrhage
- causes: diabetes, bleeding disorders, anticoagulants
- features may include sudden visual loss, dark spots

Retinal detachment
- features of vitreous detachment, which may precede retinal detachment, include flashes of light or floaters (see below)

Differentiating posterior vitreous detachment, retinal detachment and vitreous haemorrhage

Posterior vitreous detachment	**Retinal detachment**	**Vitreous haemorrhage**
Flashes of light (photopsia) - in the peripheral field of vision Floaters, often on the temporal side of the central vision	Dense shadow that starts peripherally progresses towards the central vision A veil or curtain over the field of vision Straight lines appear curved Central visual loss	Large bleeds cause sudden visual loss Moderate bleeds may be described as numerous dark spots Small bleeds may cause floaters

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

The Royal Australian College of General Practitioners 👍 16 👎 20

Sudden loss of vision review

Patient.info 👍 14 👎 11

Retinal artery occlusions

American Academy of Ophthalmology 👍 16 👎 10

Central Retinal Vein Occlusion

American Academy of Ophthalmology 👍 15 👎 7

Central Retinal Artery Occlusion

Suggest link

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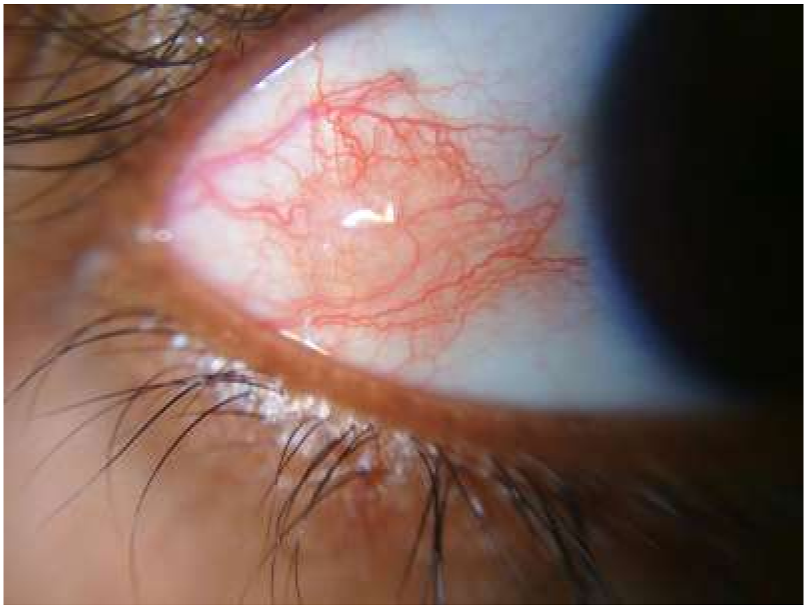
A 27-year-old man presents to the general practitioner with a 1-day history of red eyes. He reports noticing them whilst brushing his teeth in the morning. He complains that his eyes are mildly sore but manageable without the need for analgesia. There is no history of itch and he otherwise feels well in himself. His past medical history includes ankylosing spondylitis.

On examination, his visual acuity is 6/6 bilaterally. His eyes are examined, as shown below:



Given the diagnosis, what is the recommended management for this patient?

- ○ Acetazolamide
- ○ Reassurance and NSAIDs
- ○ Topical aciclovir
- ○ Topical chloramphenicol
- ○ Topical corticosteroids

Submit answer

Reference ranges ⌵

Score: **28%**

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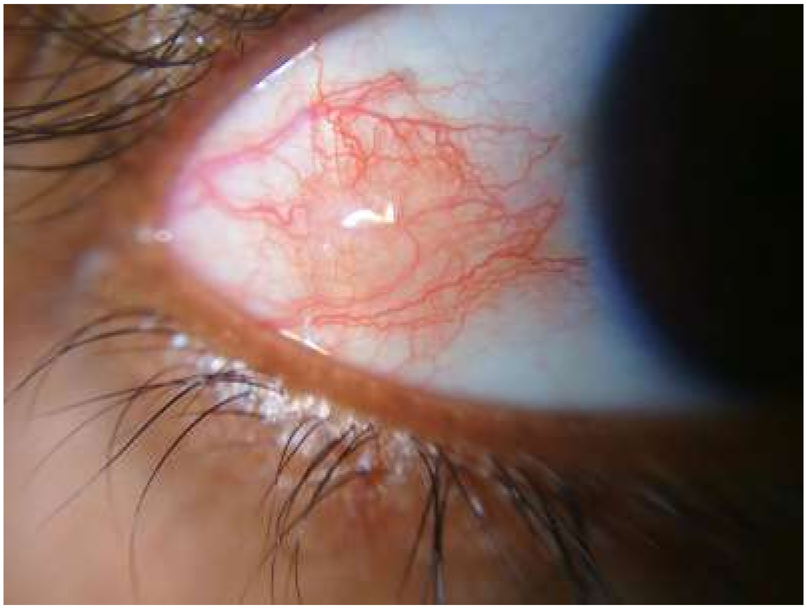


Given the diagnosis, what is the recommended management for this patient?

Acetazolamide	2%
🟩 Reassurance and NSAIDs	64%
Topical aciclovir	2%
Topical chloramphenicol	3%
🟥 Topical corticosteroids	30%

This patient has a diagnosis of episcleritis. Episcleritis is the acute inflammation of the episcleral tissues and presents as an acute-onset red eye that may be mildly irritable but should not be painful or associated with vision loss. Examination reveals dilated episcleral vessels on the surface. Most cases are idiopathic, but risk factors include inflammatory bowel disease, seronegative spondyloarthropathies and vasculitides. The condition is self-limiting and **no treatment is usually necessary**. However, the use of **NSAIDs** is sometimes recommended for the active inflammation and treatment of any discomfort, and artificial tears may also be used for the relief of symptoms.

Acetazolamide is incorrect. Acetazolamide is a carbonic anhydrase inhibitor used in the treatment of acute angle closure glaucoma. Features of acute angle closure glaucoma include a unilateral painful red eye associated with photophobia, headache and vomiting. The pupil may be partially fixed and dilated with a hazy cornea. This patient's presentation is not in keeping with this diagnosis.

Topical aciclovir is incorrect. Topical aciclovir would be indicated for an acute herpetic infection of the eye. However, the mild symptoms that this patient is presenting with make an infective cause of his symptoms unlikely.

Topical chloramphenicol is incorrect. This would be the treatment of choice for a patient with bacterial conjunctivitis. Features of bacterial conjunctivitis include an itchy, watering and gritty red eye associated with blurred vision and purulent discharge with or without crusting of the eyelashes. This is not seen in this case, making conjunctivitis unlikely.

Topical corticosteroids is incorrect. This is used in the management of anterior uveitis. Patients with inflammatory bowel disease, such as this patient, are at an increased risk of anterior uveitis. However, symptoms typically include an acute red and painful eye with patients often complaining of photophobia. These symptoms are not given in this presentation. Furthermore, this patient's eye demonstrates injection of the episcleral vessels which is uncharacteristic of anterior uveitis.

Discuss (1) | Improve

Next question

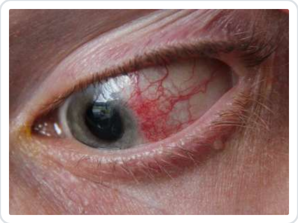
Episcleritis

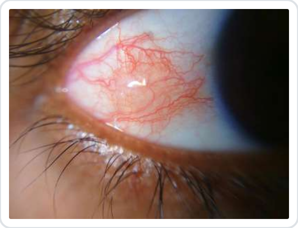
Episcleritis is describes the acute onset of inflammation in the episclera of one or both eyes.

The majority of cases are idiopathic, associated conditions include:
- inflammatory bowel disease
- rheumatoid arthritis

Features
- red eye
- classically not painful (in comparison to scleritis), but mild pain/irritation is common

- watering and mild photophobia may be present
- in episcleritis, the injected vessels are mobile when gentle pressure is applied on the sclera
    - in scleritis, vessels are deeper, hence do not move
- phenylephrine drops may be used to differentiate between episcleritis and scleritis
    - phenylephrine blanches the conjunctival and episcleral vessels but not the scleral vessels
    - if the eye redness improves after phenylephrine a diagnosis of episcleritis can be made
- approximately 50% of cases are bilateral





Management

- conservative
- artificial tears may sometimes be used

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **24.8%**

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101	✓

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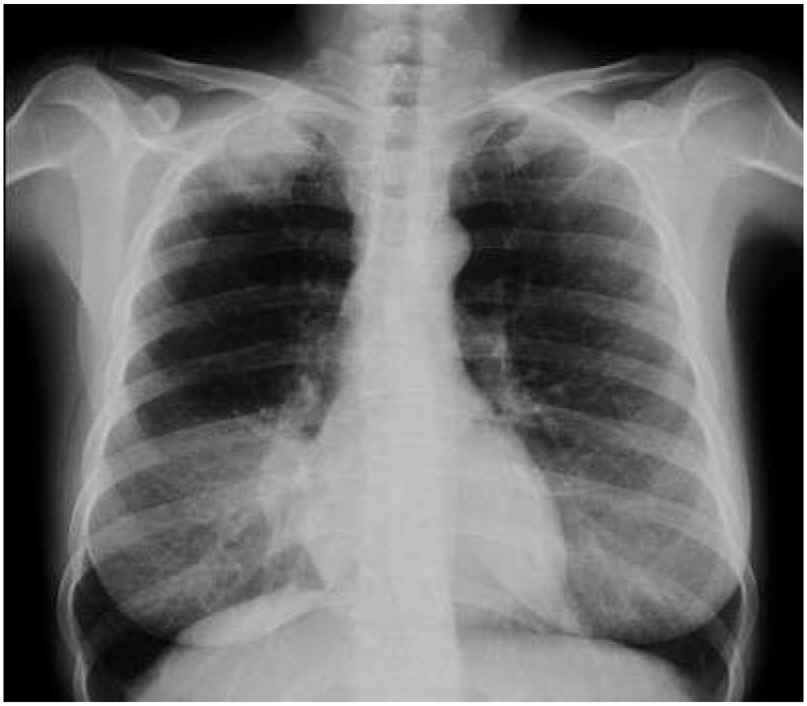
Back to top

A 56-year-old woman is referred to the respiratory clinic with a 3-month history of shortness of breath and a dry cough. She was admitted to the hospital for community-acquired pneumonia 1 year ago but had recovered well with complete resolution of symptoms. Her past medical history includes hypertension and hyperlipidaemia for which she takes amlodipine, ramipril and simvastatin. She drinks 4-6 units of alcohol per week and smokes 25 cigarettes/per day.

On examination, her observations are within normal limits. There are vesicular breath sounds bilaterally.

A chest x-ray is requested, as shown below:



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Given the diagnosis, what additional clinical finding is most likely to be present?

- ○ Erythema nodosum
- ○ Haematuria
- ○ Miosis
- ○ Pericardial friction rub

○ Purpura ×

Submit answer

Reference ranges ⌄

Score: **28%**

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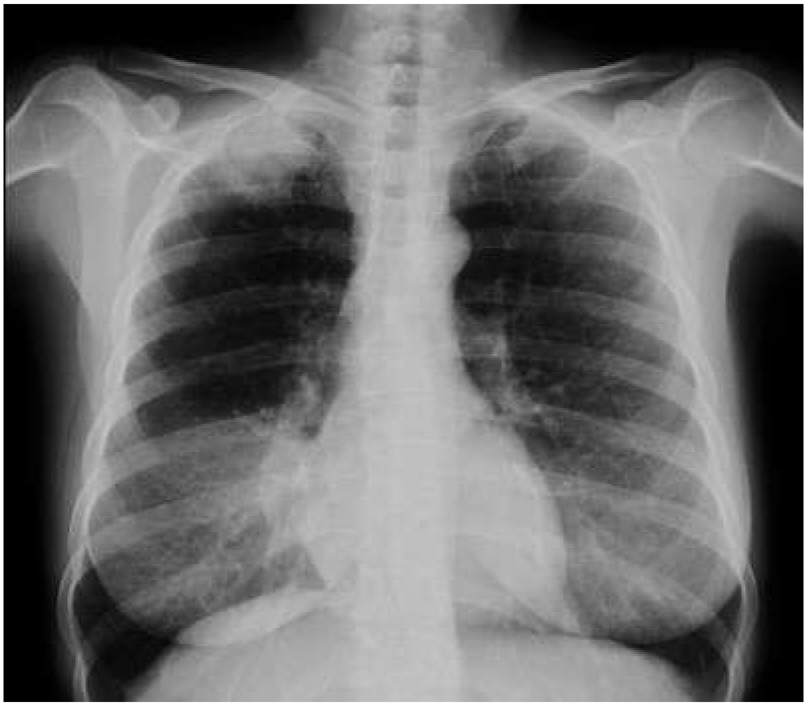


© Image used on license from Radiopaedia

Given the diagnosis, what additional clinical finding is most likely to be present?

Erythema nodosum	28%
Haematuria	3%
Miosis	64%
Pericardial friction rub	4%

Purpura 1%

This patient has a rounded opacity in the apical region of the right lung. It appears white and has a well-circumscribed outline. This is most likely in keeping with a Pancoast tumour. The history of progressive breathlessness, cough and smoking history makes malignancy a likely diagnosis. Given the location of the tumour, it may not always be detected on auscultation. A Pancoast tumour is usually a non-small cell lung carcinoma that presents with various symptoms, including malignancy (e.g. malaise, weight loss, night sweats) and compression from the tumour (e.g. thoracic outlet syndrome, recurrent laryngeal nerve compression and Horner's syndrome). Horner's syndrome results from compression of the sympathetic ganglion and the lack of sympathetic innervation to the face giving rise to the characteristic features of ptosis, **miosis** and anhidrosis.

Erythema nodosum is an extra-pulmonary manifestation of sarcoidosis. Sarcoidosis presents with shortness of breath, cough and fatigue. However, chest x-ray findings differ and range from being normal to having hilar lymphadenopathy and pulmonary infiltrates which are not seen here. Erythema nodosum is also an extra-pulmonary feature of tuberculosis. The history of prolonged dyspnoea and dry cough tuberculosis. However, the patient's significant smoking history and lack of risk factors for tuberculosis make malignancy more likely.

Haematuria is a clinical feature associated with vasculitic diseases (e.g. Goodpasture's syndrome and granulomatosis with polyangiitis). Both can cause pulmonary haemorrhage that presents with dyspnoea, haemoptysis, and chest x-ray findings of pulmonary infiltrates. This is not in keeping with the current presentation.

Pericardial friction rub is occasionally heard in patients with a pericardial effusion secondary to pericarditis. Trauma, viral and bacterial infections and autoimmune diseases (e.g. rheumatoid arthritis and systemic lupus erythematosus) are all causes of pericardial effusion. However, neither of these diagnoses accounts for the apical opacity.

Similarly to haematuria, **purpura** is a clinical feature of vasculitic diseases or a significant infection causing septicaemia. The progressive onset of symptoms and normal observations makes septicaemia unlikely.

👍 👎 Discuss (1) Improve

Next question ›

Horner's syndrome ★

Features

- miosis (small pupil)
- ptosis
- enophthalmos* (sunken eye)
- anhidrosis (loss of sweating one side)

Distinguishing between causes

- heterochromia (difference in iris colour) is seen in congenital Horner's
- anhidrosis: see below
- pharmacologic tests
    - can be useful to confirm the diagnosis of Horner syndrome and localise the lesion
    - apraclonidine drops (an alpha-adrenergic agonist) can be used: causes pupillary dilation in Horner's syndrome due to denervation supersensitivity but produces mild pupillary constriction in the normal pupil by down-regulating the norepinephrine release at the synaptic cleft

Central lesions	**Pre-ganglionic lesions**	**Post-ganglionic lesions**
Anhidrosis of the face, arm and trunk	Anhidrosis of the face	No anhidrosis
Stroke **S**yringomyelia Multiple **s**clerosis Tumour Encephalitis	Pancoast's **t**umour **T**hyroidectomy **T**rauma Cervical rib	**C**arotid artery dissection **C**arotid aneurysm **C**avernous sinus thrombosis **C**luster headache

*in reality the appearance is due to a narrow palpebral aperture rather than true enophthalmos

+ Q 123

Next question

Textbooks

High-yield textbook

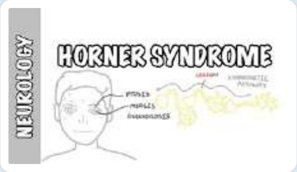
Extended textbook

Media

HORNER'S
SYNDROME

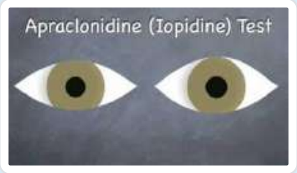
Horner's syndrome

Osmosis - YouTube 👍 12 👎 0


NEUROLOGY
HORNER SYNDROME

Horner's syndrome

Armando Hasudungan - YouTube 👍 4 👎 0


Apraclonidine (Iopidine) Test

How to Examine Horner's Syndrome

Individual - YouTube 👍 3 👎 0

Pancoast tumors

Osmosis - YouTube 👍 1 👎 0

Report broken media

Score: **24.8%**

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Question 63 of 189

A 27 year old lady with relapsing-remitting multiple sclerosis (MS) is referred to your clinic. She was diagnosed at age 18 and has been managed on interferon β 1a, with only four relapses in her total history. She has made good recovery from all four relapses and the only finding on neurological examination is of mild dysdiadochokinesis of the left arm. She scores functionally very well on an expanded disability status scale (EDSS) and has no disability as a result.

She is planning her first pregnancy and wanted advice on how her MS may be affected. She has already been advised on stopping interferon β 1a in preparation for pregnancy. Aside from the impact of stopping interferon β 1a, she wants to know how pregnancy itself will affect her MS. Which of the following is most accurate?

- ○ Relapse rates may decrease during pregnancy, increase at 3-6 months post-partum then return to pre-pregnancy rates.
- ○ There is an increased risk of developing visual disorders associated with MS.
- ○ There is a 30% chance that her RRMS will evolve into progressive MS.
- ○ There is a 15% chance that her relapse rate will decrease for the five years following pregnancy.
- ○ Relapse rates may increase during pregnancy, then return to pre-pregnancy rates at 9 months post-partum.

Submit answer

Reference ranges

Score: **28%**

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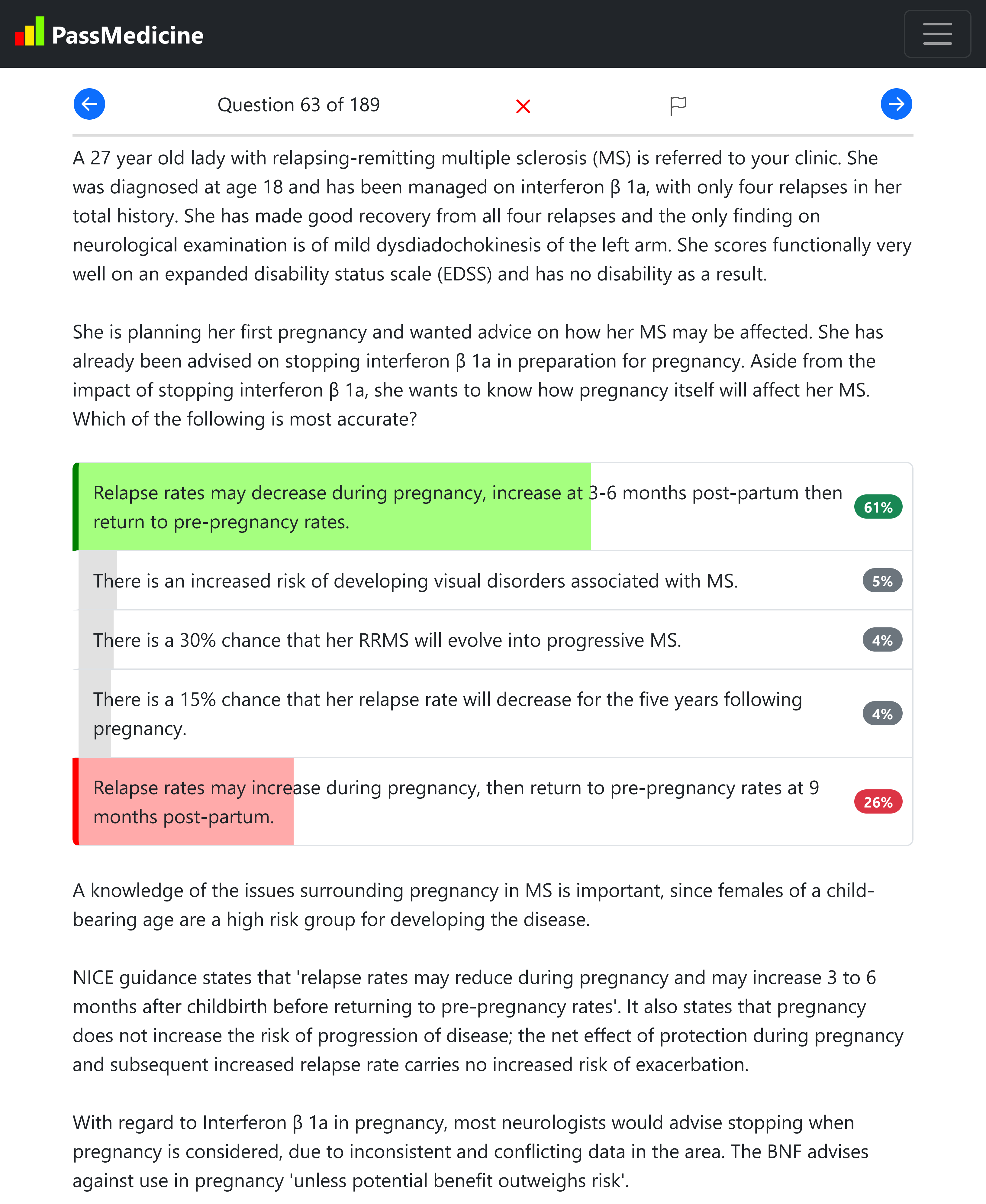


A 27 year old lady with relapsing-remitting multiple sclerosis (MS) is referred to your clinic. She was diagnosed at age 18 and has been managed on interferon β 1a, with only four relapses in her total history. She has made good recovery from all four relapses and the only finding on neurological examination is of mild dysdiadochokinesis of the left arm. She scores functionally very well on an expanded disability status scale (EDSS) and has no disability as a result.

She is planning her first pregnancy and wanted advice on how her MS may be affected. She has already been advised on stopping interferon β 1a in preparation for pregnancy. Aside from the impact of stopping interferon β 1a, she wants to know how pregnancy itself will affect her MS. Which of the following is most accurate?

Relapse rates may decrease during pregnancy, increase at 3-6 months post-partum then return to pre-pregnancy rates.	61%
There is an increased risk of developing visual disorders associated with MS.	5%
There is a 30% chance that her RRMS will evolve into progressive MS.	4%
There is a 15% chance that her relapse rate will decrease for the five years following pregnancy.	4%
Relapse rates may increase during pregnancy, then return to pre-pregnancy rates at 9 months post-partum.	26%

A knowledge of the issues surrounding pregnancy in MS is important, since females of a child-bearing age are a high risk group for developing the disease.

NICE guidance states that 'relapse rates may reduce during pregnancy and may increase 3 to 6 months after childbirth before returning to pre-pregnancy rates'. It also states that pregnancy does not increase the risk of progression of disease; the net effect of protection during pregnancy and subsequent increased relapse rate carries no increased risk of exacerbation.

With regard to Interferon β 1a in pregnancy, most neurologists would advise stopping when pregnancy is considered, due to inconsistent and conflicting data in the area. The BNF advises against use in pregnancy 'unless potential benefit outweighs risk'.

If a person with MS is thinking about pregnancy, they must have the opportunity to talk with a healthcare professional with knowledge of MS about:

- fertility
- the risk of the child developing MS
- use of vitamin D before conception and during pregnancy
- medication use in pregnancy
- pain relief during delivery (including epidurals)
- care of the child
- breastfeeding.

In this scenario the use of the expanded disability status scale (EDSS) was referred to. This provides an index of clinical disability and may be used to quantify disease progression. It has widespread use in clinical trials.

NICE guidance: multiple sclerosis
http://pathways.nice.org.uk/pathways/multiple-sclerosis/managing-multiple-sclerosis#content=view-node:nodes-provide-information-and-support

BNF: Interferon Beta.

Kurtzke JF. Rating neurologic impairment in multiple sclerosis: an expanded disability status scale (EDSS). Neurology 1983; 33:1444.

👍 👎 Discuss (2) Improve

Next question ›

Multiple sclerosis: management

Treatment in multiple sclerosis is focused on reducing the frequency and duration of relapses. There is no cure.

Acute relapse

High-dose steroids (e.g. oral or IV methylprednisolone) may be given for 5 days to shorten the length of an acute relapse. It should be noted that steroids shorten the duration of a relapse and do not alter the degree of recovery (i.e. whether a patient returns to baseline function)

Disease modifying drugs

A number of drugs have been shown to reduce the risk of relapse in patients with MS. Typical indications for disease-modifying drugs include:

- relapsing-remitting disease + 2 relapses in past 2 years + able to walk 100m unaided
- secondary progressive disease + 2 relapses in past 2 years + able to walk 10m (aided or unaided)

Drug options for reducing the risk of relapse in MS

- natalizumab
  - a recombinant monoclonal antibody that antagonises alpha-4 beta-1-integrin found on the surface of leucocytes
  - inhibit migration of leucocytes across the endothelium across the blood-brain barrier
  - generally considered to have the strongest evidence base for preventing relapse of the disease-modifying and hence is often used first-line
  - given intravenously
- ocrelizumab
  - humanized anti-CD20 monoclonal antibody
  - like natalizumab, it is considered a high-efficacy drug that is often used first-line
  - given intravenously
- fingolimod
  - sphingosine 1-phosphate (S1P) receptor modulator
  - prevents lymphocytes from leaving lymph nodes
  - oral formulations are available
- beta-interferon
  - not considered to be as effective as alternative disease-modifying drugs
  - given subcutaneously/intramuscularly
- glatiramer acetate
  - immunomodulating drug - acts as an 'immune decoy'
  - given subcutaneously
  - along with beta-interferon considered an 'older drug' with less effectiveness compared to monoclonal antibodies and S1P) receptor modulators

Some specific problems

Fatigue

- once other problems (e.g. anaemia, thyroid or depression) have been excluded NICE recommend a trial of amantadine
- other options include mindfulness training and CBT

Spasticity

- baclofen and gabapentin are first-line. Other options include diazepam, dantrolene and tizanidine
- physiotherapy is important
- cannabis and botox are undergoing evaluation

Bladder dysfunction

- may take the form of urgency, incontinence, overflow etc
- guidelines stress the importance of getting an ultrasound first to assess bladder emptying - anticholinergics may worsen symptoms in some patients
- if significant residual volume → intermittent self-catheterisation
- if no significant residual volume → anticholinergics may improve urinary frequency

Oscillopsia (visual fields appear to oscillate)

- gabapentin is first-line

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Multiple Sclerosis Society 6 15

Primary care guidelines

NICE 15 7

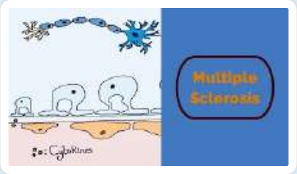
2014 Multiple Sclerosis guidelines

Suggest link   Report broken link

Media

Multiple sclerosis

Podmedics - YouTube 👍 4 👎 0


Multiple Sclerosis

Multiple sclerosis

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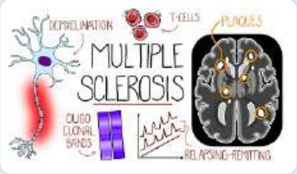
DEMYELINATION
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MULTIPLE SCLEROSIS
OLIGO CLONAL BANDS
RELAPSING-REMITTING

Multiple sclerosis

Rhesus Medicine - YouTube 👍 1 👎 0

MULTIPLE SCLEROSIS

Multiple sclerosis - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 7 👎 1

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Score: **24.8%**

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A 29-year-old man presents to the stroke ward following a right-sided middle cerebral artery territory infarct. You are reviewing him four weeks after as an outpatient. Whilst taking a history from him and his father you find out that he had a normal delivery and early development and initially performed well in school. However, from about the age of 12 he started developing focal impaired awareness seizures with secondary generalisation that were difficult to control with medications and started to struggle in school.

Since adulthood, he has suffered from frequent migraines that have again been difficult to treat with medications. He also suffers from recurrent vomiting although a thorough gastroenterological assessment including OGD with biopsies could not find any cause for the vomiting.

When aged 26 whilst out walking, he suddenly became very dizzy and unable to walk properly. These symptoms resolved in a day and he never sought medical attention. Since that incident, he has been left with a tremor that now makes his work as a painter almost impossible to do. He has also noticed that he is becoming more fatigued by walking. He goes to the gym regularly and is able to lift less and less weights as time has progressed. His mother died suddenly aged 37

Medications included: carbemazepine, clopidogrel and atorvastatin.

Examination

- Height 4ft 11inches (150 cm)
- Generalised muscle weakness with some atrophy
- Left upper limb power 3/5, right upper limb power 5/5
- Left lower limb power 5/5, right lower limb power 5/5
- Lateral nystagmus 5 beats
- Dysdiadochokinesia
- Moderate intention tremor
- MMSE 25/30

Hb	14.7 g/dl
Platelets	256 * 10^9/l
WBC	8.6 * 10^9/l

Na^+	140 mmol/l
K^+	4.5 mmol/l

Urea	4.6 mmol/l
Creatinine	54 µmol/l

Bilirubin	6 µmol/l
ALP	89 u/l
ALT	21 u/l
Total cholesterol	5.7 mmol/l
Albumin	40 g/l

CT Brain: right sided MCA territory infarct. Generalised atrophy

ABGs on air

pH	7.38
CO2	3.7 kPa
O2	15.6 kPa
Bicarb	49 mmol/l
Lactate	8.4 mmol/l

What is the most likely diagnosis?

- ○ Familial hypercholesterolaemia ×
- ○ MELAS syndrome ×
- ○ Carbemazepine poisoning ×
- ○ CADASIL ×
- ○ Pyruvate carboxylase deficiency ×

Submit answer

Reference ranges

Score: **28%**

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What is the most likely diagnosis?

Familial hypercholesterolaemia	1%
MELAS syndrome	68%
Carbemazepine poisoning	1%
CADASIL	28%
Pyruvate carboxylase deficiency	3%

The answer is B MELAS (Mitochondrial Encephalopathy with Lactic Acidosis and Stroke).

MELAS is a rare mitochondrially inherited condition characterised by short stature, recurrent migraines, vomiting, muscle weakness, seizures and then development of multiple strokes at an early age (normally before 40 for diagnosis to be considered) along with progressive dementia. There is also a raised lactic acid level in the blood and skeletal muscle biopsy will show ragged red fibres.

Regarding the vignette, it is possible that his mother had the same condition which is typical of mitochondrial inheritance. It also sounds like he may have had a posterior stroke that was not treated as he had sudden onset of cerebellar symptoms and is left with an intention tremor and has nystagmus. Cerebellar infarcts do not always show up on CT and MRI is the best modality for viewing the posterior fossa. Although is MMSE score is normal, for a young man a score of 25/30 is a potential worry in the clinical context.

Although CADASIL would also present with migraines and stroke at an early age, lactic acidosis is not a feature and subcortical infarct would be more typical. Although this gentleman has a high cholesterol it is not high enough for a diagnosis of familial hypercholesterolaemia and would not explain the muscle weakness, vomiting and lactic acidosis.

Carbamazepine poisoning could explain the cerebellar symptoms but not the other symptoms. Pyruvate carboxylase deficiency would cause muscle weakness, vomiting and lactic acidosis but would not explain the early onset of stroke

👍 👎 Discuss (3) Improve

Next question >

Mitochondrial diseases ★

Whilst most DNA is found in the cell nucleus, a small amount of double-stranded DNA is present in the mitochondria. It encodes protein components of the respiratory chain and some special types of RNA

Mitochondrial inheritance has the following characteristics:

- inheritance is only via the maternal line as the sperm contributes no cytoplasm to the zygote
- none of the children of an affected male will inherit the disease
- all of the children of an affected female will inherit the disease
- generally, encode rare neurological diseases
- poor genotype:phenotype correlation - within a tissue or cell there can be different mitochondrial populations - this is known as heteroplasmy

Histology

- muscle biopsy classically shows 'red, ragged fibres' due to increased number of mitochondria

Examples include:

- Leber's optic atrophy
    - symptoms typically develop at around the age of 30 years
    - central scotoma → loss of colour vision → rapid onset of significant visual impairment
- MELAS syndrome: mitochondrial encephalomyopathy lactic acidosis and stroke-like episodes
- MERRF syndrome: myoclonus epilepsy with ragged-red fibres
- Kearns-Sayre syndrome: onset in patients < 20 years old, external ophthalmoplegia, retinitis pigmentosa. Ptosis may be seen
- sensorineural hearing loss

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 5 👎 6

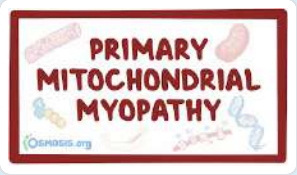
2013 How to spot mitochondrial disease in adults

Rare Disease Video Library - NORD 👍 3 👎 4

Primary Mitochondrial Myopathy

Suggest link Report broken link

Media


PRIMARY
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Osmosis.org

Primary mitochondrial myopathy

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Report broken media

Score: **24.8%**

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A 58 year old man presents to Accident and Emergency with his wife complaining of memory loss. His first noticed a problem when he had difficulty making breakfast in the morning. When you question him, he has no memory of the events of today and patchy memory of the events of the past week although his wife informs you that he had no problem with his memory yesterday. He appears extremely anxious about his memory loss and repeatedly asks you 'Have I got dementia?'. He is able to remember his name and date of birth and recognise and name his wife. However, when using the toilet, he becomes lost and is unable to find his way back to the cubicle.

On examination his HS 1 + 2 + 0, he has corneal arcus, chest is clear. There is reduced power 4/5 to all arm movements in the right arm with normal reflexes, tone and sensation. His wife tells you that this has been present for several years following a stroke. There is no facial droop, there is no dysarthric speech or any other peripheral focal neurology and there are no cerebellar signs. Gait is normal.

He is able to count back from 20-1 and can name objects you present to him without difficulty. However he scores 0/3 on the delayed recall section of the MMSE. He is alert and fully responsive throughout the consultation.
Past medical history is of previous left sided middle cerebral territory infarct and one previous TIA one year ago. Hypercholesteroalemia, hypertension. He has a 20 pack year history of smoking and used to drink 4-6 units of alcohol per day for 20 years but stopped this 25 years ago and his wife corroborates this information. His medications include Clopidogrel, Atorvastatin, Ramipril and Bendroflumethiazide.

CT Brain was performed 3 hours after waking up and is consistent with an old left sided infarct. No other abnormality detected.

What is the most appropriate treatment for this diagnosis?

○ Intravenous thiamine and Vitamin B

○ Oral thiamine and vitamin B

○ Thrombolysis

○ Donepezil

○ Supportive care only ×

Submit answer

Reference ranges ⌄

Score: **28%**

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CT Brain was performed 3 hours after waking up and is consistent with an old left sided infarct. No other abnormality detected.

What is the most appropriate treatment for this diagnosis?

Intravenous thiamine and Vitamin B	12%
Oral thiamine and vitamin B	6%
Thrombolysis	3%
Donepezil	21%

Supportive care only 58%

The answer is E. This man has a diagnosis of transient global amnesia. TGA is characterised by a sudden onset of global loss of recent memory and impaired new learning with no other cognitive defects. These patients are often very aware of their memory loss making them extremely anxious. Because of the dense anterograde amnesia, it is also characterised by repetitive questioning. This is sometimes called the broken record syndrome Loss of personal identity effectively rules out TGA Patients are alert and responsive during the episode

TGA is probably due to a transient malfunctioning of the limbo-hippocampal system which is involved in the formation of new memories and retrieval of recent memories. It normally presents in the 5th decade of life in those with pre-existing vascular risk factors, meaning there is probably a cerebro-vascular component but the cause is not known. TGA resolves spontaneously without need for treatment except for re-assurance and subsequent control of vascular risk factors.

Options A and B are inappropriate because the diagnosis is not Wernicke-Korsakoff syndrome. The presentation is too acute and too long after his heavy drinking of alcohol to be a likely diagnosis. Although there is similar defects in memory, there are normally defects in attention, concentration, frontal lobe functioning and is accompanied by symptoms such as apathy and withdrawal.

Option C is inappropriate as thrombolysis is not a treatment for TGA. In addition, thrombolysis would not usually be given to someone who has woken up with symptoms suggestive of a a stroke, even if the time window is less than 4.5 hours.

Option D is inappropriate as the diagnosis is not dementia. The onset of symptoms are too acute for this diagnosis to be considered.

👍 👎 Discuss (6) Improve

Next question ›

Transient global amnesia ★

Transient global amnesia is characterized by the acute onset of anterograde amnesia (the inability to form new memories). The aetiology is unknown, thought to be due to transient ischaemia to the thalamus (in particular the amygdala and hippocampus).

Features

- patients may appear anxious and repeatedly ask the same question

- episodes are self-limited and resolve within 24 hours

+ Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Score: **24.8%**

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101	✓

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Question 66 of 189

You review an 80-year old retired teacher in your clinic in your clinic. She has been complaining of numbness on her arms and difficulty walking. On examination you find she has reduced sensation lateral aspect of her arms and forearms in the anatomical position. She has no wasting on the muscles of her hands. She has reduced biceps and supinator reflexes and reduced power bilaterally.

When she gets up to go you notice she has a scissoring gait which is wide based. You ask her to close her eyes and she is unable to stand still without support.

She lives on her own and usually is independent, although recently she has been struggling to cope. She has a background of hypertension and high cholesterol.

What is the most appropriate investigation to diagnose this lady?

- ○ Cervical myelography
- ○ Cervical X-Ray
- ○ Nerve conduction studies
- ○ Nerve biopsy
- ○ MRI of the cervical cord

Submit answer

Reference ranges

Score: **28%**

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2 ✗

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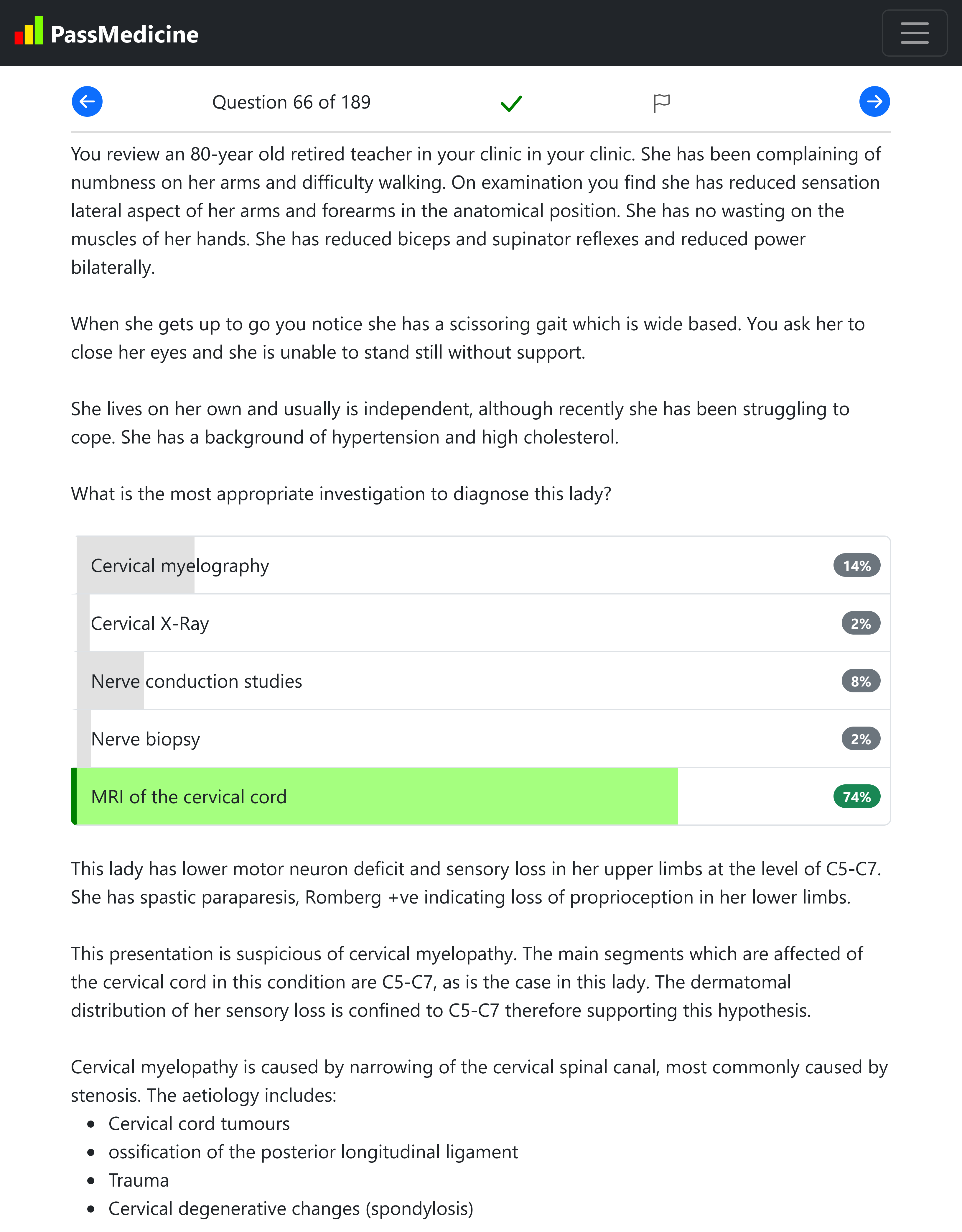

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She lives on her own and usually is independent, although recently she has been struggling to cope. She has a background of hypertension and high cholesterol.

What is the most appropriate investigation to diagnose this lady?

Cervical myelography	14%
Cervical X-Ray	2%
Nerve conduction studies	8%
Nerve biopsy	2%
MRI of the cervical cord	74%

This lady has lower motor neuron deficit and sensory loss in her upper limbs at the level of C5-C7. She has spastic paraparesis, Romberg +ve indicating loss of proprioception in her lower limbs.

This presentation is suspicious of cervical myelopathy. The main segments which are affected of the cervical cord in this condition are C5-C7, as is the case in this lady. The dermatomal distribution of her sensory loss is confined to C5-C7 therefore supporting this hypothesis.

Cervical myelopathy is caused by narrowing of the cervical spinal canal, most commonly caused by stenosis. The aetiology includes:

- Cervical cord tumours
- ossification of the posterior longitudinal ligament
- Trauma
- Cervical degenerative changes (spondylosis)

- Cervical degenerative disc disease

The investigation of choice and one which would enable you to asses spinal and foraminal stenosis would be cervical myelography or MRI of the cervical cord. Cervical myelography entails the use of contrast and can be nephrotoxic alas MRI is the preferred modality to image and diagnose cervical spondylosis.

👍 👎 Discuss (10) Improve

Next question >

Degenerative cervical myelopathy ★

Degenerative cervical myelopathy (DCM) has a number of risk factors, which include smoking due to its effects on the intervertebral discs, genetics and occupation - those exposing patients to high axial loading [1].

The presentation of DCM is very variable. Early symptoms are often subtle and can vary in severity day to day, making the disease difficult to detect initially. However as a progressive condition, worsening, deteriorating or new symptoms should be a warning sign.

DCM symptoms can include any combination of [1]:
- Pain (affecting the neck, upper or lower limbs)
- Loss of motor function (loss of digital dexterity, preventing simple tasks such as holding a fork or doing up their shirt buttons, arm or leg weakness/stiffness leading to impaired gait and imbalance
- Loss of sensory function causing numbness
- Loss of autonomic function (urinary or faecal incontinence and/or impotence) - these can occur and do not necessarily suggest cauda equina syndrome in the absence of other hallmarks of that condition
- Hoffman's sign: is a reflex test to assess for cervical myelopathy. It is performed by gently flicking one finger on a patient's hand. A positive test results in reflex twitching of the other fingers on the same hand in response to the flick.

The most common symptoms at presentation of DCM are unknown, but in one series 50% of patients were initially incorrectly diagnosed and sometimes treated for carpal tunnel syndrome [2].

An MRI of the cervical spine is the gold standard test where cervical myelopathy is suspected. It may reveal disc degeneration and ligament hypertrophy, with accompanying cord signal change.

All patients with degenerative cervical myelopathy should be urgently referred for assessment by specialist spinal services (neurosurgery or orthopaedic spinal surgery). This is due to the importance of early treatment. The timing of surgery is important, as any existing spinal cord damage can be permanent. Early treatment (within 6 months of diagnosis) offers the best chance of a full recovery but at present, most patients are presenting too late. In one study, patients averaged over 5 appointments before diagnosis, representing >2 years.

Currently, decompressive surgery is the only effective treatment. It has been shown to prevent disease progression. Close observation is an option for mild stable disease, but anything progressive or more severe requires surgery to prevent further deterioration. Physiotherapy should only be initiated by specialist services, as manipulation can cause more spinal cord damage.

References
1. Baron EM, Young WF. Cervical spondylotic myelopathy: a brief review of its pathophysiology, clinical course, and diagnosis. Neurosurgery. 2007 Jan;60(1 Supp1 1):S35-41.
2. Behrbalk E, Salame K, Regev GJ, Keynan O, Boszczyk B, Lidar Z. Delayed diagnosis of cervical spondylotic myelopathy by primary care physicians. Neurosurg Focus. 2013 Jul;35(1):E1.

Next question

Textbooks

High-yield textbook

Extended textbook

Media

MS WITH A POSITIVE HOFFMAN'S SIGN &BABINSKI TEST

MS with positive Hoffman's sign and Babinski test

Individual - YouTube 👍 0 👎 0

Hoffmann's
Sign
Assessment

Hoffmann's Sign or Reflex - Upper Motor Neuron Lesion

Physiotutors - YouTube 👍 0 👎 0

Report broken media

Score: **24.8%**

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Question 67 of 189

A 57 year-old smoker presents to you with difficulty walking. He used to be able to mobilise unaided but recently he has required the assistance of his wife. He reports being more clumsy than usual. He drinks 3 cans of lager every month. He takes simvastatin for high cholesterol and has been taking aspirin for 10 years since his GP started it. He has lost a lot of weight in recent months, which he can't quantify.

On examination, you find that he is cachectic. He has an intention tremor and exhibits dysdiadochokinesia. His gait is broad-based. He has bilateral nystagmus on horizontal gaze. On auscultating his chest you find he has a degree of bronchial breathing on the right middle lobe. His observations are unremarkable.

His blood tests are as follows:

Hb	100 g/l
MCV	79.9 fL
Platelets	239 * 10^9/l
WBC	6.6 * 10^9/l
Na^+	134 mmol/l
K^+	4.3 mmol/l
Urea	6 mmol/l
Creatinine	88 µmol/l
Anti-Hu antibody	POSITIVE
Bilirubin	6 µmol/l
ALP	62 u/l
Albumin	29 g/l

CT head: No acute intracranial pathology.

What is the most appropriate investigation?

○ Chest X-ray ×

○ Vitamin B12 levels ×

○ ECG ×

○ Visual evoked potentials ×

○ Muscle biopsy ×

Submit answer

Reference ranges ⌵

Score: **28%**

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Creatinine	88 µmol/l
Anti-Hu antibody	POSITIVE
Bilirubin	6 µmol/l
ALP	62 u/l
Albumin	29 g/l

CT head: No acute intracranial pathology.

What is the most appropriate investigation?

Chest X-ray 75%

Vitamin B12 levels 4%

ECG	1%
Visual evoked potentials	8%
Muscle biopsy	12%

This gentleman presents with signs and symptoms of the cerebellar syndrome.

He is cachectic, has a history of smoking and some signs of his chest which would raise concern given his history. Another vital clue in the question is the presence of anti-hu antibodies, which are associated with paraneoplastic cerebellar syndrome secondary to small cell lung cancer. Given the history and examination findings it is paramount to perform an X-Ray of his chest, and should that be inconclusive he will require a high-resolution CT scan.

An intracranial lesion, such as a tumour or a vascular event is also unlikely, given the normal CT head result and bilateral presentation.

Vitamin B12 deficiency is associated with subacute combined degeneration of the cord. This would present with upper and motor neuron deficits and a sensory neuropathy. It would not present with isolated cerebellar signs.

👍 👎 Discuss (4) Improve

Next question >

Paraneoplastic neurological syndromes ★

Lambert-Eaton myasthenic syndrome

- seen in small cell lung cancer
- antibody directed against voltage gated calcium channel
- similar features to myasthenia gravis
- proximal muscle weakness, hyporeflexia and autonomic features (dry mouth, impotence etc)

Other syndromes may be associated with detectable antibodies, for example:

- anti-Hu: small cell lung cancer - painful sensory neuropathy, cerebellar syndromes, encephalitis
- anti-Yo: ovarian, breast cancer - cerebellar syndrome
- anti-Ri: small cell lung cancer - retinal degeneration

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Mayo Clinic 👍 0 👎 0

Paraneoplastic Syndromes: An Approach to Diagnosis and Treatment

Suggest link

Report broken link

Score: **24.8%**

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Question 68 of 189

A 40 year-old man presents to the general medical clinic with a one year history of generalised weakness. He mentions his father suffered from the same symptoms, although from a later age.

His past medical history includes type two diabetes mellitus (diet controlled) and eczema.

On examination he has male pattern balding and an expressionless face. You note a diminished left red reflex, with a normal cranial nerve examination. He has symmetrically reduced reflexes throughout, with mild reduced power distally in both upper and lower limbs.

Hb	140 g/l
Platelets	200 * 10^9/l
WBC	8 * 10^9/l
CK	70IU/L (reference range 60- 174 IU/L)

What is the most likely diagnosis?

○ Hyperthyroidism

○ Inclusion body myositis

○ Cushing's disease

○ Becker's muscular dystrophy

○ Myotonic dystrophy

Submit answer

Reference ranges

Score: **28%**

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Hb	140 g/l
Platelets	200 * 10^9/l
WBC	8 * 10^9/l
CK	70IU/L (reference range 60- 174 IU/L)

What is the most likely diagnosis?

Hyperthyroidism	1%
Inclusion body myositis	15%
Cushing's disease	2%
Becker's muscular dystrophy	12%
Myotonic dystrophy	70%

Myotonic dystrophy is a genetic condition causing by a trinucleotide repeat expansion on chromosome 19. It demonstrates anticipation (earlier onset with successive generations).

Clinical features of the condition include:

- Frontotemporal balding
- Ptosis
- Distal weakness
- Depressed tendon reflexes
- Myotonic (slow relaxation of muscles)

Complications include arrhythmias, cataracts, testicular atrophy and diabetes mellitus.

Cushing's disease and hyperthyroidism cause a proximal myopathy, and the patient has no symptoms of either.

Becker's muscular dystrophy causes a proximal myopathy, but the average age of presentation and diagnosis is much younger (average is 11 years old), with several disability at 40.

Inclusion body myositis can cause both a proximal and distal myopathy. This patient's creatinine kinase is normal, which makes the diagnosis less likely and would not explain the genetic component and facial features.

👍 👎 💬 Discuss (5) Improve

Next question ›

Myotonic dystrophy ★

Myotonic dystrophy (also called dystrophia myotonica) is an inherited myopathy with features developing at around 20-30 years old. It affects skeletal, cardiac and smooth muscle. There are two main types of myotonic dystrophy, DM1 and DM2.

Genetics
- autosomal dominant
- a trinucleotide repeat disorder
- DM1 is caused by a CTG repeat at the end of the DMPK (Dystrophia Myotonica-Protein Kinase) gene on chromosome 19
- DM2 is caused by a repeat expansion of the ZNF9 gene on chromosome 3

The key differences are listed in table below:

DM1	**DM2**
- DMPK gene on chromosome 19 - Distal weakness more prominent	- ZNF9 gene on chromosome 3 - Proximal weakness more prominent - Severe congenital form not seen

General features

- myotonic facies (long, 'haggard' appearance)
- frontal balding
- bilateral ptosis
- cataracts
- dysarthria

Other features
- myotonia (tonic spasm of muscle)
- weakness of arms and legs (distal initially)
- mild mental impairment
- diabetes mellitus
- testicular atrophy
- cardiac involvement: heart block, cardiomyopathy
- dysphagia

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

Myotonic dystrophy

Zero To Finals - YouTube

👍 4 👎 0

Report broken media

Score: **24.8%**

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Question 69 of 189

A 73 year old gentleman is admitted to the medical assessment unit with a fall. A collateral history from his wife reveals that he has been having frequent falls (nearly 2-3 times a week) for the last 4 months. This has resulted in multiple admissions where a chest infection is thought to be the main cause for his deterioration. His wife also states he is having problems swallowing, chokes on his food frequently and has not been himself recently with frequent bouts of aggression. On examination you note the patient has an expressionless face, and kyphosed posture and mild tremor bilaterally. He has cogwheeling of both upper limbs. Examination of his legs is unremarkable. On inspection of his gait, it appears parkinsonian in character, but you stop him only after a few steps as he seems unsteady. On cranial nerve examination you note he has difficulty following your finger downwards but the remainder of the cranial nerve examination is normal. Given these features, what is the most likely underlying diagnosis?

○ Multiple system atrophy ×

○ Idiopathic Parkinsons disease ×

○ Progressive supranuclear palsy ×

○ Vascular Parkinsonism ×

○ Lewy body dementia ×

Submit answer

Reference ranges

Score: **28%**

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Multiple system atrophy	15%
Idiopathic Parkinsons disease	5%
Progressive supranuclear palsy	75%
Vascular Parkinsonism	2%
Lewy body dementia	3%

There is no doubt that this patient has an element of Parkinsons disease, the clues to the underlying condition include:

- The SYMMETRICAL tremor compared with the ASYMMETRICAL tremor of idiopathic Parkinsons.
- Swallowing difficulties suggesting repeated admissions for aspiration pneumonia.
- Changes in personality.
- Truncal instability and rigidity > limbs.
- Inability of look downwards (Vertical gaze palsy).

Parkinsons Plus syndromes:

- Progressive supranuclear palsy (PSP, also known as Steele-Richardson-Olszewski syndrome) - Characterised by early postural instability, frequent falls, truncal rigidity, asymmetrical onset, speech and swallowing problems, vertical gaze palsy.
- Multiple system atrophy - (MSA, also known as Shy-Drager syndrome). Autonomic features specifically postural blood pressure drop, bladder/bowel dysfunction, cerebellar and/or

pyramidal signs.
- Lewy body dementia - Visual hallucinations accompanying dementia.
- Vascular parkinsons - Parkinsonism worse in the legs than the arms.
- Cortico-basal degeneration (CBD) - Asterognosis (inability to recognise objects by touch), akinetic rigidity involving one limb. Some patients may experience the 'alien limb phenomenon'.

NB: Knowledge of this differential is also vital for PACES examinations.

Discuss (4) | Improve

Next question ›

Progressive supranuclear palsy

Overview
- aka Steele-Richardson-Olszewski syndrome
- a 'Parkinson Plus' syndrome

Features
- postural instability and falls
  - patients tend to have a stiff, broad-based gait
- impairment of vertical gaze (down gaze worse than up gaze - patients may complain of difficultly reading or descending stairs)
- parkinsonism
  - bradykinesia is prominent
- cognitive impairment
  - primarily frontal lobe dysfunction

Management
- poor response to L-dopa

AI + Q 123

Next question ›

B I A

Textbooks

High-yield textbook

Extended textbook

Score: **24.8%**

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A 32 year old man presented to the Emergency Department with a severe headache. The headache had started suddenly that evening while the patient was at home with his girlfriend watching television. The patient described his headache as an intense throbbing pain felt all across his head. The pain had started very quickly with the patient reporting a severity of 10 / 10 within 30 seconds. The pain had eased slightly following some morphine given by the ambulance crew. The patient denied any symptoms of neck stiffness or photophobia. There were no visual symptoms and no weakness in the patient's limbs or abnormal sensation.

The patient had no previous past medical history and did not normally suffer from headaches. He took no regular medications. The patient worked as a retail assistant and lived with his girlfriend. He rarely drank alcohol but did smoke cannabis on several occasions each month. The patient's girlfriend reported that the patient had smoked cannabis that evening prior to suffering his headache.

On examination, the patient was in visible discomfort due to his headache although had no meningitic signs. Glasgow Coma Score was 15/15 and the patient was afebrile. Cranial and peripheral nerve examination was unremarkable.

Further analgesia was given to the patient and investigations requested as detailed below.

CT brain: no evidence extra-dural or sub-dural bleeding; no intracerebral haemorrhage or sub-arachnoid blood; no evidence of cerebral infarction; normal ventricular system; no radiological contra-indication to lumbar puncture

Cerebrospinal fluid results:

Red cell count	1 / mm^3
White cell count	2 / mm^3
Pprotein	0.75 g / L
Glucose	60 % of serum value
Microscopy	No organisms seen
Red cell breakdown products	Negative
Opening pressure	19 cmCSF

Following initial investigation as above, the patient continued to experience severe recurrent headaches similar to that experienced at presentation and was observed to have a short, generalised seizure on the admission ward prompting further investigation.

CT angiography: diffuse arterial beading identified; results otherwise as for previous CT head

What is the cause for the patient's headache?

- ○ Atypical migraine ×
- ○ Cervical artery dissection ×
- ○ Reversible cerebrovascular vasoconstriction syndrome ×
- ○ Posterior reversible leucoencephalopathy syndrome ×
- ○ Pituitary apoplexy ×

Submit answer

Reference ranges ∨

Score: **28%**

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A 32 year old man presented to the Emergency Department with a severe headache. The headache had started suddenly that evening while the patient was at home with his girlfriend watching television. The patient described his headache as an intense throbbing pain felt all across his head. The pain had started very quickly with the patient reporting a severity of 10 / 10 within 30 seconds. The pain had eased slightly following some morphine given by the ambulance crew. The patient denied any symptoms of neck stiffness or photophobia. There were no visual symptoms and no weakness in the patient's limbs or abnormal sensation.

The patient had no previous past medical history and did not normally suffer from headaches. He took no regular medications. The patient worked as a retail assistant and lived with his girlfriend. He rarely drank alcohol but did smoke cannabis on several occasions each month. The patient's girlfriend reported that the patient had smoked cannabis that evening prior to suffering his headache.

On examination, the patient was in visible discomfort due to his headache although had no meningitic signs. Glasgow Coma Score was 15/15 and the patient was afebrile. Cranial and peripheral nerve examination was unremarkable.

Further analgesia was given to the patient and investigations requested as detailed below.

CT brain: no evidence extra-dural or sub-dural bleeding; no intracerebral haemorrhage or sub-arachnoid blood; no evidence of cerebral infarction; normal ventricular system; no radiological contra-indication to lumbar puncture

Cerebrospinal fluid results:

Red cell count	1 / mm³
White cell count	2 / mm³
Pprotein	0.75 g / L
Glucose	60 % of serum value
Microscopy	No organisms seen
Red cell breakdown products	Negative
Opening pressure	19 cmCSF

Following initial investigation as above, the patient continued to experience severe recurrent headaches similar to that experienced at presentation and was observed to have a short, generalised seizure on the admission ward prompting further investigation.

CT angiography: diffuse arterial beading identified; results otherwise as for previous CT head

What is the cause for the patient's headache?

Atypical migraine	3%
Cervical artery dissection	7%
Reversible cerebrovascular vascoconstriction syndrome	75%
Posterior reversible leucoencephalopathy syndrome	12%
Pituitary apoplexy	2%

The patient presents with a typical 'thunderclap' headache, with pain reaching a severe intensity within one minute of onset. Reversible cerebrovascular vasoconstriction syndrome (RCVS) is a cause of thunderclap headache with normal CT brain and normal or near-normal lumbar puncture results. RCVS may also be associated with focal neurological signs and seizures. Diagnosis is made by the finding of diffuse arterial beading on angiography (CT, MRI or catheter). One-half of cases occur during the postpartum period or after exposure to serotoninergic agents, adrenergic substances or cannabis. There are no evidence-based treatments for RCVS although bed-rest and nimodipine are usually prescribed. RCVS is not always benign, with one-in-four patients suffering sub-arachnoid haemorrhage or ischaemic / haemorrhagic stroke

The other answers listed in the stem are all causes of thunderclap headache with normal CT brain and lumbar puncture (i.e. sub-arachnoid haemorrhage excluded). None of these conditions area associated with diffuse arterial beading and so are less likely differential than RCVS.

Please note that the report states **beading**, not bleeding. For more information see: https://radiopaedia.org/articles/intracranial-arterial-beading

Ducros A, Bousser M. Thunderclap headache. BMJ 2012;345:e8557.

Discuss (10) | Improve

Next question ›

Thunderclap headache ★

Thunderclap headache describes a sudden (reaches maximum severity within seconds to minutes of onset) and severe headache.

Causes

- subarachnoid hemorrhage
- cerebral venous sinus thrombosis
- internal carotid artery dissection
- pituitary apoplexy
- reversible cerebral vasoconstriction syndrome
- primary sexual headache
- posterior reversible leucoencephalopathy syndrome
- acute hypertensive crisis

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **24.8%**

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Question 71 of 189

A 68-year-old man is admitted with weakness. He states that it came on at 09:45 (2 hours and 15 minutes ago). He states that his right arm and leg are weak. He also complains of word finding difficulties. He has a past medical history of hypertension and hypercholesterolaemia. On examination he has grade 4 weakness of his right arm and leg, and there is an obvious right sided facial droop. He has a right homonymous hemianopia. He can follow complex 3 stage commands, however he struggles to name simple objects.

You suspect an acute stroke and are considering suitability for thrombolysis. You perform blood tests and arrange an urgent CT scan.

What scoring system will you utilise to further assess this patient?

- ○ CHA,,DS,,-VASc
- ○ ABCD2
- ○ HAS-BLED
- ○ Total Health Risk In Vascular Events (THRIVE)
- ○ National Institutes of Health Stroke Scale (NIHSS)

Submit answer

Reference ranges

Score: **28%**

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You suspect an acute stroke and are considering suitability for thrombolysis. You perform blood tests and arrange an urgent CT scan.

What scoring system will you utilise to further assess this patient?

CHA,,DS,,-VASc	2%
ABCD2	4%
HAS-BLED	7%
Total Health Risk In Vascular Events (THRIVE)	2%
National Institutes of Health Stroke Scale (NIHSS)	85%

The NIHSS score is useful in stroke for assessing patients suitability for thrombolysis

Important for me Less important

The most immediate concern is to assess the suitability for thrombolysis. Thrombolysis can result in extremely good outcomes for patients, however it is also has significant risks and it is therefore essential to balance benefits versus potential risks. The National Institutes of Health Stroke Scale (NIHSS) is an extremely useful tool for assessing the severity of the stroke. Some hospitals use an NIHSS of less than 5 (mild stroke) to exclude patients from thrombolysis. The patient in this case is likely suffering from a total anterior circulation stroke due to the presence of hemiparesis, homonymous hemianopia and higher function involvement (e.g. expressive dysphasia). He will therefore likely have a high NIHSS score.

The CHA,,DS,,-VASc score is used to predict the risk of stroke in patients with atrial fibrillation.

The ABCD2 score is used in transient ischaemic attacks to predict the likelihood of a stroke in the short term period following the TIA. The ABCD2 score ranges from 0 to 7. Recent studies have shown that the ABCD3-I scoring system is superior in the assessment of TIAs. The ABCD3-I incorporates recurrent TIA events and imaging. The minimum score is 0 and the maximum is 13. The broader range of scores allows for potentially greater refinement of risk prediction.

The HAS-BLED score is used to predict the risk of bleeding from anticoagulation in atrial fibrillation.

The Total Health Risk In Vascular Events (THRIVE) score uses NIHSS score, age, and chronic disease to predict long-term neurologic outcomes in stroke patients

👍 👎 Discuss (1) Improve

Next question >

Stroke: management of acute ischaemic stroke ★

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
    - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
    - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:
- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.
- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
    - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
    - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	**Relative**
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians Thrombolysis and thrombectomy for acute ischaemic stroke	👍 11	👎 15
Clinical Knowledge Summaries Stroke and TIA guidelines	👍 16	👎 15
Age and Ageing Interesting article on managing blood pressure during acute stroke	👍 20	👎 16

National Clinical Guideline for Stroke 👍 5 👎 3

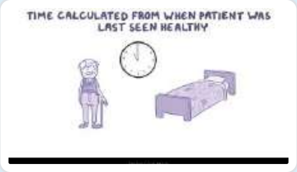
2023 National Clinical Guideline for Stroke

NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia
Acute Ischaemic Stroke

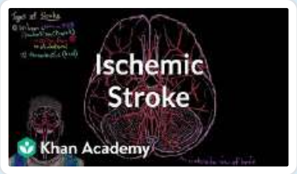
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

Khan Academy - YouTube

👍 1 👎 1

Report broken media

Score: **24.8%**

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A 54-year-old woman is seen in the neurology clinic. She initially presented to her general practitioner with paraesthesia and a burning sensation down her right side. She reports a similar episode two years ago, during which she had a four-week period of paraesthesia in the left lower limb which self resolved. In between these episodes, she experienced total resolution of her symptoms.

An MRI of the brain and spinal cord was performed and concluded as below:

MRI brain and spinal cord	Several areas of T1 weighted hypointensities and T2 weighted hyperintensities consistent with multiple sclerosis

Which of the features in this woman represents a poor prognostic factor?

○ Female sex

○ Long interval between relapses

○ Older age of onset

○ Relapsing-remitting disease

○ Sensory symptoms only

Submit answer

Reference ranges

Score: **28%**

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A 54-year-old woman is seen in the neurology clinic. She initially presented to her general practitioner with paraesthesia and a burning sensation down her right side. She reports a similar episode two years ago, during which she had a four-week period of paraesthesia in the left lower limb which self resolved. In between these episodes, she experienced total resolution of her symptoms.

An MRI of the brain and spinal cord was performed and concluded as below:

MRI brain and spinal cord	Several areas of T1 weighted hypointensities and T2 weighted hyperintensities consistent with multiple sclerosis

Which of the features in this woman represents a poor prognostic factor?

Female sex	7%
Long interval between relapses	2%
Older age of onset	81%
Relapsing-remitting disease	7%
Sensory symptoms only	3%

Older age of onset is associated with a poor prognosis in multiple sclerosis

Important for me Less important

As stated in the question, this woman has been diagnosed with multiple sclerosis (MS). Of the features listed, only older age is associated with a poor prognosis. The onset of symptoms in patients, who are in their 20s or 30s, is associated with a good prognosis.

MS is less common in males; however, when diagnosed, the prognosis is worse. Female sex is therefore associated with a better prognosis and is, therefore, an incorrect answer here.

This woman has had two years separating her two attacks which is a good prognostic indicator rather than a poor one. A long interval between relapses is, therefore, an incorrect answer.

This woman exhibits a relapsing-remitting pattern of disease with distinct episodes of symptoms with improvement in between. A relapsing-remitting disease has a significantly better prognosis than a progressive disease course (continuous symptoms with gradual worsening), and therefore this is not the correct answer.

Motor symptoms are associated with a poorer prognosis. This woman has only experienced sensory symptoms, which is a good prognostic factor in this case.

Discuss (2) | Improve

Next question

Multiple sclerosis: prognostic features

Good prognosis features
- female sex
- age: young age of onset (i.e. 20s or 30s)
- relapsing-remitting disease
- sensory symptoms only
- long interval between first two relapses
- complete recovery between relapses

Ways of remembering prognostic features
- the typical patient carries a better prognosis than an atypical presentation

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **24.8%**

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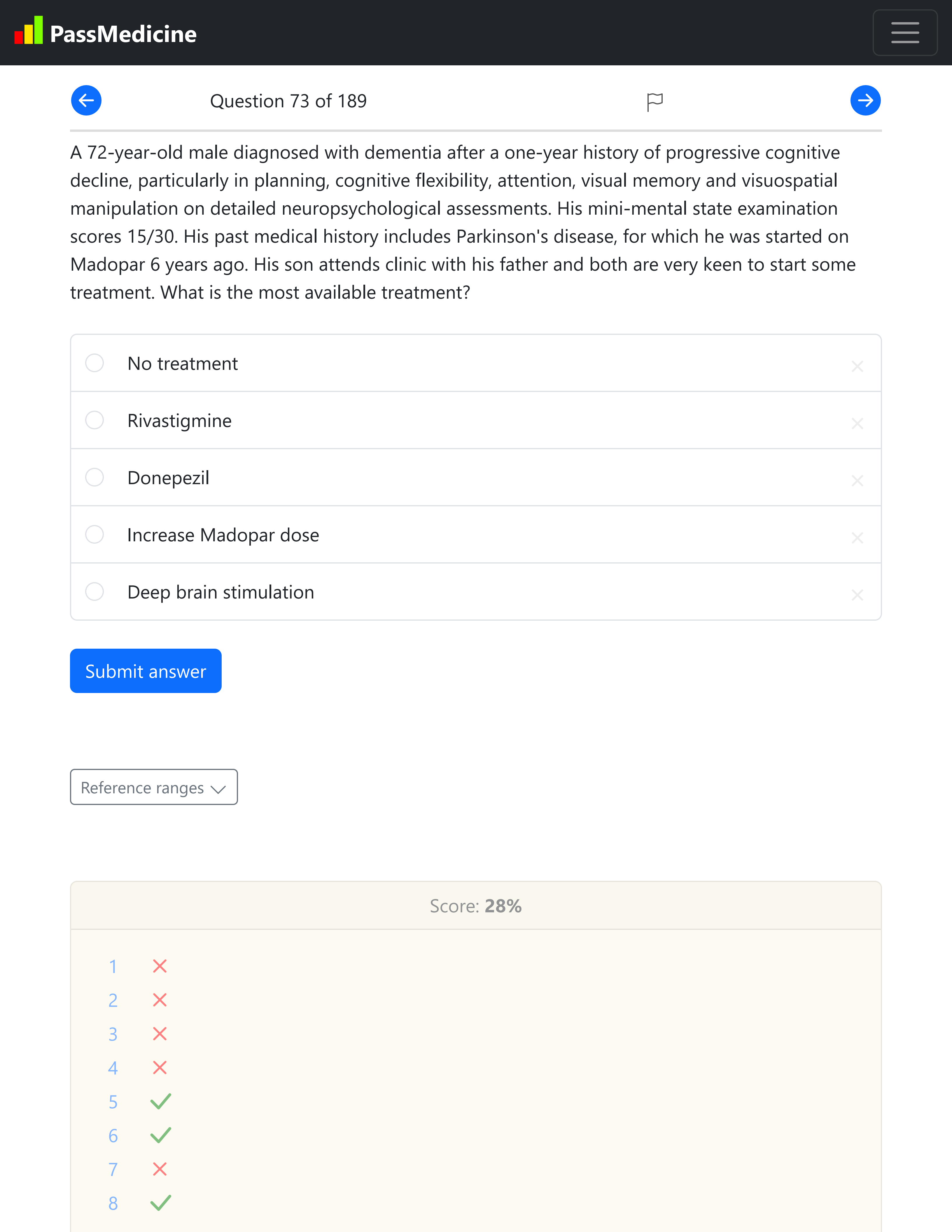

Question 73 of 189

A 72-year-old male diagnosed with dementia after a one-year history of progressive cognitive decline, particularly in planning, cognitive flexibility, attention, visual memory and visuospatial manipulation on detailed neuropsychological assessments. His mini-mental state examination scores 15/30. His past medical history includes Parkinson's disease, for which he was started on Madopar 6 years ago. His son attends clinic with his father and both are very keen to start some treatment. What is the most available treatment?

- ○ No treatment
- ○ Rivastigmine
- ○ Donepezil
- ○ Increase Madopar dose
- ○ Deep brain stimulation

Score: **28%**

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A 72-year-old male diagnosed with dementia after a one-year history of progressive cognitive decline, particularly in planning, cognitive flexibility, attention, visual memory and visuospatial manipulation on detailed neuropsychological assessments. His mini-mental state examination scores 15/30. His past medical history includes Parkinson's disease, for which he was started on Madopar 6 years ago. His son attends clinic with his father and both are very keen to start some treatment. What is the most available treatment?

No treatment	9%
Rivastigmine	34%
Donepezil	48%
Increase Madopar dose	5%
Deep brain stimulation	4%

The patient describes a syndrome of executive and visuospatial dysfunction on a background of PD diagnosis more than 12 months before diagnosis of cognitive impairment. This represents PD dementia (instead of Lewy Body dementia). The only licensed proven treatment for PD dementia demonstrated in a randomised clinical trial is rivastigmine. Donepezil is also useful for PD dementia but is not currently licensed in the UK. Neither DBS nor increasing PD medications improve cognitive impairment.

Emre M, Aarsland D, Albanese A et al. Rivastigmine for dementia associated with Parkinson's disease. NEJM 2004; Dec 9;351(24):2509-18

Discuss (8) Improve

Next question >

Parkinson's disease: management

Parkinsons disease should only be diagnosed, and management initiated, by a specialist with expertise in movement disorders. However, it is important for all doctors to be aware of the medications used in Parkinson's given the prevalence of this condition. NICE published guidelines in 2017 regarding the management of Parkinson's disease.

For first-line treatment:

- if the motor symptoms are affecting the patient's quality of life: levodopa
- if the motor symptoms are not affecting the patient's quality of life: dopamine agonist (non-ergot derived), levodopa or monoamine oxidase B (MAO-B) inhibitor

Whilst all drugs used to treat Parkinson's can cause a wide variety of side-effects NICE produced tables to help with decision making:

	Levodopa	**Dopamine agonists**	**MAO-B inhibitors**
Motor symptoms	More improvement in motor symptoms	Less improvement in motor symptoms	Less improvement in motor symptoms
Activities of daily living	More improvement in activities of daily living	Less improvement in activities of daily living	Less improvement in activities of daily living
Motor complications	More motor complications	Fewer motor complications	Fewer motor complications
Adverse events	Fewer specified adverse events*	More specified adverse events*	Fewer specified adverse events*

* excessive sleepiness, hallucinations and impulse control disorders

If a patient continues to have symptoms despite optimal levodopa treatment or has developed dyskinesia then NICE recommend the addition of a dopamine agonist, MAO-B inhibitor or catechol-O-methyl transferase (COMT) inhibitor as an adjunct. Again, NICE summarise the main points in terms of decision making:

	Dopamine agonists	**MAO-B inhibitors**	**COMT inhibitors**	**Amantadine**
Motor symptoms	Improvement in motor symptoms	Improvement in motor symptoms	Improvement in motor symptoms	No evidence of improvement in motor symptoms

	Dopamine agonists	MAO-B inhibitors	COMT inhibitors	Amantadine
Activities of daily living	Improvement in activities of daily living	Improvement in activities of daily living	Improvement in activities of daily living	No evidence of improvement in activities of daily living
Off time	More off-time reduction	Off-time reduction	Off-time reduction	No studies reporting this outcome
Adverse events	Intermediate risk of adverse events	Fewer adverse events	More adverse events	No studies reporting this outcome
Hallucinations	More risk of hallucinations	Lower risk of hallucinations	Lower risk of hallucinations	No studies reporting this outcome

Specific points regarding Parkinson's medication

NICE reminds us of the risk of acute akinesia or neuroleptic malignant syndrome if medication is not taken/absorbed (for example due to gastroenteritis) and advise against giving patients a 'drug holiday' for the same reason.

Impulse control disorders have become a significant issue in recent years. These can occur with any dopaminergic therapy but are more common with:

- dopamine agonist therapy
- a history of previous impulsive behaviours
- a history of alcohol consumption and/or smoking

If excessive daytime sleepiness develops then patients should not drive. Medication should be adjusted to control symptoms. Modafinil can be considered if alternative strategies fail.

If orthostatic hypotension develops then a medication review looking at potential causes should be done. If symptoms persist then midodrine (acts on peripheral alpha-adrenergic receptors to increase arterial resistance) can be considered.

Consider glycopyrronium bromide to manage drooling of saliva in people with Parkinson's

disease.

Further information regarding specific anti-Parkinson's medication

Levodopa

- nearly always combined with a decarboxylase inhibitor (e.g. carbidopa or benserazide)
    - this prevents the peripheral metabolism of levodopa to dopamine outside of the brain and hence can reduce side effects
- common adverse effects:
    - dry mouth
    - anorexia
    - palpitations
    - postural hypotension
    - psychosis
- some adverse effects are due to the difficulty in achieving a steady dose of levodopa
    - end-of-dose wearing off: symptoms often worsen towards the end of dosage interval. This results in a decline of motor activity
    - 'on-off' phenomenon: large variations in motor performance, with normal function during the 'on' period, and weakness and restricted mobility during the 'off' period
    - dyskinesias at peak dose: dystonia, chorea and athetosis (involuntary writhing movements)
    - these effects may worsen over time with - clinicians therefore may limit doses until necessary
- it is important not to acutely stop levodopa, for example, if a patient is admitted to hospital
    - if a patient with Parkinson's disease cannot take levodopa orally, they can be given a dopamine agonist patch as rescue medication to prevent acute dystonia

Dopamine receptor agonists

- e.g. bromocriptine, ropinirole, cabergoline, apomorphine
- ergot-derived dopamine receptor agonists (bromocriptine, cabergoline) have been associated with pulmonary, retroperitoneal and cardiac fibrosis. The Committee on Safety of Medicines advice that an echocardiogram, ESR, creatinine and chest x-ray should be obtained prior to treatment and patients should be closely monitored
- patients should be warned about the potential for dopamine receptor agonists to cause impulse control disorders and excessive daytime somnolence
- more likely than levodopa to cause hallucinations in older patients. Nasal congestion and postural hypotension are also seen in some patients

MAO-B (Monoamine Oxidase-B) inhibitors

- e.g. selegiline
- inhibits the breakdown of dopamine secreted by the dopaminergic neurons

Amantadine

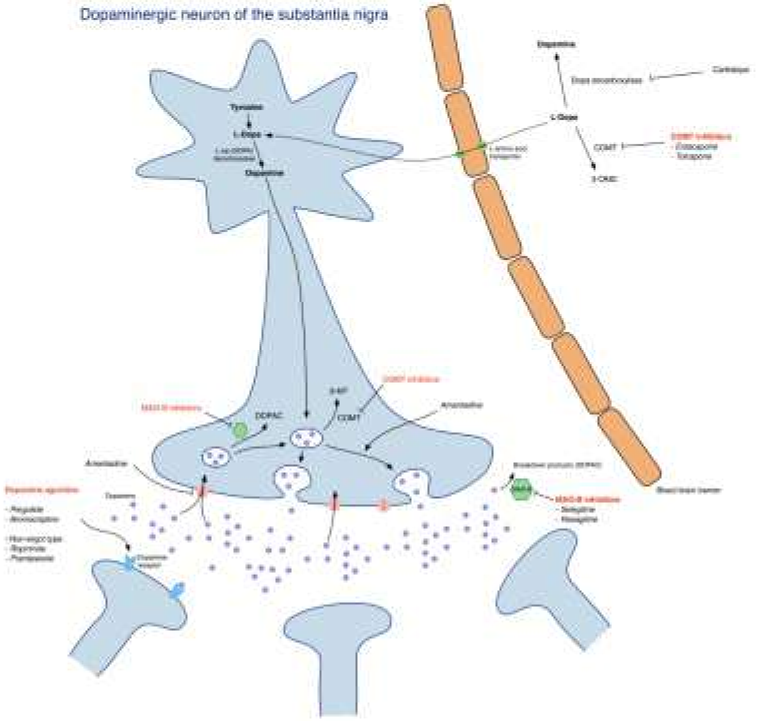
- mechanism is not fully understood, probably increases dopamine release and inhibits its uptake at dopaminergic synapses
- side-effects include ataxia, slurred speech, confusion, dizziness and livedo reticularis

COMT (Catechol-O-Methyl Transferase) inhibitors

- e.g. entacapone, tolcapone
- COMT is an enzyme involved in the breakdown of dopamine, and hence may be used as an adjunct to levodopa therapy
- used in conjunction with levodopa in patients with established PD

Antimuscarinics

- block cholinergic receptors
- now used more to treat drug-induced parkinsonism rather than idiopathic Parkinson's disease
- help tremor and rigidity
- e.g. procyclidine, benzotropine, trihexyphenidyl (benzhexol)


Dopaminergic neuron of the substantia nigra
Tyrosine
L-Dopa
Dopamine
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COMT
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3-MT
COMT
Amantadine
Amantadine
Dopamine agonists
MAO-B inhibitors
Blood brain barrier

Image sourced from Wikipedia

Diagram showing the mechanism of action of Parkinson's drugs

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

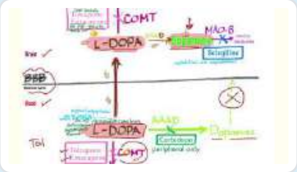
NICE 👍 23 👎 12

2017 Parkinson's disease guidelines

Suggest link

Report broken link

Media


COMT
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MAO-B
BBB
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AAAD
Carbidopa
COMT
Dopamine

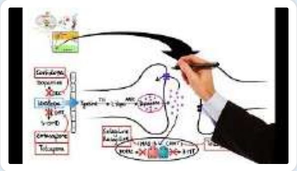
The use of Levodopa/ Carbidopa in the treatment of Parkinson's disease

Medicosis Perfectionalis - YouTube 👍 13 👎 0

PARKINSON'S
DISEASE
PHARMACOLOGY
MAO-B
COMT

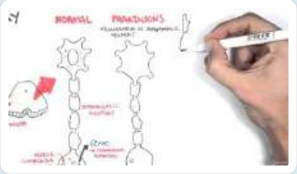
Parkinson's disease pharmacology

Soton Brain Hub - YouTube 👍 4 👎 0



Pharmacology - DRUGS FOR PARKINSON'S DISEASE

Speed Pharmacology - YouTube 👍 8 👎 1


NORMAL
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Pharmacology - Parkinson's Disease

Armando Hasudungan - YouTube 👍 16 👎 2

PARKINSON'S
DISEASE
Osmosis.org

Parkinson's disease

Osmosis - YouTube 👍 4 👎 1

PARKINSON'S
DISEASE
SHUFFLING GAIT
PILL ROLLING TREMOR
COG-WHEEL RIGIDITY
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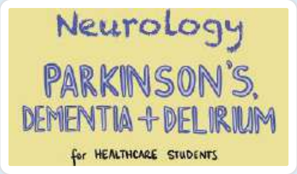
Understanding Parkinson's Disease

Rhesus Medicine - YouTube 👍 2 👎 1


Neurology
PARKINSON'S,
DEMENTIA + DELIRIUM
for HEALTHCARE STUDENTS

Parkinson's disease

MedFlix - YouTube 👍 1 👎 2

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Score: **24.8%**

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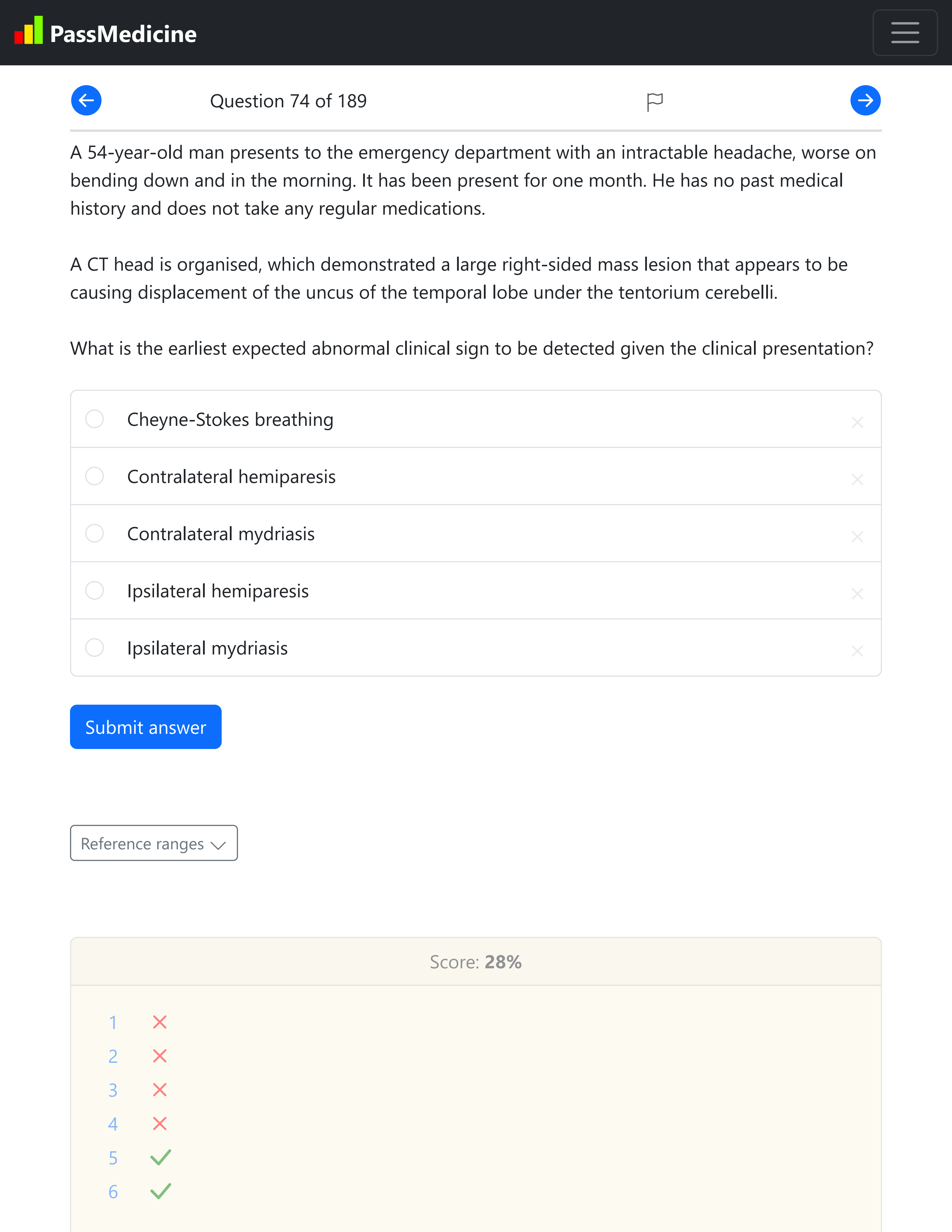

A 54-year-old man presents to the emergency department with an intractable headache, worse on bending down and in the morning. It has been present for one month. He has no past medical history and does not take any regular medications.

A CT head is organised, which demonstrated a large right-sided mass lesion that appears to be causing displacement of the uncus of the temporal lobe under the tentorium cerebelli.

What is the earliest expected abnormal clinical sign to be detected given the clinical presentation?

- ○ Cheyne-Stokes breathing
- ○ Contralateral hemiparesis
- ○ Contralateral mydriasis
- ○ Ipsilateral hemiparesis
- ○ Ipsilateral mydriasis

Score: **28%**

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A 54-year-old man presents to the emergency department with an intractable headache, worse on bending down and in the morning. It has been present for one month. He has no past medical history and does not take any regular medications.

A CT head is organised, which demonstrated a large right-sided mass lesion that appears to be causing displacement of the uncus of the temporal lobe under the tentorium cerebelli.

What is the earliest expected abnormal clinical sign to be detected given the clinical presentation?

Cheyne-Stokes breathing	16%
Contralateral hemiparesis	21%
Contralateral mydriasis	21%
Ipsilateral hemiparesis	5%
Ipsilateral mydriasis	37%

Uncal herniation results in pressure on the midbrain resulting in ipsilateral CN III palsy (mydriasis is the earliest sign) and contralateral hemiparesis

Important for me Less important

Ipsilateral mydriasis is correct. The CT demonstrates uncal herniation as a consequence of an intracranial mass lesion. The ipsilateral dilated pupil is caused by compression of the ipsilateral oculomotor nerve.

Contralateral mydriasis is incorrect. This is a rarer clinical sign. It may develop from contralateral oculomotor nerve compression from midline shift.

Contralateral hemiparesis is incorrect. This is an expected consequence of progressive uncal herniation but typically occurs subsequent to ipsilateral mydriasis.

Ipsilateral hemiparesis is incorrect. This is rarer than contralateral hemiparesis. However, it can occur as a consequence of the 'Kernohan phenomenon'. This is a radiological description of extensive midline shift causing mass effect. The result is denting of the contralateral cerebral crus by the tentorium cerebelli. . The corticospinal tracts are damaged and a hemiparesis contralateral to the crus cerebri results. This is ipsilateral to the mass lesion and is a false localizing sign. It is a rarer occurrence and would occur subsequent to mydriasis.

Cheyne-Stokes breathing is incorrect. This signifies impending death and is a terminal sign of progressive cerebral herniation.

👍 👎 Discuss (2) Improve

Next question >

Brain herniation ★

As intracranial pressure rises to pathological levels, normal brain structures are forcefully displaced. This is called herniation.

Herniation is a decompensation of normal brain anatomy and physiology. Displacement of brain causes compression of important structures, the most important of which is the brain stem. Brainstem compression is called 'coning'. It is a grave sign and usually indicates a neurosurgical emergency requiring intervention (e.g. osmotherapy with hypertonic saline or manitol, or surgical decompression).

Type of herniation	Description
Subfalcine	Displacement of the cingulate gyrus under the falx cerebri
Central	Downwards displacement of the brain
Transtentorial / uncal herniation	Displacement of the uncus of the temporal lobe under the tentorium cerebelli. Clinical consequences include an ipsilateral fixed, dilated pupil (due to parasympathetic compression of the third cranial nerve) and contralateral paralysis (due to compression of the cerebral peduncle)
Tonsillar	Displacement of the cerebellar tonsils through the foramen magnum. This is called 'coning'. In raised ICP this causes compression of the cardiorespiratory centre. In Chiari 1 malformation, tonsillar herniation is seen without raised ICP
Transcalvarial	Occurs when brain is displaced through a defect in the skull (e.g. a fracture or craniotomy site)

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

INCREASED INTRACRANIAL PRESSURE AND
BRAIN HERNIATION
Osmosis.org

Brain herniation

Osmosis - YouTube 👍 4 👎 0

Report broken media

Score: **24.8%**

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Question 75 of 189

A 65-year-old female is referred to the neurology clinic with complaints of tingling and loss of sensation. Five years previously she experienced an episode of numbness and tingling affecting her right upper limb. Most recently, there has been a loss of sensation affecting her left lower limb. There has been a full recovery between episodes. She has no past medical history and does not take any regular medications.

The examination at the time of review is unremarkable.

An MRI of her brain demonstrates lesions in the periventricular white matter.

Given the likely diagnosis, what clinical feature of her presentation portends the worst prognosis?

○ Age of onset

○ Complete recovery between relapses

○ Relapsing-remitting disease

○ Sensory symptoms only

○ 5 year interval between first two relapses

Submit answer

Reference ranges

Score: **28%**

1 ×
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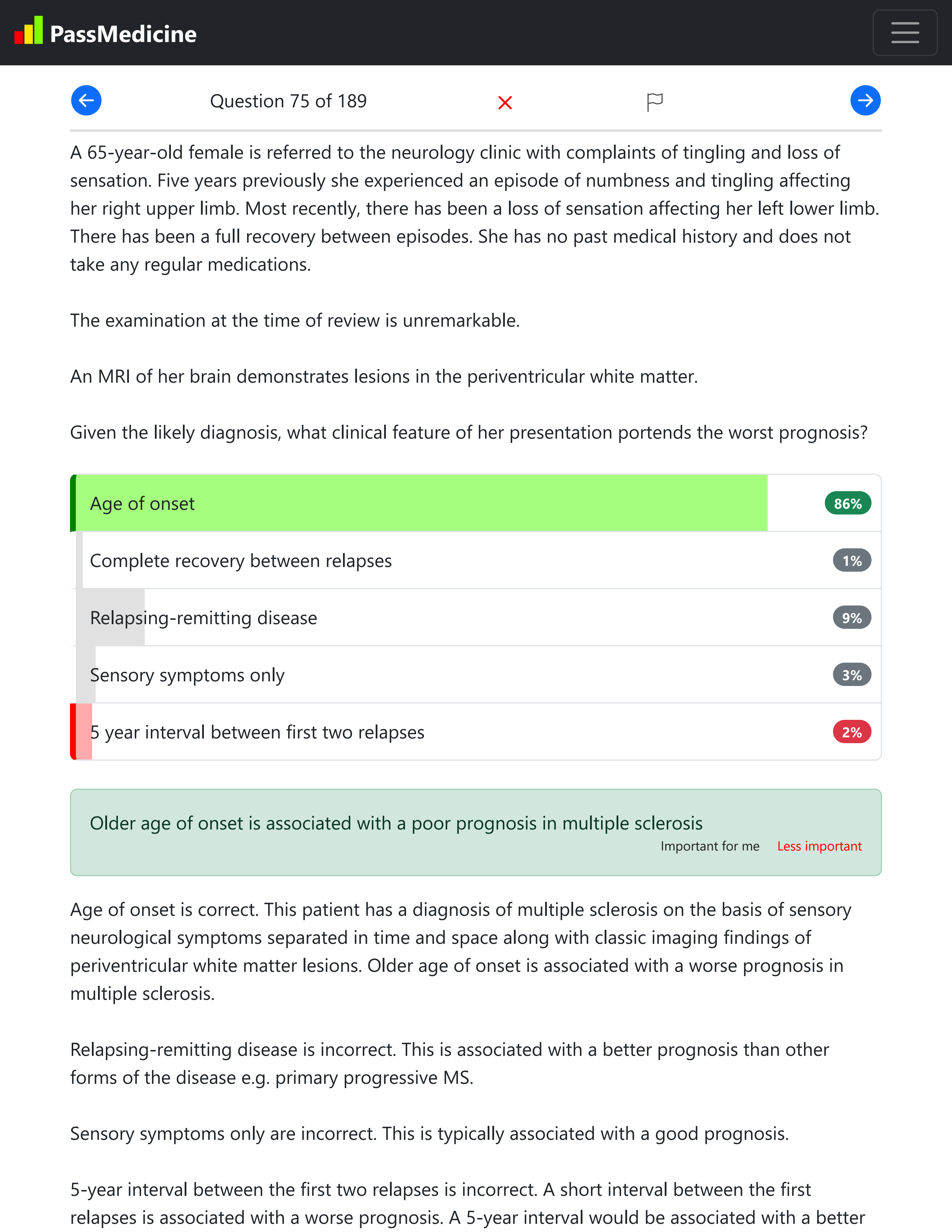

A 65-year-old female is referred to the neurology clinic with complaints of tingling and loss of sensation. Five years previously she experienced an episode of numbness and tingling affecting her right upper limb. Most recently, there has been a loss of sensation affecting her left lower limb. There has been a full recovery between episodes. She has no past medical history and does not take any regular medications.

The examination at the time of review is unremarkable.

An MRI of her brain demonstrates lesions in the periventricular white matter.

Given the likely diagnosis, what clinical feature of her presentation portends the worst prognosis?

Age of onset	86%
Complete recovery between relapses	1%
Relapsing-remitting disease	9%
Sensory symptoms only	3%
5 year interval between first two relapses	2%

Older age of onset is associated with a poor prognosis in multiple sclerosis

Important for me Less important

Age of onset is correct. This patient has a diagnosis of multiple sclerosis on the basis of sensory neurological symptoms separated in time and space along with classic imaging findings of periventricular white matter lesions. Older age of onset is associated with a worse prognosis in multiple sclerosis.

Relapsing-remitting disease is incorrect. This is associated with a better prognosis than other forms of the disease e.g. primary progressive MS.

Sensory symptoms only are incorrect. This is typically associated with a good prognosis.

5-year interval between the first two relapses is incorrect. A short interval between the first relapses is associated with a worse prognosis. A 5-year interval would be associated with a better prognosis.

Complete recovery between relapse is incorrect. If the patient did not recover fully between relapses then this would be associated with a worse prognosis.

Discuss Improve

Next question ›

Multiple sclerosis: prognostic features

Good prognosis features

- female sex
- age: young age of onset (i.e. 20s or 30s)
- relapsing-remitting disease
- sensory symptoms only
- long interval between first two relapses
- complete recovery between relapses

Ways of remembering prognostic features

- the typical patient carries a better prognosis than an atypical presentation

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Score: **24.8%**

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A 56-year-old gentleman is brought by his wife with unusual behaviour. This afternoon, she found him doing gardening in the back garden and he was repeatedly cutting at the same shrubs. He was conscious but asked her repeatedly when dinner was ready. He returned to the house where he had no recall of gardening and continued to ask the same questions repeatedly.

In the department, he seems well. He is a little confused over what has happened but is orientated in time and place. On examination, there is no ophthalmoplegia but there were two beats of nystagmus seen. He has 5/5 power in all limbs with normal tone and no myoclonus or fasciculations. His gait is normal.

His wife reports that there have been no memory problems or change in personality prior to today. He seems to be back to normal at present. He denies any excessive alcohol use and his father did have Alzheimer's disease which was diagnosed at 60 years of age.

Na^+	138 mmol/l
K^+	4.2 mmol/l
Urea	5.6 mmol/l
Creatinine	78 µmol/l

Bilirubin	27 µmol/l
ALP	105 u/l
ALT	30 u/l
Albumin	34 g/l

Hb	110 g/l
Platelets	394 * 10^9/l
WBC	5.2 * 10^9/l
MCV	96 fL (normal 80-100)
B12	250 pg/ml (normal 180-1000)
Folate	5 ng/ml (normal >4)
CT head	age related involutional changes, old lacunar infarcts seen on left side, no intracranial haemorrhage

What is the likely diagnosis?

- ○ Transient ischaemic attack ×
- ○ Creutzfeldt-Jacob Disease ×
- ○ Alzheimer's Disease ×
- ○ Wernicke's encephalopathy ×
- ○ Transient global amnesia ×

Submit answer

Reference ranges ∨

Score: **28%**

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Folate	5 ng/ml (normal >4)
CT head	age related involutional changes, old lacunar infarcts seen on left side, no intracranial haemorrhage

What is the likely diagnosis?

Transient ischaemic attack	4%
Creutzfeldt-Jacob Disease	5%
Alzheimer's Disease	3%
Wernicke's encephalopathy	2%
Transient global amnesia	86%

Transient global amnesia - acute onset of anterograde amnesia, patent may keep asking the same questions, resolves within 24 hours

Important for me Less important

This gentleman presents with an acute episode of amnesia that has resolved in a number of hours. He has no neurological abnormality on examination and has returned to normal. Alzheimer's disease and Wernicke's encephalopathy would not present this acutely. Creutzfeldt-Jacob Disease has a rapidly progressive dementia and would not resolve after some hours. A TIA is possible but usually presents with stroke-like symptoms and isolated amnesia would be hard to reconcile neurologically. Transient global amnesia is a poorly understood phenomenon that may be related to migraines in pathophysiology where the individual has a profound amnesia for hours to days and is not able to form new memories. They resolve and are well following this but it can occur again.

👍 👎 Discuss (5) Improve

Next question ›

Transient global amnesia ★

Transient global amnesia is characterized by the acute onset of anterograde amnesia (the inability to form new memories). The aetiology is unknown, thought to be due to transient ischaemia to the thalamus (in particular the amygdala and hippocampus).

Features

- patients may appear anxious and repeatedly ask the same question
- episodes are self-limited and resolve within 24 hours

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **24.8%**

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Question 77 of 189

A 32 year old man presents to your haematology clinic with a 4 year history of fatigue, weight loss and a feeling of fullness in the stomach. He has been off sick from work for the past 3 months and has recently been divorced. He also complains of low mood and has attempted suicide in the past. He has a family history of NHL. He has been investigated in the past with CT scans, blood films and lymph node biopsies on several occasions with the most recent set of investigations occurring 6 months ago.

Despite your reassurances that he does not have a lymphoma, he is still convinced that he does and that he is dying. You suggest a referral to a psychiatrist and he becomes very angry. What is the most likely underlying diagnosis?

- ○ Atypical depression
- ○ Specific phobia
- ○ Hypochondriasis
- ○ Conversion disorder
- ○ Somatoform disorder

Submit answer

Reference ranges

Score: **28%**

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Despite your reassurances that he does not have a lymphoma, he is still convinced that he does and that he is dying. You suggest a referral to a psychiatrist and he becomes very angry. What is the most likely underlying diagnosis?

Atypical depression	3%
Specific phobia	2%
Hypochondriasis	73%
Conversion disorder	9%
Somatoform disorder	13%

Unexplained symptoms
- **S**omatisation = **S**ymptoms
- hypo**C**hondria = **C**ancer

Important for me Less important

The answer is hypochondriasis. For a diagnosis is hypochondriasis to be made the patient needs to have a persistent belief that he or she has a serious illness which may be related to misperception of bodily symptoms. Physical causes need to have been ruled out and the patient is not reassured despite overwhelming evidence to the contrary. In addition there needs to be an impact on psychosocial functioning. In this case the patient fits all of these criteria.

Patients with hypochondriasis are likely to have concurrent depression, although depression is not the primary diagnosis here. Hypochondriasis is more common in those who have lost a close family member to a serious illness.

In conversion disorder and somatoform psychological trauma is manifested as physical symptoms. In these disorders, it is a preoccupation with the symptoms which is the problem, rather than fear of a diagnosis. Whilst this patient has a fear of having lymphoma, hypochondriasis is a more appropriate diagnosis.

👍 👎 Discuss (2) Improve

Next question ›

Unexplained symptoms ★

There are a wide variety of psychiatric terms for patients who have symptoms for which no organic cause can be found:

Somatisation disorder
- multiple physical SYMPTOMS present for at least 2 years
- patient refuses to accept reassurance or negative test results

Illness anxiety disorder (hypochondriasis)
- persistent belief in the presence of an underlying serious DISEASE, e.g. cancer
- patient again refuses to accept reassurance or negative test results

Functional neurological disorder (conversion disorder)
- typically involves loss of motor or sensory function
- the patient doesn't consciously feign the symptoms (factitious disorder) or seek material gain (malingering)
- patients may be indifferent to their apparent disorder - la belle indifference - although this has not been backed up by some studies

Dissociative disorder
- dissociation is a process of 'separating off' certain memories from normal consciousness
- in contrast to conversion disorder involves psychiatric symptoms e.g. Amnesia, fugue, stupor
- dissociative identity disorder (DID) is the new term for multiple personality disorder as is the most severe form of dissociative disorder

Factitious disorder
- also known as Munchausen's syndrome

- the intentional production of physical or psychological symptoms

Malingering
- fraudulent simulation or exaggeration of symptoms with the intention of financial or other gain

+ Q 123

Next question >

B *I*

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 12 👎 16

2013 Functional neurological symptoms

Suggest link

Report broken link

Media

Somatic symptom disorder

Osmosis - YouTube 👍 1 👎 0

Factitious disorder

Osmosis - YouTube 👍 1 👎 0

DISSOCIATIVE DISORDERS

Dissociative disorders

Osmosis - YouTube 👍 1 👎 0

Report broken media

Score: **24.8%**

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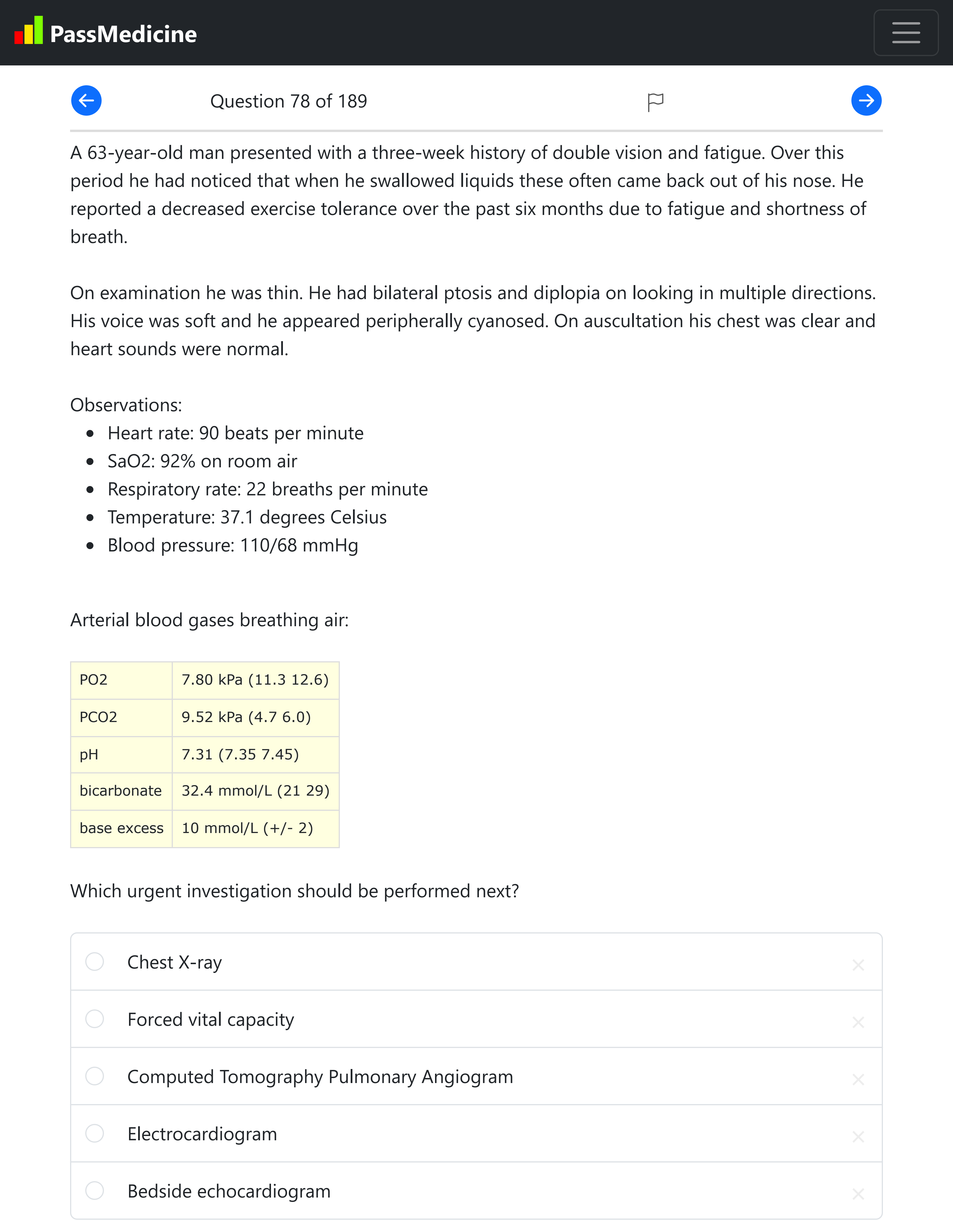

A 63-year-old man presented with a three-week history of double vision and fatigue. Over this period he had noticed that when he swallowed liquids these often came back out of his nose. He reported a decreased exercise tolerance over the past six months due to fatigue and shortness of breath.

On examination he was thin. He had bilateral ptosis and diplopia on looking in multiple directions. His voice was soft and he appeared peripherally cyanosed. On auscultation his chest was clear and heart sounds were normal.

Observations:
- Heart rate: 90 beats per minute
- SaO2: 92% on room air
- Respiratory rate: 22 breaths per minute
- Temperature: 37.1 degrees Celsius
- Blood pressure: 110/68 mmHg

Arterial blood gases breathing air:

PO2	7.80 kPa (11.3 12.6)
PCO2	9.52 kPa (4.7 6.0)
pH	7.31 (7.35 7.45)
bicarbonate	32.4 mmol/L (21 29)
base excess	10 mmol/L (+/- 2)

Which urgent investigation should be performed next?

- ○ Chest X-ray ×
- ○ Forced vital capacity ×
- ○ Computed Tomography Pulmonary Angiogram ×
- ○ Electrocardiogram ×
- ○ Bedside echocardiogram ×

Submit answer

Reference ranges ⌵

Score: **28%**

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Which urgent investigation should be performed next?

Chest X-ray	13%
Forced vital capacity	82%
Computed Tomography Pulmonary Angiogram	3%
Electrocardiogram	1%
Bedside echocardiogram	2%

This patient has myasthenia gravis as suggested by his fatigue, ptosis, complex ophthalmoplegia and bulbar weakness. He has an element of neuromuscular weakness which is resulting in type 2 respiratory failure. The most important step after adequately resuscitating this patient is to perform a bedside Forced Vital Capacity measurement. If this is < 1.5 then it may be necessary to involve the Intensive Treatment Unit team and consider ventilatory support.

👍 👎 Discuss (7) Improve

Next question >

Myasthenia gravis

Myasthenia gravis is an autoimmune disorder resulting in insufficient functioning acetylcholine receptors. Antibodies to acetylcholine receptors are seen in 85-90% of cases*. Myasthenia is more common in women (2:1)

The key feature is muscle fatigability - muscles become progressively weaker during periods of activity and slowly improve after periods of rest:

- extraocular muscle weakness: diplopia
- proximal muscle weakness: face, neck, limb girdle
- ptosis
- dysphagia

Associations

- thymomas in 15%
- autoimmune disorders: pernicious anaemia, autoimmune thyroid disorders, rheumatoid, SLE
- thymic hyperplasia in 50-70%

Investigations

- single fibre electromyography: high sensitivity (92-100%)
- CT thorax to exclude thymoma
- CK normal
- antibodies to acetylcholine receptors
    - positive in around 85-90% of patients
    - n the remaining patients, about about 40% are positive for anti-muscle-specific tyrosine kinase antibodies
- Tensilon test: IV edrophonium reduces muscle weakness temporarily - not commonly used any more due to the risk of cardiac arrhythmia

Management

- long-acting acetylcholinesterase inhibitors
    - pyridostigmine is first-line
- immunosuppression is usually not started at diagnosis, but the majority of patients eventually require it in addition to long-acting acetylcholinesterase inhibitors:
    - prednisolone initially
    - azathioprine, cyclosporine, mycophenolate mofetil may also be used
- thymectomy

Management of myasthenic crisis

- plasmapheresis
- intravenous immunoglobulins

*antibodies are less commonly seen in disease limited to the ocular muscles

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

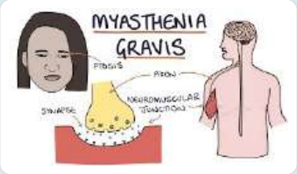
Postgraduate Medical Journal 13 3

Review of myasthenia gravis

Suggest link

Report broken link

Media


MYASTHENIA GRAVIS
PTOSIS
SYNAPSE
AXON
NEUROMUSCULAR JUNCTION

Myasthenia gravis

Zero to Finals - YouTube 👍 12 👎 1

NEUROLOGY
MYASTHENIA GRAVIS

Myasthenia gravis

Armando Hasudungan - YouTube 👍 1 👎 1

MYASTHENIA GRAVIS

Myasthenia gravis - causes, symptoms, treatment, pathology

Osmosis - YouTube 👍 2 👎 2

Report broken media

Score: **24.8%**

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PassMedicine

A 59-year-old woman is referred by her GP to neurology clinic for assessment. The patient describes intermittent episodes of pain affecting the left side of her face. She recalls the first attack vividly as a sudden onset of severe 'electric-shock' pain coming on suddenly around one year previously as she had cleaned her teeth with her electric toothbrush. The pain was felt around the cheek extending down to the jaw-line. The first attack had lasted several hours and she had ultimately attended her dentist and received a filling to a left molar tooth. While symptoms from that episode had resolved, she had then suffered from similar episodes at increasing frequency, approximately every fortnight over the last couple of months. These attacks had on occasion been unprovoked but were sometimes induced by stimulation of the affected area or cold winds. The patient was tearful as she recounted the negative impact her symptoms had had one her lifestyle. There was no history of headaches, sinus symptoms or seizures.

The patient had a fairly unremarkable past-medical history, notable only for hypothyroidism and long-standing struggles to avoid overweight. Her only regular medication was thyroxine 125 micrograms daily and the patient reported no allergies. There was no family history of neurological disease. The patient worked as a law clerk and did not smoke or drink significant alcohol.

Examination demonstrated an unremarkable cranial nerve and peripheral nerve examinations. In particular, colour vision, visual acuity and hearing where normal.

What is the correct first line management of the patients symptoms?

- ○ Gabapentin
- ○ Carbamazepine
- ○ Pregabalin
- ○ Baclofen
- ○ Lamotrigine

Submit answer

Reference ranges

Score: **28%**

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A 59-year-old woman is referred by her GP to neurology clinic for assessment. The patient describes intermittent episodes of pain affecting the left side of her face. She recalls the first attack vividly as a sudden onset of severe 'electric-shock' pain coming on suddenly around one year previously as she had cleaned her teeth with her electric toothbrush. The pain was felt around the cheek extending down to the jaw-line. The first attack had lasted several hours and she had ultimately attended her dentist and received a filling to a left molar tooth. While symptoms from that episode had resolved, she had then suffered from similar episodes at increasing frequency, approximately every fortnight over the last couple of months. These attacks had on occasion been unprovoked but were sometimes induced by stimulation of the affected area or cold winds. The patient was tearful as she recounted the negative impact her symptoms had had one her lifestyle. There was no history of headaches, sinus symptoms or seizures.

The patient had a fairly unremarkable past-medical history, notable only for hypothyroidism and long-standing struggles to avoid overweight. Her only regular medication was thyroxine 125 micrograms daily and the patient reported no allergies. There was no family history of neurological disease. The patient worked as a law clerk and did not smoke or drink significant alcohol.

Examination demonstrated an unremarkable cranial nerve and peripheral nerve examinations. In particular, colour vision, visual acuity and hearing where normal.

What is the correct first line management of the patients symptoms?

Gabapentin	4%
Carbamazepine	92%
Pregabalin	3%
Baclofen	1%
Lamotrigine	1%

The patient reports a classical history of trigeminal neuralgia. Notable features are the memorable first occurrence, attacks precipitated by touch or vibration and the history of inappropriate dental treatment. Time between attacks often gradually reduces leaving patients quality of life significantly impaired.

The patient has no other symptoms or neurological signs that might suggest an alternative diagnosis such as multiple sclerosis, sinusitis or a space-occupying lesion. In the presence of such features, an MRI brain is the most appropriate investigation.

Carbamazepine is the only drug licensed specifically for trigeminal neuralgia in the UK. In 70 % of patients it provides initial 100 % pain relief. However, caution must be used due to side-effects and drug interactions. Oxcarbazepine is a derivative of carbamazepine with similar efficacy but improved tolerability and side-effect profile.

In the evidence of allergy or intolerance to the above drugs then use of baclofen or lamotrigine should be considered, however the evidence base for both is thin. Gabapentin in combination with ropivacaine injections did show benefit in one randomised controlled trial but this result has not been replicated in subsequent investigations. Pregabalin had reported effectiveness in one prospective study of 53 patients.

Zakrzewska J, Linskey M. Trigeminal neuralgia. BMJ 2014;348:g474.

👍 👎 Discuss (1) Improve

Next question >

Trigeminal neuralgia

Trigeminal neuralgia is a pain syndrome characterised by severe unilateral pain. The vast majority of cases are idiopathic but compression of the trigeminal roots by tumours or vascular problems may occur.

The International Headache Society defines trigeminal neuralgia as:

- a unilateral disorder characterised by brief electric shock-like pains, abrupt in onset and termination, limited to one or more divisions of the trigeminal nerve
- the pain is commonly evoked by light touch, including washing, shaving, smoking, talking, and brushing the teeth (trigger factors), and frequently occurs spontaneously
- small areas in the nasolabial fold or chin may be particularly susceptible to the precipitation of pain (trigger areas)
- the pains usually remit for variable periods

The following as red flag symptoms and signs suggesting a serious underlying cause:

- Sensory changes
- Deafness or other ear problems
- History of skin or oral lesions that could spread perineurally

- Pain only in the ophthalmic division of the trigeminal nerve (eye socket, forehead, and nose), or bilaterally
- Optic neuritis
- A family history of multiple sclerosis
- Age of onset before 40 years

Management
- carbamazepine is first-line
- failure to respond to treatment or atypical features (e.g. < 50 years old) should prompt referral to neurology

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 19 👎 3

Trigeminal Neuralgia guidelines

Suggest link Report broken link

Score: **24.8%**

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A 78-year-old man is admitted following being found on the floor at home. He has no recollection of how he got to floor or how long he had been there. He reports feeling generally unwell and having a cough for a number of days. There is no medical history of note and he takes no regular medications. He lives alone and appears unkempt. Examination reveals bronchial breathing throughout his left mid zone. Neurologically, he has new onset weakness of left sided shoulder abduction and adduction, alongside mild weakness in left elbow flexion. Additionally, reduced sensation in the lateral aspect of his upper arm is noted. A CT head is undertaken.

CT head report	Age related involutional change. No evidence of intracranial haemorrhage or recent ischaemic event.

What is the most likely diagnosis?

- ○ Stroke
- ○ Rotator cuff tear
- ○ Brachial plexus injury
- ○ Brown-Sequard syndrome
- ○ Botulism

Submit answer

Reference ranges

Score: **28%**

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Question 80 of 189 ✗

A 78-year-old man is admitted following being found on the floor at home. He has no recollection of how he got to floor or how long he had been there. He reports feeling generally unwell and having a cough for a number of days. There is no medical history of note and he takes no regular medications. He lives alone and appears unkempt. Examination reveals bronchial breathing throughout his left mid zone. Neurologically, he has new onset weakness of left sided shoulder abduction and adduction, alongside mild weakness in left elbow flexion. Additionally, reduced sensation in the lateral aspect of his upper arm is noted. A CT head is undertaken.

CT head report	Age related involutional change. No evidence of intracranial haemorrhage or recent ischaemic event.

What is the most likely diagnosis?

Stroke	5%
Rotator cuff tear	7%
Brachial plexus injury	79%
Brown-Sequard syndrome	5%
Botulism	4%

Given the pattern of neurological signs in his left limb, this would not fit with a stroke. Global weakness and sensory loss would be expected in the affected limb.

A rotator cuff tear is possible given the likely trauma. However, sensory loss would not be expected.

Brown-Sequard syndrome is caused by damage to half of the spinal cord. It produces ipsilateral paralysis and proprioception loss, and contralateral temperature and pain loss.

Botulism classically presents with cranial nerve deficits, followed by a descending weakness and autonomic dysfunction.

A brachial plexus injury may be sustained following trauma, as in this case. The level of injury will correlate with the signs. In this patient, it is at C5, causing the specific distribution of weakness and sensory loss.

Discuss (7) | Improve

Next question >

Brachial plexus injuries ★

Erb-Duchenne paralysis

- damage to C5,6 roots
- winged scapula
- may be caused by a breech presentation

Klumpke's paralysis

- damage to T1
- loss of intrinsic hand muscles
- due to traction

+ Q 123

Next question >

Textbooks

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Media

KLUMPKE'S PALSY & THORACIC OUTLET SYNDROME
Osmosis.org

Klumpke's palsy and thoracic outlet syndrome

Osmosis - YouTube 👍 5 👎 0

Report broken media

Score: **24.8%**

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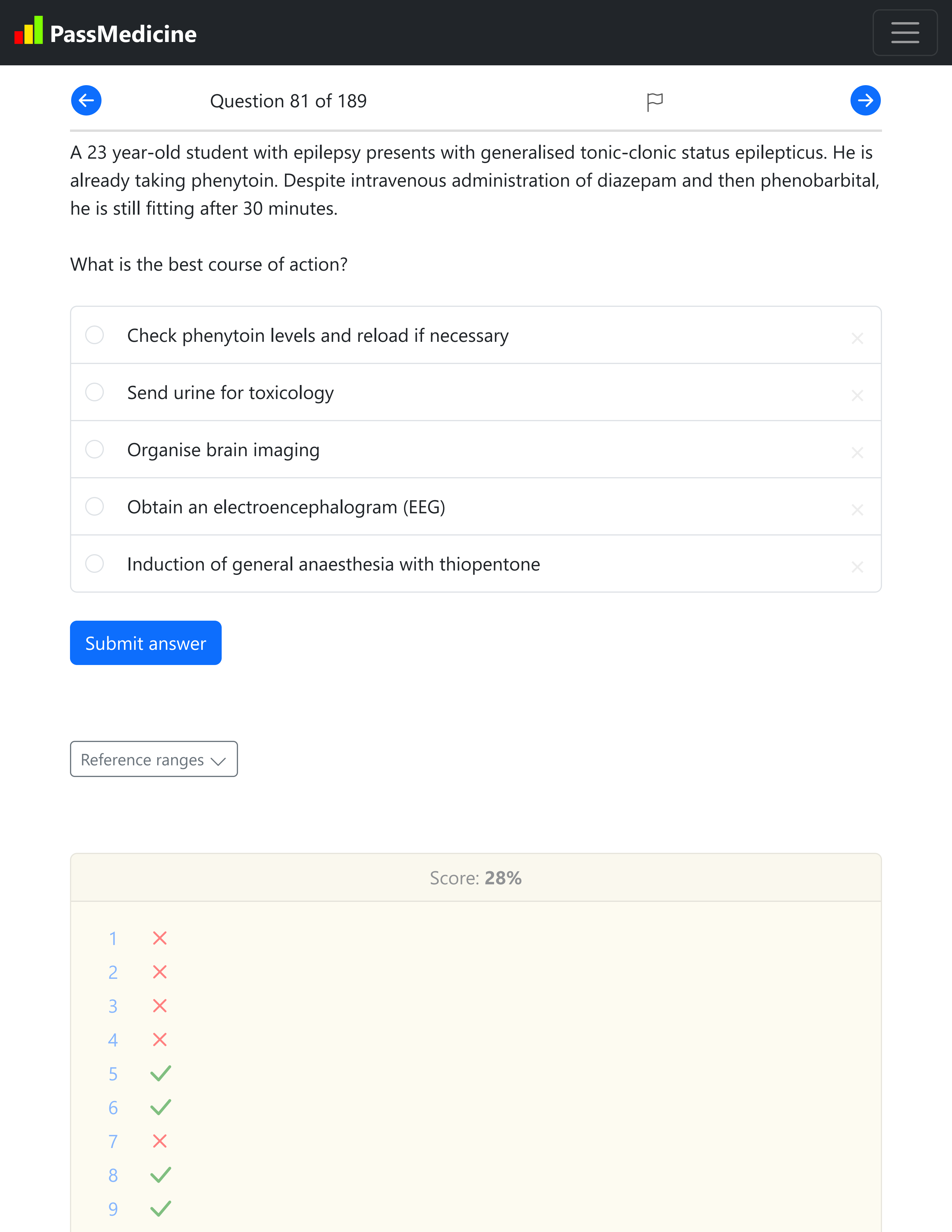

Question 81 of 189

A 23 year-old student with epilepsy presents with generalised tonic-clonic status epilepticus. He is already taking phenytoin. Despite intravenous administration of diazepam and then phenobarbital, he is still fitting after 30 minutes.

What is the best course of action?

- ○ Check phenytoin levels and reload if necessary
- ○ Send urine for toxicology
- ○ Organise brain imaging
- ○ Obtain an electroencephalogram (EEG)
- ○ Induction of general anaesthesia with thiopentone

Submit answer

Reference ranges

Score: **28%**

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A 23 year-old student with epilepsy presents with generalised tonic-clonic status epilepticus. He is already taking phenytoin. Despite intravenous administration of diazepam and then phenobarbital, he is still fitting after 30 minutes.

What is the best course of action?

Check phenytoin levels and reload if necessary	5%
Send urine for toxicology	1%
Organise brain imaging	2%
Obtain an electroencephalogram (EEG)	1%
Induction of general anaesthesia with thiopentone	91%

The correct answer in this scenario is the **induction of general anaesthesia with thiopentone**. In a case of refractory status epilepticus, where seizures have not responded to first and second-line treatments such as benzodiazepines (e.g., diazepam) and anti-epileptic drugs (e.g., phenobarbital), it is important to escalate treatment rapidly. Induction of general anaesthesia with thiopentone can help achieve rapid control of seizures, prevent further neuronal injury, and reduce the risk of long-term complications.

Regarding the other options:

Check phenytoin levels and reload if necessary: While checking phenytoin levels may be useful in determining whether the patient's ongoing seizures are due to subtherapeutic drug levels, it would not be appropriate to delay more definitive treatment in this case. The urgency of treating status epilepticus outweighs waiting for lab results, which could take hours.

Send urine for toxicology: Although identifying an underlying cause for the seizure is important, sending urine for toxicology would not provide immediate information needed to manage this acute situation. Management should prioritize stopping the seizure activity first before investigating potential causes.

Organise brain imaging: Brain imaging can provide valuable information about structural abnormalities that may be contributing to the patient's seizures; however, it is not an immediate priority during an ongoing status epilepticus episode. Once seizure activity has been controlled and stabilised, brain imaging may be considered as part of a comprehensive evaluation.

Obtain an electroencephalogram (EEG): An EEG can help confirm the diagnosis of status epilepticus and monitor response to treatment. However, obtaining an EEG should not delay urgent interventions like induction of general anaesthesia with thiopentone. Ideally, continuous EEG monitoring should be initiated after initiating anaesthesia to assess seizure control and guide further management.

👍 | 👎 | Discuss (3) | Improve

Next question >

Status epilepticus ★

Status epilepticus is defined as:
- a single seizure lasting >5 minutes, or
- >= 2 seizures within a 5-minute period without the person returning to normal between them

This is a medical emergency. The priority is the termination of seizure activity, which if prolonged will lead to irreversible brain damage.

Management
- ABC
  - airway adjunct
  - oxygen
  - check blood glucose
- First-line drugs are benzodiazepines
  - in the prehospital setting PR diazepam or buccal midazolam may be given
  - in hospital IV lorazepam is generally used. This may be repeated once after 5-10minutes
- If ongoing (or 'established') status it is appropriate to start a second-line agent such as levetiracetam, phenytoin or sodium valproate
  - NICE state 'Take into account that levetiracetam may be quicker to administer and have fewer adverse effects than the alternative options.'
- If no response ('refractory status') within 45 minutes from onset, then the best way to achieve rapid control of seizure activity is induction of general anaesthesia or phenobarbital

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 14 👎 9

2022 Epilepsy guidelines

Suggest link Report broken link

Score: **24.8%**

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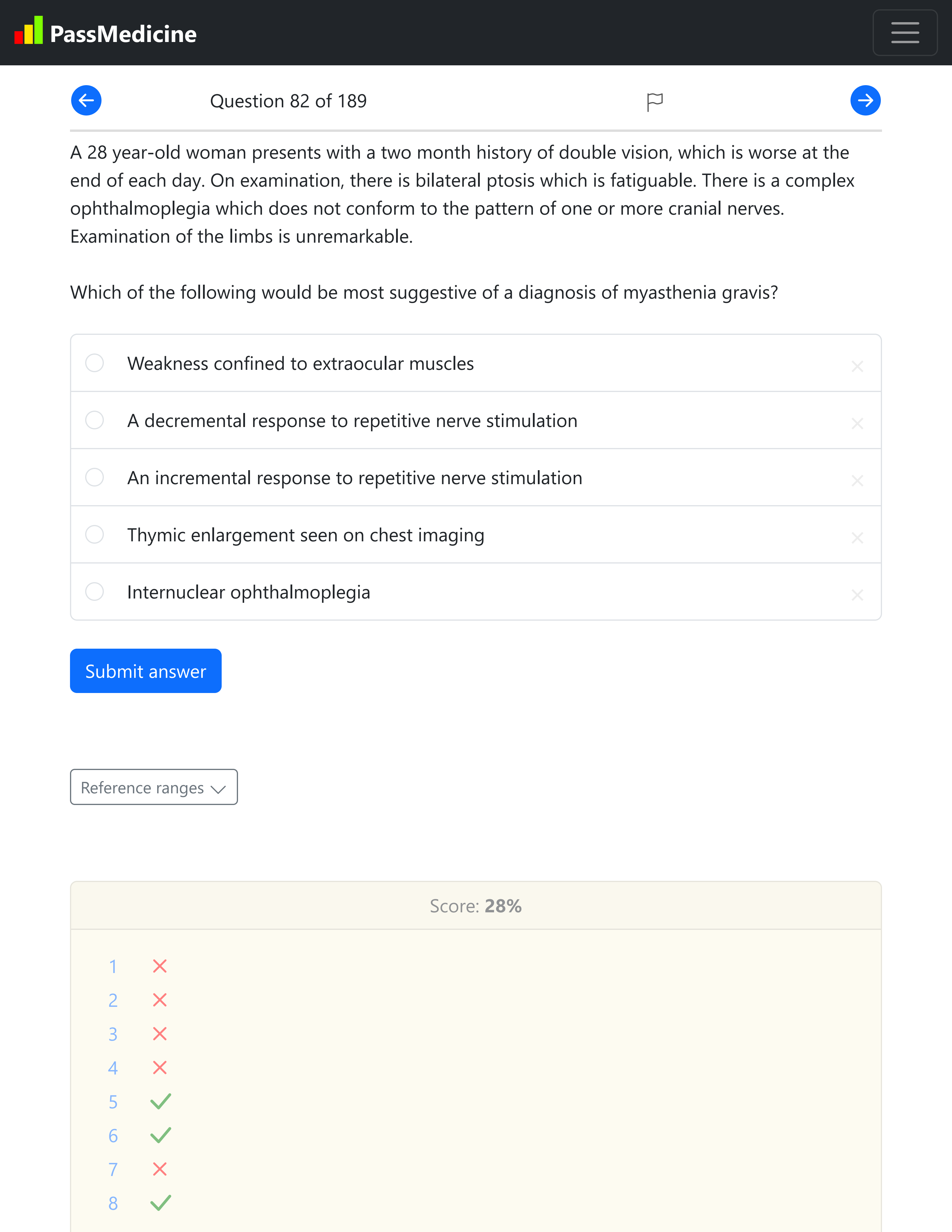

A 28 year-old woman presents with a two month history of double vision, which is worse at the end of each day. On examination, there is bilateral ptosis which is fatiguable. There is a complex ophthalmoplegia which does not conform to the pattern of one or more cranial nerves. Examination of the limbs is unremarkable.

Which of the following would be most suggestive of a diagnosis of myasthenia gravis?

- ○ Weakness confined to extraocular muscles
- ○ A decremental response to repetitive nerve stimulation
- ○ An incremental response to repetitive nerve stimulation
- ○ Thymic enlargement seen on chest imaging
- ○ Internuclear ophthalmoplegia

Score: **28%**

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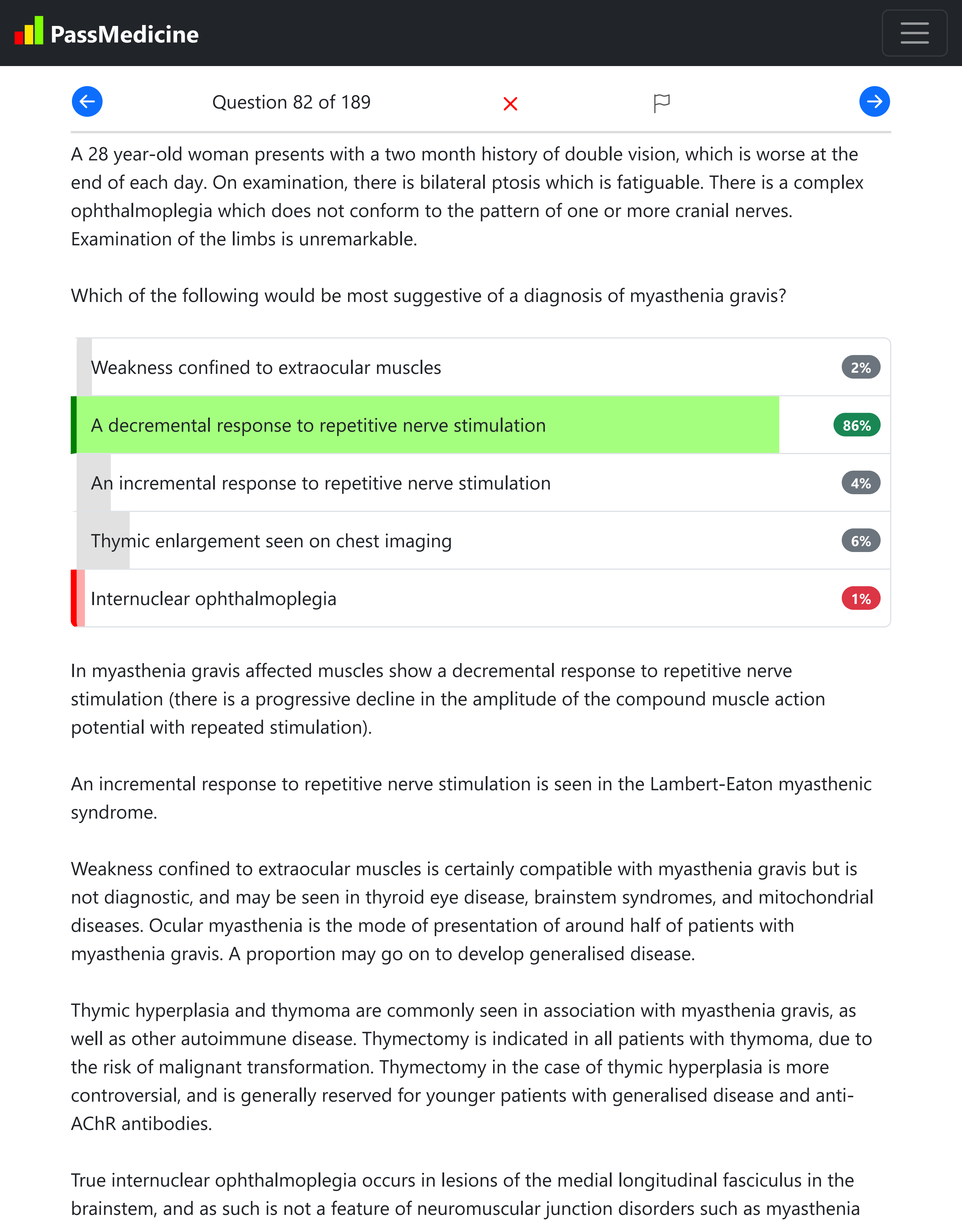

A 28 year-old woman presents with a two month history of double vision, which is worse at the end of each day. On examination, there is bilateral ptosis which is fatiguable. There is a complex ophthalmoplegia which does not conform to the pattern of one or more cranial nerves. Examination of the limbs is unremarkable.

Which of the following would be most suggestive of a diagnosis of myasthenia gravis?

Weakness confined to extraocular muscles	2%
A decremental response to repetitive nerve stimulation	86%
An incremental response to repetitive nerve stimulation	4%
Thymic enlargement seen on chest imaging	6%
Internuclear ophthalmoplegia	1%

In myasthenia gravis affected muscles show a decremental response to repetitive nerve stimulation (there is a progressive decline in the amplitude of the compound muscle action potential with repeated stimulation).

An incremental response to repetitive nerve stimulation is seen in the Lambert-Eaton myasthenic syndrome.

Weakness confined to extraocular muscles is certainly compatible with myasthenia gravis but is not diagnostic, and may be seen in thyroid eye disease, brainstem syndromes, and mitochondrial diseases. Ocular myasthenia is the mode of presentation of around half of patients with myasthenia gravis. A proportion may go on to develop generalised disease.

Thymic hyperplasia and thymoma are commonly seen in association with myasthenia gravis, as well as other autoimmune disease. Thymectomy is indicated in all patients with thymoma, due to the risk of malignant transformation. Thymectomy in the case of thymic hyperplasia is more controversial, and is generally reserved for younger patients with generalised disease and anti-AChR antibodies.

True internuclear ophthalmoplegia occurs in lesions of the medial longitudinal fasciculus in the brainstem, and as such is not a feature of neuromuscular junction disorders such as myasthenia gravis. However, the involvement of extraocular muscles (one or both medial recti) may on occasion mimic internuclear ophthalmoplegia.

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Next question >

Myasthenia gravis

Myasthenia gravis is an autoimmune disorder resulting in insufficient functioning acetylcholine receptors. Antibodies to acetylcholine receptors are seen in 85-90% of cases*. Myasthenia is more common in women (2:1)

The key feature is muscle fatigability - muscles become progressively weaker during periods of activity and slowly improve after periods of rest:

- extraocular muscle weakness: diplopia
- proximal muscle weakness: face, neck, limb girdle
- ptosis
- dysphagia

Associations

- thymomas in 15%
- autoimmune disorders: pernicious anaemia, autoimmune thyroid disorders, rheumatoid, SLE
- thymic hyperplasia in 50-70%

Investigations

- single fibre electromyography: high sensitivity (92-100%)
- CT thorax to exclude thymoma
- CK normal
- antibodies to acetylcholine receptors
    - positive in around 85-90% of patients
    - n the remaining patients, about about 40% are positive for anti-muscle-specific tyrosine kinase antibodies
- Tensilon test: IV edrophonium reduces muscle weakness temporarily - not commonly used any more due to the risk of cardiac arrhythmia

Management

- long-acting acetylcholinesterase inhibitors
    - pyridostigmine is first-line

- immunosuppression is usually not started at diagnosis, but the majority of patients eventually require it in addition to long-acting acetylcholinesterase inhibitors:
    - prednisolone initially
    - azathioprine, cyclosporine, mycophenolate mofetil may also be used
- thymectomy

Management of myasthenic crisis
- plasmapheresis
- intravenous immunoglobulins

*antibodies are less commonly seen in disease limited to the ocular muscles

+ Q 123

Next question >

B I

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal 👍 13 👎 3

Review of myasthenia gravis

Suggest link

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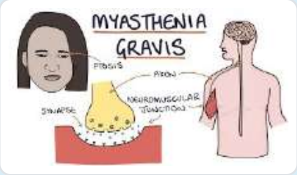
Media


MYASTHENIA GRAVIS
PTOSIS
AXON
NEUROMUSCULAR JUNCTION
SYNAPSE

Myasthenia gravis

Zero to Finals - YouTube 👍 12 👎 1

NEUROLOGY
MYASTHENIA GRAVIS

Myasthenia gravis

Armando Hasudungan - YouTube 👍 1 👎 1

MYASTHENIA GRAVIS

Myasthenia gravis - causes, symptoms, treatment, pathology

Osmosis - YouTube 👍 2 👎 2

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Score: **24.8%**

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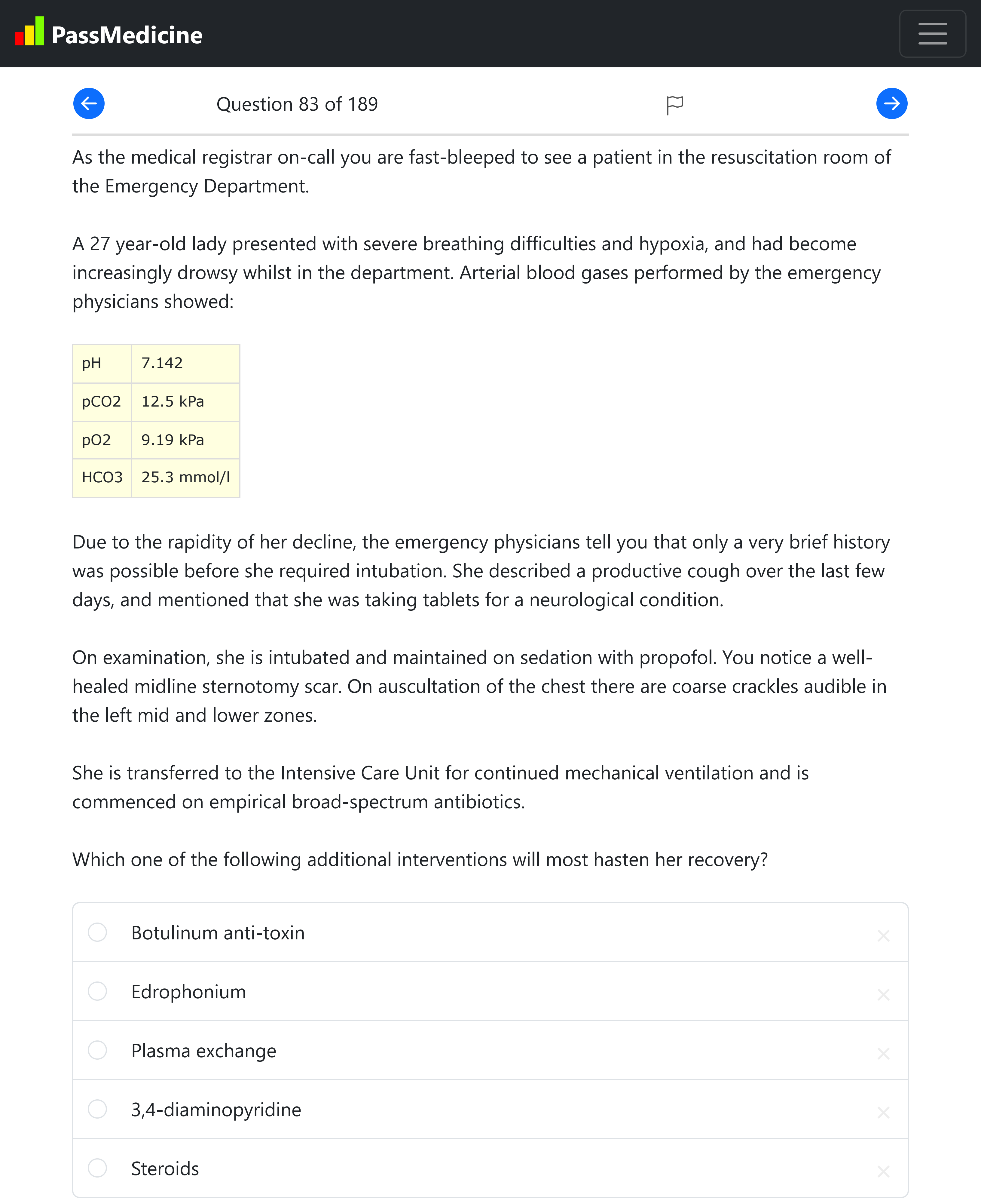

Question 83 of 189

As the medical registrar on-call you are fast-bleeped to see a patient in the resuscitation room of the Emergency Department.

A 27 year-old lady presented with severe breathing difficulties and hypoxia, and had become increasingly drowsy whilst in the department. Arterial blood gases performed by the emergency physicians showed:

pH	7.142
pCO2	12.5 kPa
pO2	9.19 kPa
HCO3	25.3 mmol/l

Due to the rapidity of her decline, the emergency physicians tell you that only a very brief history was possible before she required intubation. She described a productive cough over the last few days, and mentioned that she was taking tablets for a neurological condition.

On examination, she is intubated and maintained on sedation with propofol. You notice a well-healed midline sternotomy scar. On auscultation of the chest there are coarse crackles audible in the left mid and lower zones.

She is transferred to the Intensive Care Unit for continued mechanical ventilation and is commenced on empirical broad-spectrum antibiotics.

Which one of the following additional interventions will most hasten her recovery?

○ Botulinum anti-toxin ×

○ Edrophonium ×

○ Plasma exchange ×

○ 3,4-diaminopyridine ×

○ Steroids ×

Submit answer

Reference ranges ⌵

Score: **28%**

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She is transferred to the Intensive Care Unit for continued mechanical ventilation and is commenced on empirical broad-spectrum antibiotics.

Which one of the following additional interventions will most hasten her recovery?

Botulinum anti-toxin	4%
Edrophonium	7%
🟩 Plasma exchange	69%
3,4-diaminopyridine	6%
🟥 Steroids	14%

This lady has myasthenia gravis and is taking pyridostigmine tablets for symptomatic relief. She has had a thymectomy in the past.

The acute presentation is of myasthenic crisis, with dramatically deteriorating weakness progressing to respiratory failure. Crisis is typically precipitated by infection (in this case pneumonia), or non-compliance with medication.

The initial priority is ventilatory support. Falling oxygen saturations and PaO2 are late indicators of ventilatory compromise. A rising PaCO2 is a more sensitive marker. Although inappropriate in this case, forced vital capacity should be measured and generally if below 1.5L ventilatory support should be considered.

In considering antibiotic cover, gentamicin should be avoided as it may interfere with neuromuscular transmission. Similarly, the anaesthetists should be warned that the patient will be extremely sensitive to small doses of non-depolarising muscle relaxants such as atracurium and vecuronium, which are commonly used for rapid sequence induction in the emergency setting.

Plasmapheresis and intravenous immunoglobulin are used to provide a rapid disease-modifying effect in myasthenic crisis. In plasma exchange, circulating anti-acetylcholine receptor antibodies are removed, relieving neuromuscular blockade and improving weakness. The major side effects are worsening of sepsis, due to removal of protective antibodies, and coagulopathy due to removal of clotting factors and destruction of platelets.

Botulinum anti-toxin is a specific treatment for botulism.

Edrophonium (Tensilon) is a short-acting acetylcholinesterase inhibitor which is used in the diagnosis of myasthenia gravis.

3,4-diaminopyridine is a specific treatment for the Lambert-Eaton myasthenic syndrome.

Steroids and other immunopressive agents may be used for the treatment of myasthenia gravis but in this case plasma exchange is preferred as a rapid response is required.

Discuss (9) Improve

Next question

Myasthenia gravis

Myasthenia gravis is an autoimmune disorder resulting in insufficient functioning acetylcholine receptors. Antibodies to acetylcholine receptors are seen in 85-90% of cases*. Myasthenia is more common in women (2:1)

The key feature is muscle fatigability - muscles become progressively weaker during periods of activity and slowly improve after periods of rest:

- extraocular muscle weakness: diplopia
- proximal muscle weakness: face, neck, limb girdle
- ptosis
- dysphagia

Associations

- thymomas in 15%
- autoimmune disorders: pernicious anaemia, autoimmune thyroid disorders, rheumatoid, SLE
- thymic hyperplasia in 50-70%

Investigations

- single fibre electromyography: high sensitivity (92-100%)
- CT thorax to exclude thymoma
- CK normal
- antibodies to acetylcholine receptors
  - positive in around 85-90% of patients
  - n the remaining patients, about about 40% are positive for anti-muscle-specific tyrosine kinase antibodies
- Tensilon test: IV edrophonium reduces muscle weakness temporarily - not commonly used any more due to the risk of cardiac arrhythmia

Management

- long-acting acetylcholinesterase inhibitors
  - pyridostigmine is first-line
- immunosuppression is usually not started at diagnosis, but the majority of patients eventually require it in addition to long-acting acetylcholinesterase inhibitors:
  - prednisolone initially
  - azathioprine, cyclosporine, mycophenolate mofetil may also be used
- thymectomy

Management of myasthenic crisis

- plasmapheresis
- intravenous immunoglobulins

*antibodies are less commonly seen in disease limited to the ocular muscles

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal 👍 13 👎 3

Review of myasthenia gravis

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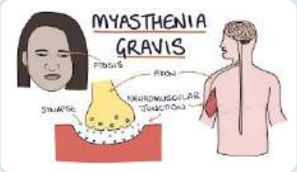
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Myasthenia gravis

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MYASTHENIA GRAVIS

Myasthenia gravis - causes, symptoms, treatment, pathology

Osmosis - YouTube 👍 2 👎 2

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A 46 year-old man is admitted to hospital after he is found wandering the streets, unable to remember who he his or how he got there. After a collateral history is obtained, he is referred to the neurologists for further investigation.

Over the last two months he has become increasingly forgetful and clumsy, and his behaviour has become progressively more bizarre. His speech has become confused, and he struggles with daily tasks. Others have commented that he has become very 'twitchy', and he sometimes drops things out of his hands without meaning to.

He was previously fit and well and the main carer for his father who suffers from motor neuron disease. His sister who previously helped him care for their father was recently killed in a car accident.

His only past medical history is that at age 22 he underwent extensive surgical resection of a large parafalcine meningioma which was discovered after investigation of chronic headache. Since the operation he has been free of headache.

On examination he is completely disoriented in time, place, and person. His gait is broad-based and uncoordinated, and during the examination you notice several shock-like jerks of his arms. Tone, power, reflexes, and sensation are otherwise normal, and cranial nerve examination is unremarkable.

What is the most likely diagnosis?

- ○ Viral encephalitis
- ○ Creutzfeld-Jakob disease
- ○ Frontotemporal dementia
- ○ Alzheimer's disease
- ○ Dissociative fugue

Submit answer

Reference ranges

Score: **28%**

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What is the most likely diagnosis?

Viral encephalitis	1%
Creutzfeld-Jakob disease	85%
Frontotemporal dementia	7%
Alzheimer's disease	1%
Dissociative fugue	5%

The history of rapidly progressive dementia, ataxia, and myoclonus is highly suggestive of Creutzfeld-Jakob disease (CJD). The previous neurosurgery may have required the use of a dural graft and if so this is the likely method of transmission of the prion protein (iatrogenic CJD).

Viral encephalitis is unlikely over a two month period.

Frontotemporal dementia causes a dementia with prominent personality change and behavioural disturbance, which may progress rapidly over months to years. There is an association with motor neuron disease.

Alzheimers disease would not typically occur at such an early age, or cause such a rapid deterioration. Ataxia would be unusual, and although myoclonus can occur in advanced dementia, this would not be typical early in the disease.

Dissociative fugue is a psychiatric condition characterised by disintegration of personality and memory, typically precipitated by a stressful event, and accompanied by wandering.

👍 👎 Discuss (7) Improve

Next question >

Creutzfeldt-Jakob disease ★

Creutzfeldt-Jakob disease (CJD) is rapidly progressive neurological condition caused by prion proteins. These proteins induce the formation of amyloid folds resulting in tightly packed beta-pleated sheets resistant to proteases.

Features
- dementia (rapid onset)
- myoclonus

Investigation
- CSF is usually normal
- EEG: biphasic, high amplitude sharp waves (only in sporadic CJD)
- MRI: hyperintense signals in the basal ganglia and thalamus

Sporadic CJD
- accounts for 85% of cases
- 10-15% of cases are familial
- mean age of onset is 65 years

New variant CJD

- younger patients (average age of onset = 25 years)
- psychological symptoms such as anxiety, withdrawal and dysphonia are the most common presenting features
- the 'prion protein' is encoded on chromosome 20 - it's role is not yet understood
- methionine homozygosity at codon 129 of the prion protein is a risk factor for developing CJD - all patients who have so far died have had this
- median survival = 13 months

Other prion diseases

- kuru
- fatal familial insomnia
- Gerstmann Straussler-Scheinker disease

+ Q 123

Next question >

B I

Textbooks

High-yield textbook

Extended textbook

Media

PRIONS
(SPONGIFORM
ENCEPHALOPATHY)

Prions (Spongiform encephalopathy)

Osmosis - YouTube 7 0

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Score: **24.8%**

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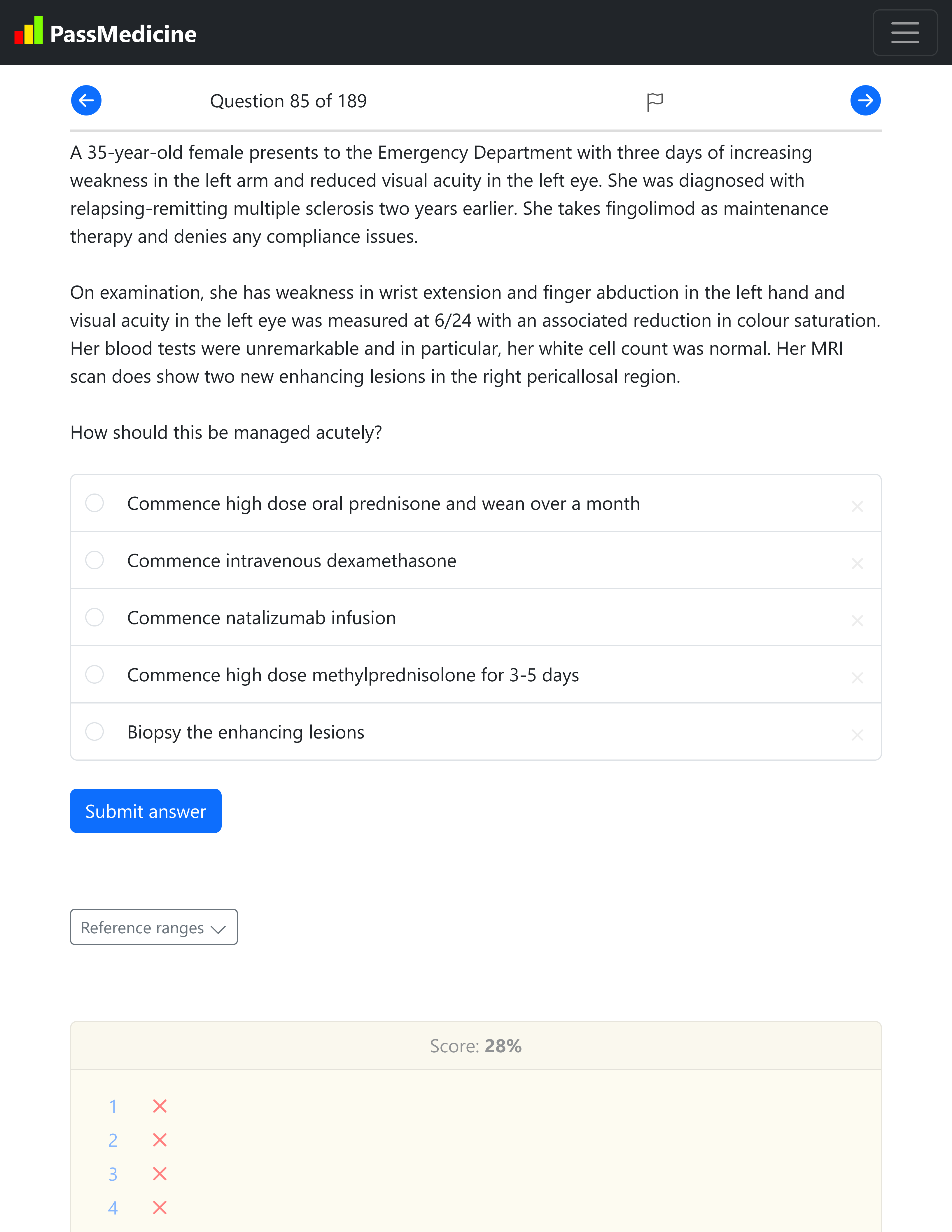

Question 85 of 189

A 35-year-old female presents to the Emergency Department with three days of increasing weakness in the left arm and reduced visual acuity in the left eye. She was diagnosed with relapsing-remitting multiple sclerosis two years earlier. She takes fingolimod as maintenance therapy and denies any compliance issues.

On examination, she has weakness in wrist extension and finger abduction in the left hand and visual acuity in the left eye was measured at 6/24 with an associated reduction in colour saturation. Her blood tests were unremarkable and in particular, her white cell count was normal. Her MRI scan does show two new enhancing lesions in the right pericallosal region.

How should this be managed acutely?

- ○ Commence high dose oral prednisone and wean over a month
- ○ Commence intravenous dexamethasone
- ○ Commence natalizumab infusion
- ○ Commence high dose methylprednisolone for 3-5 days
- ○ Biopsy the enhancing lesions

Score: **28%**

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How should this be managed acutely?

Commence high dose oral prednisone and wean over a month	8%
Commence intravenous dexamethasone	4%
Commence natalizumab infusion	8%
Commence high dose methylprednisolone for 3-5 days	78%
Biopsy the enhancing lesions	1%

Given that this patient has relapsed on maintenance therapy, and that she has multi-focal signs affecting vision and motor function, she should be admitted to hospital and commenced on high-dose intravenous methylprednisolone.

Acute relapses can be treated with either high dose oral prednisolone or intravenous therapy. The choice depends on multiple factors including the severity of symptoms, the requirement for hospital admission for monitoring, and co-morbidities such as diabetes and depression.

After this patient has recovered from this acute relapse a review of her maintenance therapy should occur, and this may be escalated through her neurologist based on a number of criteria.

Discuss (6) | Improve

Next question ›

Multiple sclerosis: management ★

Treatment in multiple sclerosis is focused on reducing the frequency and duration of relapses. There is no cure.

Acute relapse

High-dose steroids (e.g. oral or IV methylprednisolone) may be given for 5 days to shorten the length of an acute relapse. It should be noted that steroids shorten the duration of a relapse and do not alter the degree of recovery (i.e. whether a patient returns to baseline function)

Disease modifying drugs

A number of drugs have been shown to reduce the risk of relapse in patients with MS. Typical indications for disease-modifying drugs include:
- relapsing-remitting disease + 2 relapses in past 2 years + able to walk 100m unaided
- secondary progressive disease + 2 relapses in past 2 years + able to walk 10m (aided or unaided)

Drug options for reducing the risk of relapse in MS
- natalizumab
  - a recombinant monoclonal antibody that antagonises alpha-4 beta-1-integrin found on the surface of leucocytes
  - inhibit migration of leucocytes across the endothelium across the blood-brain barrier
  - generally considered to have the strongest evidence base for preventing relapse of the disease-modifying and hence is often used first-line
  - given intravenously
- ocrelizumab
  - humanized anti-CD20 monoclonal antibody
  - like natalizumab, it is considered a high-efficacy drug that is often used first-line
  - given intravenously
- fingolimod
  - sphingosine 1-phosphate (S1P) receptor modulator
  - prevents lymphocytes from leaving lymph nodes
  - oral formulations are available
- beta-interferon
  - not considered to be as effective as alternative disease-modifying drugs
  - given subcutaneously/intramuscularly
- glatiramer acetate

    - immunomodulating drug - acts as an 'immune decoy'
    - given subcutaneously
    - along with beta-interferon considered an 'older drug' with less effectiveness compared to monoclonal antibodies and S1P) receptor modulators

Some specific problems

Fatigue

- once other problems (e.g. anaemia, thyroid or depression) have been excluded NICE recommend a trial of amantadine
- other options include mindfulness training and CBT

Spasticity

- baclofen and gabapentin are first-line. Other options include diazepam, dantrolene and tizanidine
- physiotherapy is important
- cannabis and botox are undergoing evaluation

Bladder dysfunction

- may take the form of urgency, incontinence, overflow etc
- guidelines stress the importance of getting an ultrasound first to assess bladder emptying - anticholinergics may worsen symptoms in some patients
- if significant residual volume → intermittent self-catheterisation
- if no significant residual volume → anticholinergics may improve urinary frequency

Oscillopsia (visual fields appear to oscillate)

- gabapentin is first-line

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Multiple Sclerosis Society 👍 6 👎 15

Primary care guidelines

NICE 👍 15 👎 7

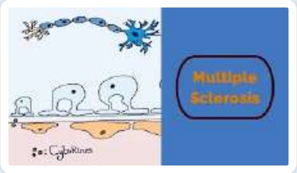
2014 Multiple Sclerosis guidelines

Suggest link Report broken link

Media

Multiple sclerosis

Podmedics - YouTube 👍 4 👎 0


Multiple Sclerosis

Multiple sclerosis

Soton Brain Hub - YouTube 👍 3 👎 0

DEMYELINATION
T-CELLS
PLAQUES
MULTIPLE SCLEROSIS
OLIGO CLONAL BANDS
RELAPSING-REMITTING

Multiple sclerosis

Rhesus Medicine - YouTube

👍 1 👎 0

MULTIPLE SCLEROSIS

Multiple sclerosis - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 7 👎 1

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Question 86 of 189

A 67-year-old male presents to the hospital with 2 hours of acute onset right sided weakness and speech difficulties.

A CT cerebral angiogram shows proximal left middle cerebral artery (LMCA) thrombosis.

Seven days prior to this presentation he underwent a laparotomy for bowel obstruction secondary to an incarcerated umbilical hernia. In the absence of IV thrombolysis which of the following emergency therapies may benefit the patient?

- ○ Intra-arterial clot retrieval
- ○ Prasugrel
- ○ Mannitol
- ○ Intravenous heparin infusion
- ○ Rosuvastatin

Submit answer

Reference ranges

Score: **28%**

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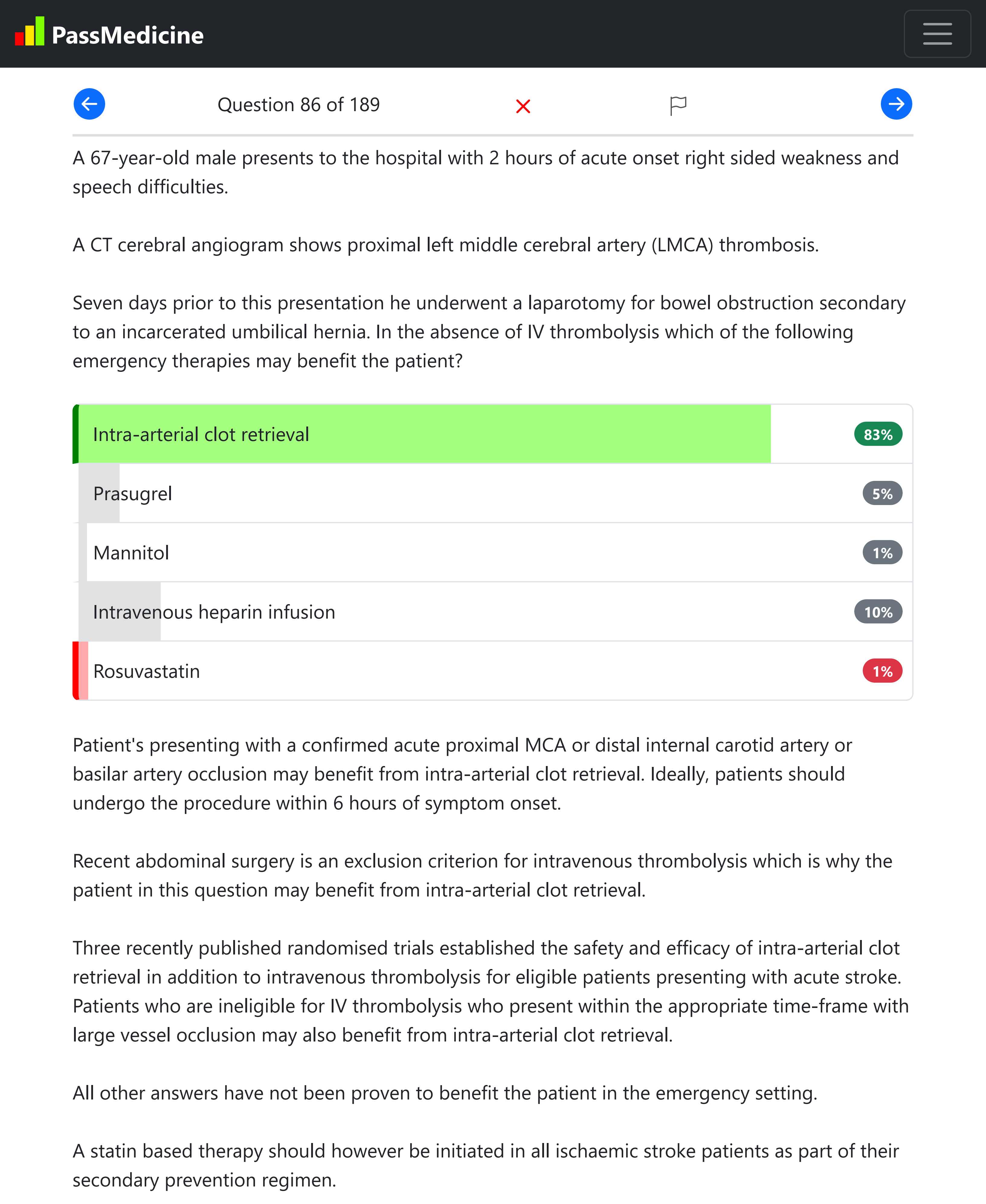


A 67-year-old male presents to the hospital with 2 hours of acute onset right sided weakness and speech difficulties.

A CT cerebral angiogram shows proximal left middle cerebral artery (LMCA) thrombosis.

Seven days prior to this presentation he underwent a laparotomy for bowel obstruction secondary to an incarcerated umbilical hernia. In the absence of IV thrombolysis which of the following emergency therapies may benefit the patient?

Intra-arterial clot retrieval	83%
Prasugrel	5%
Mannitol	1%
Intravenous heparin infusion	10%
Rosuvastatin	1%

Patient's presenting with a confirmed acute proximal MCA or distal internal carotid artery or basilar artery occlusion may benefit from intra-arterial clot retrieval. Ideally, patients should undergo the procedure within 6 hours of symptom onset.

Recent abdominal surgery is an exclusion criterion for intravenous thrombolysis which is why the patient in this question may benefit from intra-arterial clot retrieval.

Three recently published randomised trials established the safety and efficacy of intra-arterial clot retrieval in addition to intravenous thrombolysis for eligible patients presenting with acute stroke. Patients who are ineligible for IV thrombolysis who present within the appropriate time-frame with large vessel occlusion may also benefit from intra-arterial clot retrieval.

All other answers have not been proven to benefit the patient in the emergency setting.

A statin based therapy should however be initiated in all ischaemic stroke patients as part of their secondary prevention regimen.

👍 👎 Discuss (2) Improve

Next question ›

Stroke: management of acute ischaemic stroke ★

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:
- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
  - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
  - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:
- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.

- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
  - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
  - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 11 👎 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 15

Stroke and TIA guidelines

Age and Ageing 👍 20 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

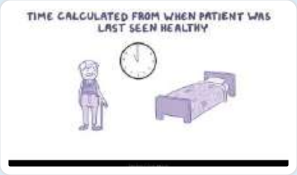
2023 National Clinical Guideline for Stroke

NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

[Stroke]

Osmosis - YouTube 👍 12 👎 0

Radiopaedia
Acute ischaemic Stroke

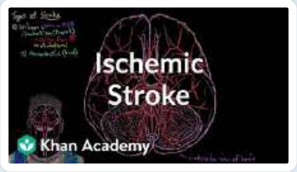
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic
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Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic
Strokes
Khan Academy

Haemorrhagic stroke

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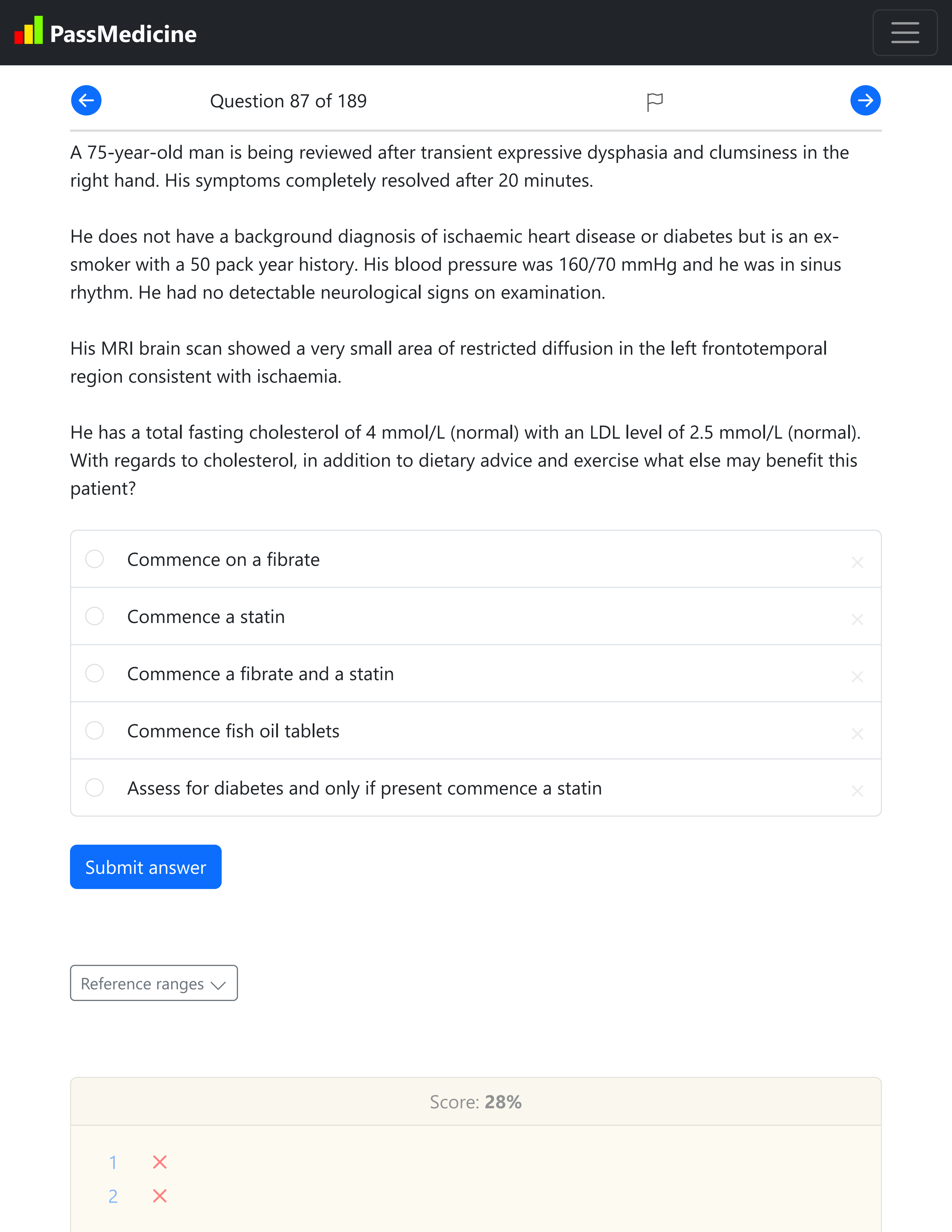

A 75-year-old man is being reviewed after transient expressive dysphasia and clumsiness in the right hand. His symptoms completely resolved after 20 minutes.

He does not have a background diagnosis of ischaemic heart disease or diabetes but is an ex-smoker with a 50 pack year history. His blood pressure was 160/70 mmHg and he was in sinus rhythm. He had no detectable neurological signs on examination.

His MRI brain scan showed a very small area of restricted diffusion in the left frontotemporal region consistent with ischaemia.

He has a total fasting cholesterol of 4 mmol/L (normal) with an LDL level of 2.5 mmol/L (normal). With regards to cholesterol, in addition to dietary advice and exercise what else may benefit this patient?

○ Commence on a fibrate

○ Commence a statin

○ Commence a fibrate and a statin

○ Commence fish oil tablets

○ Assess for diabetes and only if present commence a statin

Score: **28%**

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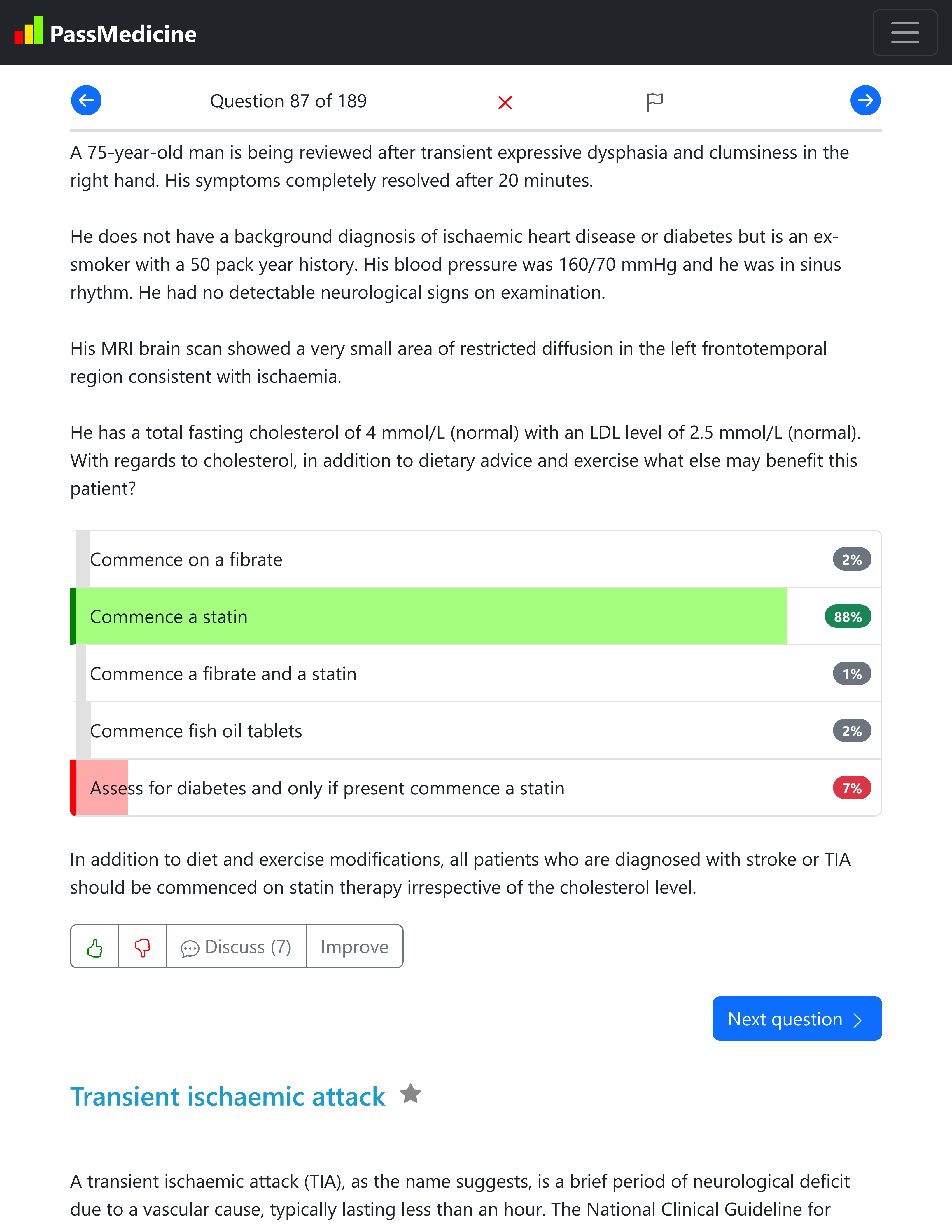

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His MRI brain scan showed a very small area of restricted diffusion in the left frontotemporal region consistent with ischaemia.

He has a total fasting cholesterol of 4 mmol/L (normal) with an LDL level of 2.5 mmol/L (normal). With regards to cholesterol, in addition to dietary advice and exercise what else may benefit this patient?

Commence on a fibrate	2%
Commence a statin	88%
Commence a fibrate and a statin	1%
Commence fish oil tablets	2%
Assess for diabetes and only if present commence a statin	7%

In addition to diet and exercise modifications, all patients who are diagnosed with stroke or TIA should be commenced on statin therapy irrespective of the cholesterol level.

Transient ischaemic attack

A transient ischaemic attack (TIA), as the name suggests, is a brief period of neurological deficit due to a vascular cause, typically lasting less than an hour. The National Clinical Guideline for

Stroke was published in 2023 and made a number of updated recommendations, including the use of dual antiplatelet therapy (DAPT). It should be noted that DAPT is not currently recommended for 'major' ischaemic stroke as the risk of haemorrhagic transformation is too high.

The original definition of a TIA was time-based: a sudden onset of a focal neurologic symptom and/or sign lasting less than 24 hours, brought on by a transient decrease in blood flow. However, this has now changed as it is recognised that even short periods of ischaemia can result in pathological changes to the brain. Therefore, a new **'tissue-based'** definition is now used: a transient episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischaemia, without acute infarction. REF

Patients often use the term 'mini-stroke' for TIAs.

Clinical features

The clinical features are similar to those of a stroke, i.e. sudden onset, focal neurological deficit but, rather than persisting, the features resolve, typically within 1 hour.

Possible features include
- unilateral weakness or sensory loss.
- aphasia or dysarthria
- ataxia, vertigo, or loss of balance
- visual problems
  - sudden transient loss of vision in one eye (amaurosis fugax)
  - diplopia
  - homonymous hemianopia

Assessment and referral

Patients with acute focal neurological symptoms that resolve completely within 24 hours of onset (i.e. suspected TIA) should:
- be given aspirin 300 mg immediately unless contraindicated
- assessed urgently within 24 hours by a stroke specialist clinician

Examples of TIA mimics that require exclusion
- hypoglycaemia
- intracranial haemorrhage
  - all patients on anticoagulants or with similar risk factors should be admitted for urgent imaging to exclude haemorrhage

If a patient presents more than 7 days ago they should be seen by a stroke specialist clinician as soon as possible within 7 days.

The ABCD2 prognostic score has previously been used to risk stratify patients who present with a suspected TIA. However, data from studies have suggested it performs poorly and it is therefore no longer recommended.

Patients with suspected TIA should be assessed by a stroke specialist clinician before a decision on brain imaging NICE
- NICE recommend that CT brains should not be done '*unless there is clinical suspicion of an alternative diagnosis that CT could detect*'
  - an example exception would be when there is a concern about haemorrhage as the patient is taking anticoagulants
- MRI (including diffusion-weighted and blood-sensitive sequences) is preferred to determine the territory of ischaemia, or to detect haemorrhage or alternative pathologies
  - it should be done on the same day as the specialist assessment if possible

Management of TIA

It should be remembered that patients who've had a TIA are at high risk of further vascular events, particularly in the first few days. This section covers management following a diagnosis, i.e. after being seen by a stroke specialist clinician.

Medication

Immediate antithrombotic therapy: REF
- patients with TIA or minor ischaemic stroke should be given antiplatelet therapy provided there is neither a contraindication nor a high risk of bleeding
- for patients within 24 hours of onset of TIA or minor ischaemic stroke and with a low risk of bleeding, the following DAPT regimes should be considered:
  - clopidogrel (initial dose 300 mg followed by 75 mg od) + aspirin (initial dose 300 mg followed by 75 mg od for 21 days) followed by monotherapy with clopidogrel 75 mg od
  - ticagrelor + clopidogrel is an alternative
- if not appropriate for DAPT:
  - clopidogrel 300 mg loading dose followed by 75 mg od should be given
- proton pump inhibitor therapy should be considered for DAPT

The different antithrombotic recommendations may seem confusing - it is helpful to think of 3 different phases for a typical patient:

Resolved TIA symptoms, awaiting specialist review within 24 hours	Reviewed by specialist, initial 21 days when at high risk of further events	Long-term secondary prevention after 21 days
Aspirin	Aspirin + Clopidogrel	Clopidogrel

If a patient has atrial fibrillation they should be anticoagulated as soon as intracranial haemorrhage has been excluded.

Lipid modification
- high-intensity statin (such as atorvastatin 20-80 mg daily) - the aim of statin therapy is to reduce non-HDL cholesterol by more than 40%

Further investigation

Carotid imaging
- atherosclerosis in the carotid artery may be a source of emboli in some patients
- patients who are considered candidates for carotid intervention should have carotid imaging performed within 24 hours of assessment
    - carotid duplex ultrasound or either CT angiography or MR angiography
- carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled NICE
    - should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

+ it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%
- it should be performed as soon as possible within 7 days

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

RCP 👍 13 👎 14

2016 Stroke guidelines

National Clinical Guideline for Stroke 👍 7 👎 3

2023 National Clinical Guideline for Stroke

NICE 👍 17 👎 6

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Clinical Knowledge Summaries 👍 8 👎 3

Transient ischaemic attack

Radiopaedia 👍 7 👎 1

Transient ischaemic attack

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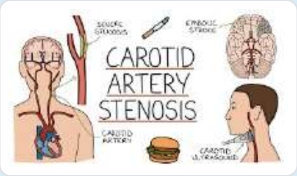
Media



Understanding Carotid Artery Stenosis

Zero To Finals - YouTube 👍 2 👎 1

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Question 88 of 189

A 63-year-old man presents to the emergency department with a 5-minute episode of slurred speech earlier in the day. His wife noticed that his face was drooping to one side as well. He had no arm weakness and is now completely back to normal. He is normally well and on no regular medication and is not allergic to any medication. He works as a plumber and smokes 10 cigarettes per day for the last 40 years and drinks alcohol socially. On further questioning he mentions that he had a similar episode also lasting 5 minutes four days ago whilst at work. On examination, his blood pressure is 135/70 mmHg and his heart rate is 58/min. He has no focal neurology and his cardiovascular and respiratory examinations are unremarkable. He has been given 300mg of Aspirin by the paramedics.

His blood tests are as follows:

Hb	138 g/l
Platelets	283 * 10^9/l
WBC	8.1 * 10^9/l
INR	1.1
PT	13 seconds

Na^+	142 mmol/l
K^+	4.4 mmol/l
Urea	6.4 mmol/l
Creatinine	89 µmol/l
CRP	5 mg/l
Total cholesterol	3.8 mmol/l
HDL	1.3 mmol/l

His ECG shows normal sinus rhythm and rate of 65/min.

What is the most appropriate management for this patient?

○ Outpatient TIA clinic appointment

○ Outpatient CT head and carotid dopplers within the next week

○ Admit for urgent (< 24 hours) CT head & carotid dopplers ×

○ Thrombolysis ×

○ Long term clopidogrel ×

Submit answer

Reference ranges ⌄

Score: **28%**

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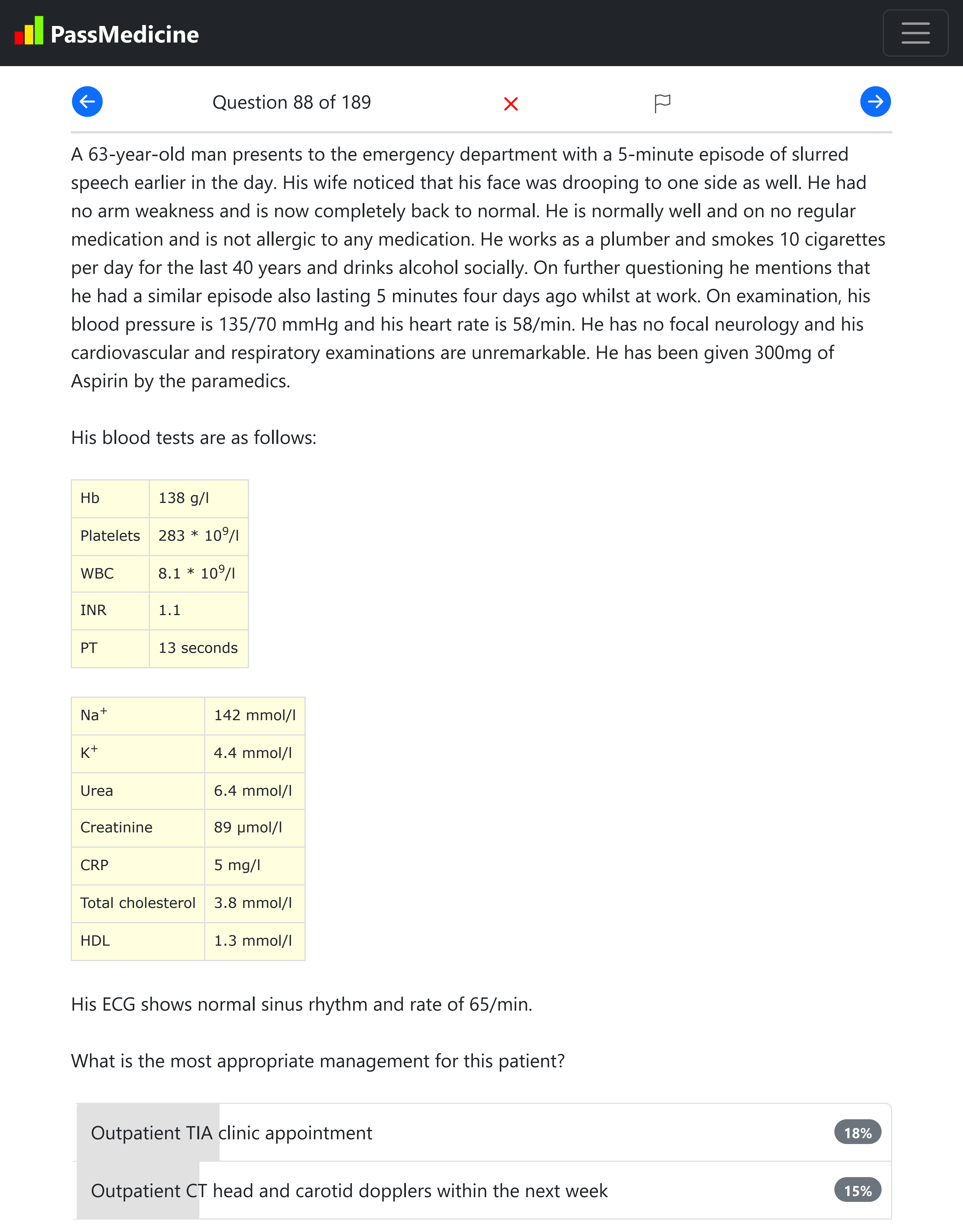

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His ECG shows normal sinus rhythm and rate of 65/min.

What is the most appropriate management for this patient?

Outpatient TIA clinic appointment 18%

Outpatient CT head and carotid dopplers within the next week 15%

Admit for urgent (< 24 hours) CT head & carotid dopplers 61%

Thrombolysis 1%

Long term clopidogrel 6%

This gentleman has had crescendo TIAs (2 in a 7 day period). This necessitates treatment as high risk i.e. admission and urgent (<24 hours) CT head and carotid dopplers. His ABCD score is 3 (1 for age and 2 for symptoms). This would normally put him in the low-risk category (see below) but the presence of crescendo episodes the ABCD score is irrelevant. Thrombolysis is not appropriate as the neurology has resolved. OP TIA clinic and imaging and dopplers within the week are not appropriate given the crescendo episodes.

👍 👎 💬 Discuss (4) Improve

Next question >

Transient ischaemic attack ★

A transient ischaemic attack (TIA), as the name suggests, is a brief period of neurological deficit due to a vascular cause, typically lasting less than an hour. The National Clinical Guideline for Stroke was published in 2023 and made a number of updated recommendations, including the use of dual antiplatelet therapy (DAPT). It should be noted that DAPT is not currently recommended for 'major' ischaemic stroke as the risk of haemorrhagic transformation is too high.

The original definition of a TIA was time-based: a sudden onset of a focal neurologic symptom and/or sign lasting less than 24 hours, brought on by a transient decrease in blood flow. However, this has now changed as it is recognised that even short periods of ischaemia can result in pathological changes to the brain. Therefore, a new '**tissue-based**' definition is now used: a transient episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischaemia, without acute infarction. REF

Patients often use the term 'mini-stroke' for TIAs.

Clinical features

The clinical features are similar to those of a stroke, i.e. sudden onset, focal neurological deficit but, rather than persisting, the features resolve, typically within 1 hour.

Possible features include

- unilateral weakness or sensory loss.
- aphasia or dysarthria
- ataxia, vertigo, or loss of balance
- visual problems
    - sudden transient loss of vision in one eye (amaurosis fugax)
    - diplopia
    - homonymous hemianopia

Assessment and referral

Patients with acute focal neurological symptoms that resolve completely within 24 hours of onset (i.e. suspected TIA) should:

- be given aspirin 300 mg immediately unless contraindicated
- assessed urgently within 24 hours by a stroke specialist clinician

Examples of TIA mimics that require exclusion

- hypoglycaemia
- intracranial haemorrhage
    - all patients on anticoagulants or with similar risk factors should be admitted for urgent imaging to exclude haemorrhage

If a patient presents more than 7 days ago they should be seen by a stroke specialist clinician as soon as possible within 7 days.

The ABCD2 prognostic score has previously been used to risk stratify patients who present with a suspected TIA. However, data from studies have suggested it performs poorly and it is therefore no longer recommended.

Patients with suspected TIA should be assessed by a stroke specialist clinician before a decision on brain imaging NICE

- NICE recommend that CT brains should not be done '*unless there is clinical suspicion of an alternative diagnosis that CT could detect*'
    - an example exception would be when there is a concern about haemorrhage as the patient is taking anticoagulants
- MRI (including diffusion-weighted and blood-sensitive sequences) is preferred to determine the territory of ischaemia, or to detect haemorrhage or alternative pathologies
    - it should be done on the same day as the specialist assessment if possible

Management of TIA

It should be remembered that patients who've had a TIA are at high risk of further vascular events, particularly in the first few days. This section covers management following a diagnosis, i.e. after being seen by a stroke specialist clinician.

Medication

Immediate antithrombotic therapy: REF

- patients with TIA or minor ischaemic stroke should be given antiplatelet therapy provided there is neither a contraindication nor a high risk of bleeding
- for patients within 24 hours of onset of TIA or minor ischaemic stroke and with a low risk of bleeding, the following DAPT regimes should be considered:
    - clopidogrel (initial dose 300 mg followed by 75 mg od) + aspirin (initial dose 300 mg followed by 75 mg od for 21 days) followed by monotherapy with clopidogrel 75 mg od
    - ticagrelor + clopidogrel is an alternative
- if not appropriate for DAPT:
    - clopidogrel 300 mg loading dose followed by 75 mg od should be given
- proton pump inhibitor therapy should be considered for DAPT

The different antithrombotic recommendations may seem confusing - it is helpful to think of 3 different phases for a typical patient:

Resolved TIA symptoms, awaiting specialist review within 24 hours	Reviewed by specialist, initial 21 days when at high risk of further events	Long-term secondary prevention after 21 days
Aspirin	Aspirin + Clopidogrel	Clopidogrel

If a patient has atrial fibrillation they should be anticoagulated as soon as intracranial haemorrhage has been excluded.

Lipid modification

- high-intensity statin (such as atorvastatin 20-80 mg daily) - the aim of statin therapy is to reduce non-HDL cholesterol by more than 40%

Further investigation

Carotid imaging

- atherosclerosis in the carotid artery may be a source of emboli in some patients

- patients who are considered candidates for carotid intervention should have carotid imaging performed within 24 hours of assessment
  - carotid duplex ultrasound or either CT angiography or MR angiography
- carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled NICE
  - should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

+ it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

RCP 👍 13 👎 14

2016 Stroke guidelines

National Clinical Guideline for Stroke 👍 7 👎 3

2023 National Clinical Guideline for Stroke

NICE 👍 17 👎 6

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Clinical Knowledge Summaries 👍 8 👎 3

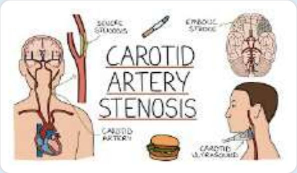
Transient ischaemic attack

Radiopaedia 👍 7 👎 1

Transient ischaemic attack

Suggest link Report broken link

Media


SEVERE STENOSIS
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CAROTID ARTERY STENOSIS
CAROTID ARTERY
CAROTID ULTRASOUND

Understanding Carotid Artery Stenosis

Zero To Finals - YouTube 👍 2 👎 1

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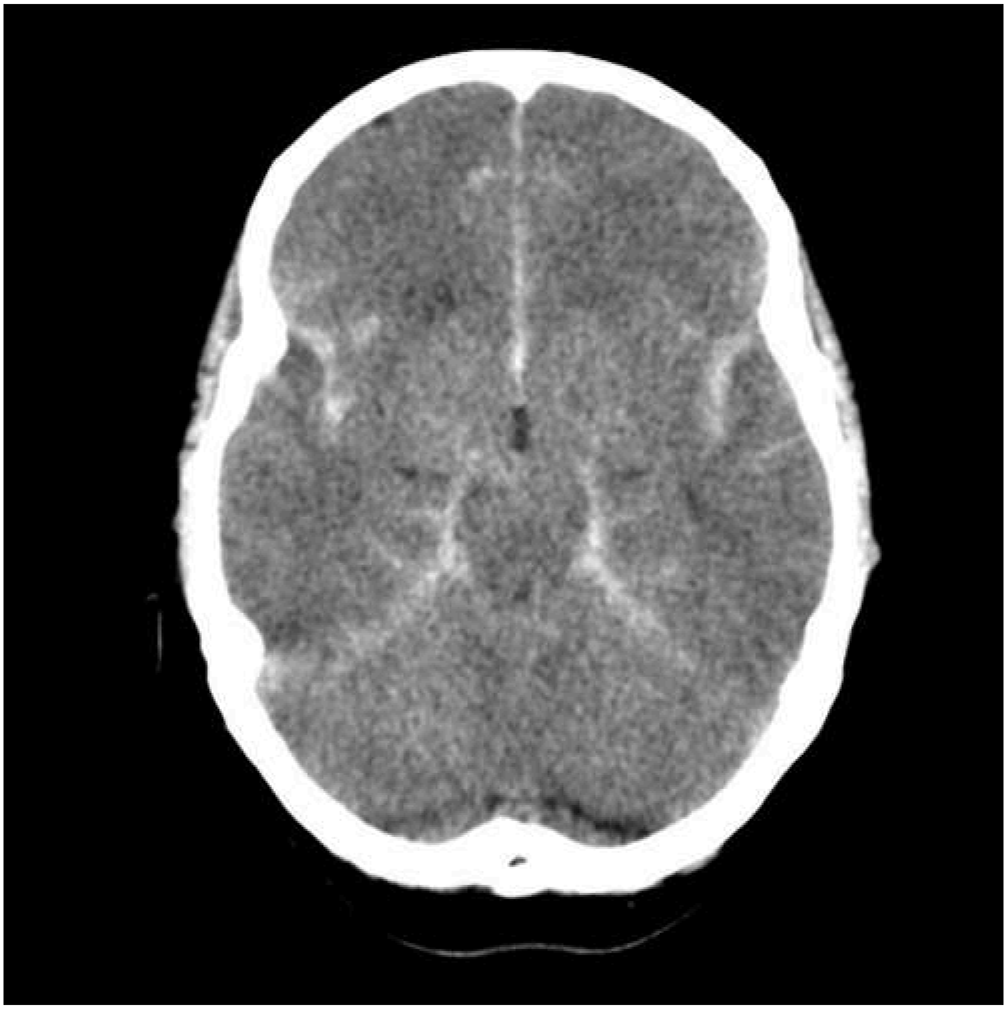
A 79-year-old woman is admitted to the Emergency Department via an ambulance after being found in an unresponsive, drowsy state. Her daughter phoned her earlier in the day routinely. At this point she complained of a headache. Upon arriving at her mother's house she found her confused and lying on the floor.

On examination her GCS is 9/15 (M4V3E2), pulse 96/min and blood pressure 140/78 mmHg.

A CT head is arranged:



© Image used on license from Radiopaedia

What is the most likely diagnosis?

○ Epidural haematoma

○ Subdural haematoma

○ Subarachnoid haemorrhage ×

○ Sagittal sinus thrombosis ×

○ Cavernous sinus thrombosis ×

Submit answer

Reference ranges ∨

Score: **28%**

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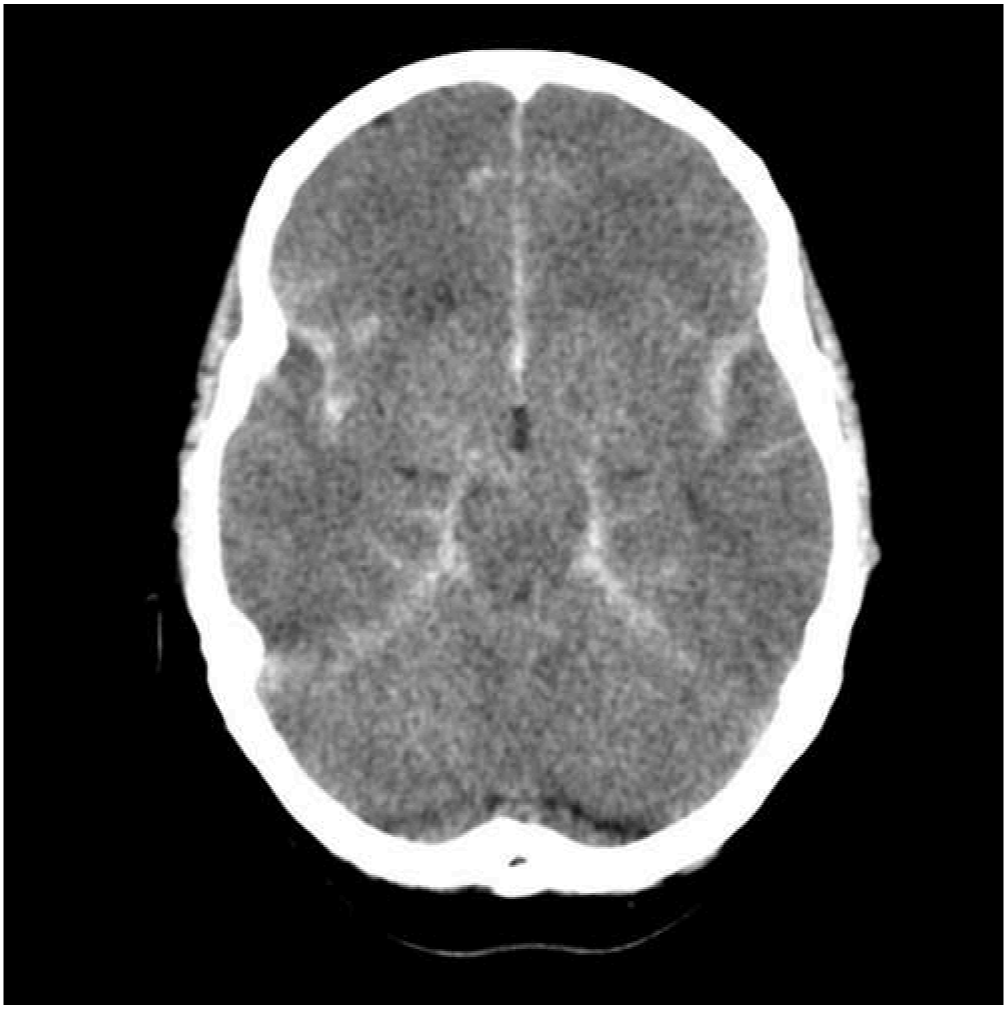

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What is the most likely diagnosis?

Epidural haematoma 1%

Subdural haematoma 2%

Subarachnoid haemorrhage	89%
Sagittal sinus thrombosis	4%
Cavernous sinus thrombosis	3%

The CT shows diffuse subarachnoid haemorrhage in all basal cisterns, bilateral sylvian fissures and the inter-hemispheric fissure. This case demonstrates the typical distribution that takes the blood into the subarachnoid space in a subarachnoid hemorrhage.

👍 👎 Discuss (3) Improve

Next question >

Subarachnoid haemorrhage ★

A subarachnoid haemorrhage (SAH) is an intracranial haemorrhage that is defined as the presence of blood within the subarachnoid space, i.e. deep to the subarachnoid layer of the meninges.

The most common cause of SAH is head injury and this is called *traumatic SAH* . In the absence of trauma, SAH is termed *spontaneous SAH* . The rest of this note focuses on spontaneous SAH.

Causes of spontaneous SAH include:
- intracranial aneurysm (saccular 'berry' aneurysms)
    - accounts for around 85% of cases
    - conditions associated with berry aneurysms include hypertension,adult polycystic kidney disease, Ehlers-Danlos syndrome and coarctation of the aorta
- arteriovenous malformation
- pituitary apoplexy
- mycotic (infective) aneurysms

Classical presenting features include:
- headache
    - usually of sudden-onset ('thunderclap' or 'hit with a baseball bat')
    - severe ('worst of my life')
    - occipital
    - typically peaking in intensity within 1 to 5 minutes
    - there may be a history of a less-severe 'sentinel' headache in the weeks prior to presentation

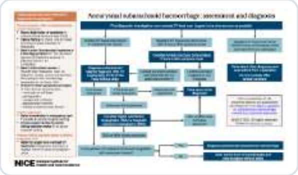
- nausea and vomiting
- meningism (photophobia, neck stiffness)
- coma
- seizures
- ECG changes including ST elevation may be seen
  - this may be secondary to either autonomic neural stimulation from the hypothalamus or elevated levels of circulating catecholamines

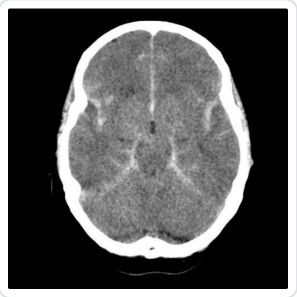
Investigation

- non-contrast CT head is the first-line investigation of choice
  - acute blood (hyperdense/bright on CT) is typically distributed in the basal cisterns, sulci and in severe cases the ventricular system.
- if CT head is done within 6 hours of symptom onset and is normal
  - new guidelines suggest **not** doing a lumbar puncture
  - consider an alternative diagnosis
- if CT head is done more than 6 hours after symptom onset and is normal
  - do a lumber puncture (LP)
  - timing wise the LP should be performed at least 12 hours following the onset of symptoms to allow the development of xanthochromia (the result of red blood cell breakdown).
  - xanthochromia helps to distinguish true SAH from a 'traumatic tap' (blood introduced by the LP procedure).
  - as well as xanthochromia, CSF findings consistent with subarachnoid haemorrhage include a normal or raised opening pressure
- if the CT shows evidence of a SAH
  - referral to neurosurgery to be made as soon as SAH is confirmed

After spontaneous SAH is confirmed, the aim of investigation is to identify a causative pathology that needs urgent treatment:

- CT intracranial angiogram (to identify a vascular lesion e.g. aneurysm or AVM)
- +/- digital subtraction angiogram (catheter angiogram)





Management of a confirmed aneurysmal subarachnoid haemorrhage

- supportive

    - bed rest
    - analgesia
    - venous thromboembolism prophylaxis
    - discontinuation of antithrombotics (reversal of anticoagulation if present)
- vasospasm is prevented using a course of oral nimodipine
- intracranial aneurysms are at risk of rebleeding and therefore require prompt intervention, preferably within 24 hours
    - most intracranial aneurysms are now treated with a coil by interventional neuroradiologists, but a minority require a craniotomy and clipping by a neurosurgeon

Complications of aneurysmal SAH:

- re-bleeding
    - happens in around 10% of cases and most common in the first 12 hours
    - if rebleeding is suspected (e.g. sudden worsening of neurological symptoms) then a repeat CT should be arranged
    - associated with a high mortality (up to 70%)
- hydrocephalus
    - hydrocephalus is temporarily treated with an external ventricular drain (CSF diverted into a bag at the bedside) or, if required, a long-term ventriculoperitoneal shunt
- vasospasm (also termed delayed cerebral ischaemia), typically 7-14 days after onset
    - ensure euvolaemia (normal blood volume)
    - consider treatment with a vasopressor if symptoms persist
- hyponatraemia (most typically due to syndrome inappropriate anti-diuretic hormone (SIADH))
- seizures

Important predictive factors in SAH:

- conscious level on admission
- age
- amount of blood visible on CT head

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia 👍 15 👎 15

Subarachnoid haemorrhage

Life in the Fast Lane 👍 19 👎 10

Example ECG changes in subarachnoid haemorrhage

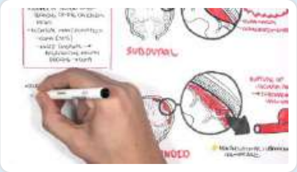
NICE 👍 9 👎 4

2022 Subarachnoid haemorrhage caused by a ruptured aneurysm: diagnosis and management

Suggest link

Report broken link

Media



Intracranial Haemorrhage Types

Armando Hasudungan - YouTube 👍 2 👎 0

Arteriovenous malformation

Osmosis - YouTube 👍 0 👎 0

NEUROLOGY
NEUROSURGERY
SUBARACHNOID
HAEMORRHAGE

Subarachnoid Haemorrhage / pathophysiology, complications and management

Armando Hasudungan - YouTube 👍 0 👎 0

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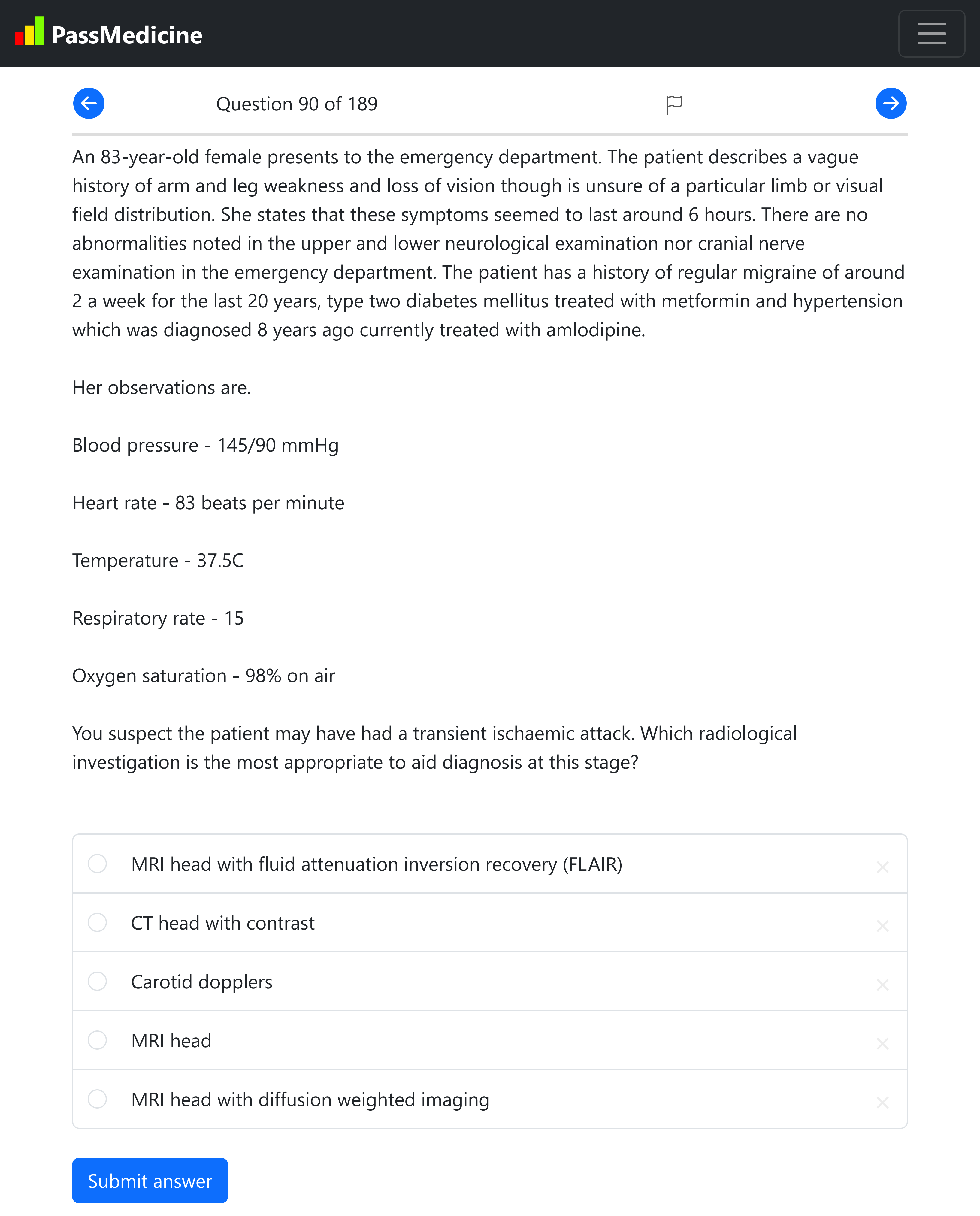

An 83-year-old female presents to the emergency department. The patient describes a vague history of arm and leg weakness and loss of vision though is unsure of a particular limb or visual field distribution. She states that these symptoms seemed to last around 6 hours. There are no abnormalities noted in the upper and lower neurological examination nor cranial nerve examination in the emergency department. The patient has a history of regular migraine of around 2 a week for the last 20 years, type two diabetes mellitus treated with metformin and hypertension which was diagnosed 8 years ago currently treated with amlodipine.

Her observations are.

Blood pressure - 145/90 mmHg

Heart rate - 83 beats per minute

Temperature - 37.5C

Respiratory rate - 15

Oxygen saturation - 98% on air

You suspect the patient may have had a transient ischaemic attack. Which radiological investigation is the most appropriate to aid diagnosis at this stage?

○ MRI head with fluid attenuation inversion recovery (FLAIR)

○ CT head with contrast

○ Carotid dopplers

○ MRI head

○ MRI head with diffusion weighted imaging

Reference ranges

Score: **28%**

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An 83-year-old female presents to the emergency department. The patient describes a vague history of arm and leg weakness and loss of vision though is unsure of a particular limb or visual field distribution. She states that these symptoms seemed to last around 6 hours. There are no abnormalities noted in the upper and lower neurological examination nor cranial nerve examination in the emergency department. The patient has a history of regular migraine of around 2 a week for the last 20 years, type two diabetes mellitus treated with metformin and hypertension which was diagnosed 8 years ago currently treated with amlodipine.

Her observations are.

Blood pressure - 145/90 mmHg

Heart rate - 83 beats per minute

Temperature - 37.5C

Respiratory rate - 15

Oxygen saturation - 98% on air

You suspect the patient may have had a transient ischaemic attack. Which radiological investigation is the most appropriate to aid diagnosis at this stage?

MRI head with fluid attenuation inversion recovery (FLAIR)	9%
CT head with contrast	5%
Carotid dopplers	14%
MRI head	5%
MRI head with diffusion weighted imaging	67%

This patient is an example of someone who has had a suspected transient ischaemic attack (TIA) who requires urgent brain imaging.

The national institute of clinical excellence (NICE) state that patients with a suspected TIA who are at a high risk of stroke (ABCD2 score of 4 or above) where the pathology or vascular territory is uncertain require urgent brain imaging, preferably diffusion weighted MRI.

The patient in this question has an ABCD2 score of at least 5, the vague clinical history also make the vascular territory uncertain. Therefore diffusion weighted MRI is most suitable. She may require a Doppler ultrasound of her carotid arteries in the future, though this would preferably happen after the MRI.

👍 👎 Discuss (5) Improve

Next question >

Transient ischaemic attack ★

A transient ischaemic attack (TIA), as the name suggests, is a brief period of neurological deficit due to a vascular cause, typically lasting less than an hour. The National Clinical Guideline for Stroke was published in 2023 and made a number of updated recommendations, including the use of dual antiplatelet therapy (DAPT). It should be noted that DAPT is not currently recommended for 'major' ischaemic stroke as the risk of haemorrhagic transformation is too high.

The original definition of a TIA was time-based: a sudden onset of a focal neurologic symptom and/or sign lasting less than 24 hours, brought on by a transient decrease in blood flow. However, this has now changed as it is recognised that even short periods of ischaemia can result in pathological changes to the brain. Therefore, a new '**tissue-based**' definition is now used: a transient episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischaemia, without acute infarction. REF

Patients often use the term 'mini-stroke' for TIAs.

Clinical features

The clinical features are similar to those of a stroke, i.e. sudden onset, focal neurological deficit but, rather than persisting, the features resolve, typically within 1 hour.

Possible features include
- unilateral weakness or sensory loss.
- aphasia or dysarthria
- ataxia, vertigo, or loss of balance
- visual problems

    - sudden transient loss of vision in one eye (amaurosis fugax)
    - diplopia
    - homonymous hemianopia

Assessment and referral

Patients with acute focal neurological symptoms that resolve completely within 24 hours of onset (i.e. suspected TIA) should:

- be given aspirin 300 mg immediately unless contraindicated
- assessed urgently within 24 hours by a stroke specialist clinician

Examples of TIA mimics that require exclusion

- hypoglycaemia
- intracranial haemorrhage
    - all patients on anticoagulants or with similar risk factors should be admitted for urgent imaging to exclude haemorrhage

If a patient presents more than 7 days ago they should be seen by a stroke specialist clinician as soon as possible within 7 days.

The ABCD2 prognostic score has previously been used to risk stratify patients who present with a suspected TIA. However, data from studies have suggested it performs poorly and it is therefore no longer recommended.

Patients with suspected TIA should be assessed by a stroke specialist clinician before a decision on brain imaging NICE

- NICE recommend that CT brains should not be done '*unless there is clinical suspicion of an alternative diagnosis that CT could detect*'
    - an example exception would be when there is a concern about haemorrhage as the patient is taking anticoagulants
- MRI (including diffusion-weighted and blood-sensitive sequences) is preferred to determine the territory of ischaemia, or to detect haemorrhage or alternative pathologies
    - it should be done on the same day as the specialist assessment if possible

Management of TIA

It should be remembered that patients who've had a TIA are at high risk of further vascular events, particularly in the first few days. This section covers management following a diagnosis, i.e. after being seen by a stroke specialist clinician.

Medication

Immediate antithrombotic therapy: REF
- patients with TIA or minor ischaemic stroke should be given antiplatelet therapy provided there is neither a contraindication nor a high risk of bleeding
- for patients within 24 hours of onset of TIA or minor ischaemic stroke and with a low risk of bleeding, the following DAPT regimes should be considered:
    - clopidogrel (initial dose 300 mg followed by 75 mg od) + aspirin (initial dose 300 mg followed by 75 mg od for 21 days) followed by monotherapy with clopidogrel 75 mg od
    - ticagrelor + clopidogrel is an alternative
- if not appropriate for DAPT:
    - clopidogrel 300 mg loading dose followed by 75 mg od should be given
- proton pump inhibitor therapy should be considered for DAPT

The different antithrombotic recommendations may seem confusing - it is helpful to think of 3 different phases for a typical patient:

Resolved TIA symptoms, awaiting specialist review within 24 hours	Reviewed by specialist, initial 21 days when at high risk of further events	Long-term secondary prevention after 21 days
Aspirin	Aspirin + Clopidogrel	Clopidogrel

If a patient has atrial fibrillation they should be anticoagulated as soon as intracranial haemorrhage has been excluded.

Lipid modification
- high-intensity statin (such as atorvastatin 20-80 mg daily) - the aim of statin therapy is to reduce non-HDL cholesterol by more than 40%

Further investigation

Carotid imaging
- atherosclerosis in the carotid artery may be a source of emboli in some patients
- patients who are considered candidates for carotid intervention should have carotid imaging performed within 24 hours of assessment
    - carotid duplex ultrasound or either CT angiography or MR angiography
- carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled NICE
    - should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

+ it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

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Next question >

Textbooks

High-yield textbook

Extended textbook

Links

RCP 👍 13 👎 14

2016 Stroke guidelines

National Clinical Guideline for Stroke 👍 7 👎 3

2023 National Clinical Guideline for Stroke

NICE 👍 17 👎 6

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Clinical Knowledge Summaries 👍 8 👎 3

Transient ischaemic attack

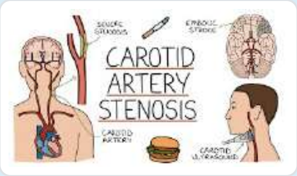
Radiopaedia

👍 7 👎 1

Transient ischaemic attack

Suggest link Report broken link

Media


CAROTID
ARTERY
STENOSIS

Understanding Carotid Artery Stenosis

Zero To Finals - YouTube 👍 2 👎 1

Report broken media

Score: **24.8%**

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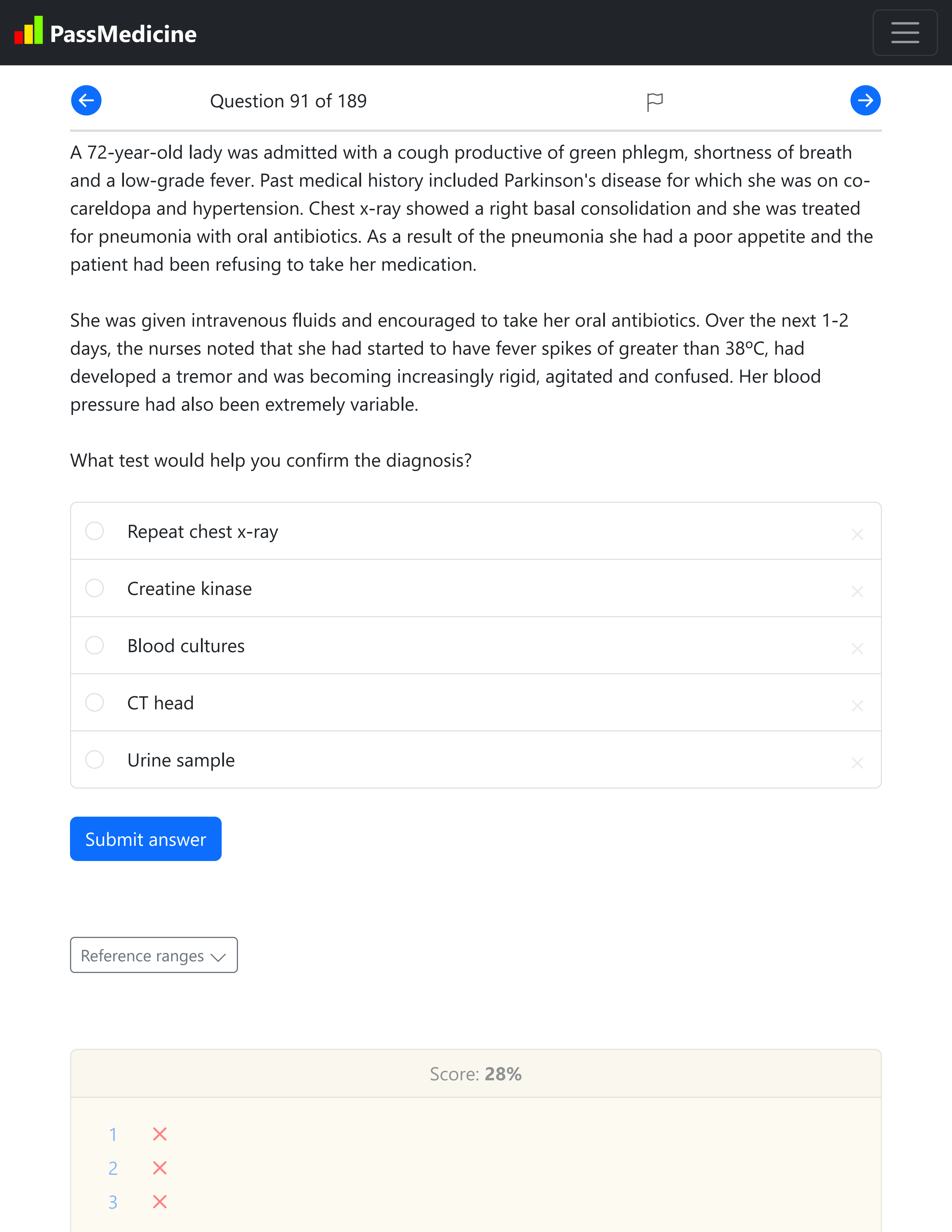

Question 91 of 189

A 72-year-old lady was admitted with a cough productive of green phlegm, shortness of breath and a low-grade fever. Past medical history included Parkinson's disease for which she was on co-careldopa and hypertension. Chest x-ray showed a right basal consolidation and she was treated for pneumonia with oral antibiotics. As a result of the pneumonia she had a poor appetite and the patient had been refusing to take her medication.

She was given intravenous fluids and encouraged to take her oral antibiotics. Over the next 1-2 days, the nurses noted that she had started to have fever spikes of greater than 38°C, had developed a tremor and was becoming increasingly rigid, agitated and confused. Her blood pressure had also been extremely variable.

What test would help you confirm the diagnosis?

- ○ Repeat chest x-ray
- ○ Creatine kinase
- ○ Blood cultures
- ○ CT head
- ○ Urine sample

Score: **28%**

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A 72-year-old lady was admitted with a cough productive of green phlegm, shortness of breath and a low-grade fever. Past medical history included Parkinson's disease for which she was on co-careldopa and hypertension. Chest x-ray showed a right basal consolidation and she was treated for pneumonia with oral antibiotics. As a result of the pneumonia she had a poor appetite and the patient had been refusing to take her medication.

She was given intravenous fluids and encouraged to take her oral antibiotics. Over the next 1-2 days, the nurses noted that she had started to have fever spikes of greater than 38ºC, had developed a tremor and was becoming increasingly rigid, agitated and confused. Her blood pressure had also been extremely variable.

What test would help you confirm the diagnosis?

Repeat chest x-ray	2%
Creatine kinase	83%
Blood cultures	5%
CT head	6%
Urine sample	4%

This patient has developed neuroleptic malignant syndrome (NMS) as a result of not taking her Parkinson's medication. NMS is a life-threatening neurological disorder most often caused by an adverse reaction to antipsychotic drugs (e.g. olanzapine, risperidone, aripiprazole, amisulpride, quetiapine, chlorpromazine, haloperidol and clozapine). However, a wide range of drugs can also cause NMS. These include dopaminergic medication i.e. levodopa and this often occurs when these medications are stopped abruptly, anti-dopaminergic medication such as metoclopramide and even drugs without anti-dopaminergic effects such as lithium can also cause NMS. NMS is caused by a sudden, reduction in dopamine activity, either from blockade of dopamine receptors or withdrawal of dopaminergic agents.

NMS should be suspected if a patient has muscle rigidity, muscle cramps, fever, autonomic instability and alterations in their mental status (confusion, agitation, coma). Symptoms can last anywhere from eight hours to forty days. These features are coupled with an elevated plasma creatine kinase (CK). A raised white cell count may occur and patients can develop rhabdomyolysis.

Metabolic acidosis and hypertensive crisis have also been reported.

Treatment is needed urgently as death can occur if untreated. Culprit medication should be stopped. Hyperthermia can be treated with fan therapy, cold blankets and ice packs. Dantrolene is the treatment of choice, however, amantadine and bromocriptine are also used. Patients may require supportive care in an intensive care unit to help support capable of ventilation and circulation.

👍 👎 Discuss (2) Improve

Next question >

Neuroleptic malignant syndrome

Neuroleptic malignant syndrome is a rare but dangerous condition seen in patients taking antipsychotic medication. It carries a mortality of up to 10% and can also occur with atypical antipsychotics. It may also occur with dopaminergic drugs (such as levodopa) for Parkinson's disease, usually when the drug is suddenly stopped or the dose reduced.

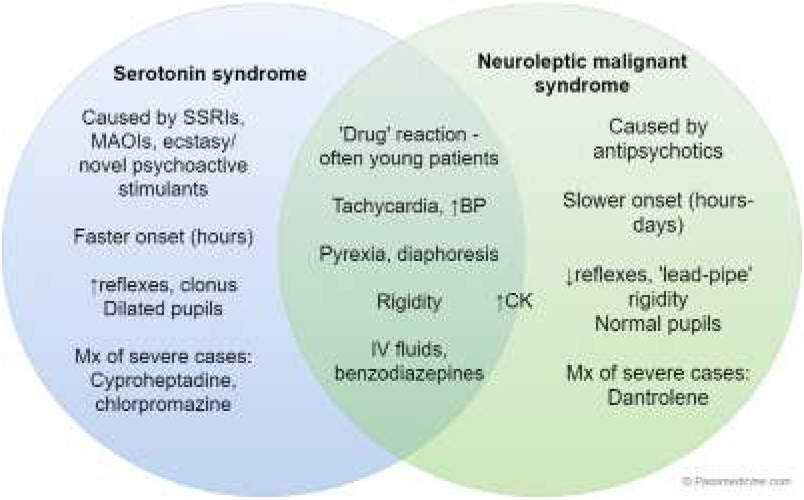
The pathophysiology is unknown but one theory is that the dopamine blockade induced by antipsychotics triggers massive glutamate release and subsequent neurotoxicity and muscle damage.

It occurs within hours to days of starting an antipsychotic (antipsychotics are also known as neuroleptics, hence the name) and the typical features are:
- pyrexia
- muscle rigidity
- autonomic lability: typical features include hypertension, tachycardia and tachypnoea
- agitated delirium with confusion

A raised creatine kinase is present in most cases. Acute kidney injury (secondary to rhabdomyolysis) may develop in severe cases. A leukocytosis may also be seen

Management
- stop antipsychotic
- patients should be transferred to a medical ward if they are on a psychiatric ward and often they are nursed in intensive care units
- IV fluids to prevent renal failure
- dantrolene may be useful in selected cases

    - thought to work by decreasing excitation-contraction coupling in skeletal muscle by binding to the ryanodine receptor, and decreasing the release of calcium from the sarcoplasmic reticulum
- bromocriptine, dopamine agonist, may also be used


Serotonin syndrome
Caused by SSRIs, MAOIs, ecstasy/ novel psychoactive stimulants
Faster onset (hours)
↑reflexes, clonus
Dilated pupils
Mx of severe cases: Cyproheptadine, chlorpromazine
'Drug' reaction - often young patients
Tachycardia, ↑BP
Pyrexia, diaphoresis
Rigidity
↑CK
IV fluids, benzodiazepines
Neuroleptic malignant syndrome
Caused by antipsychotics
Slower onset (hours-days)
↓reflexes, 'lead-pipe' rigidity
Normal pupils
Mx of severe cases: Dantrolene
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Venn diagram showing contrasting serotonin syndrome with neuroleptic malignant syndrome. Note that both conditions can cause a raised creatine kinase (CK) but it tends to be more associated with NMS.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 8 👎 5

Neuroleptic Malignant Syndrome

Medsafe 👍 15 👎 4

Neuroleptice malignant syndrome Vs serotonin syndrome

Suggest link

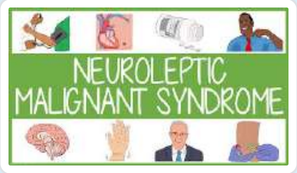
Report broken link

Media

DOPAMINE ANTAGONISM
AUTONOMIC INSTABILITY
NEUROLEPTIC MALIGNANT SYNDROME
FEVER
ANTIPSYCHOTICS

Neuroleptic malignant syndrome

Rhesus Medicine - YouTube 👍 1 👎 0


NEUROLEPTIC MALIGNANT SYNDROME

Neuroleptic malignant syndrome

Townsend Teaching - YouTube 👍 0 👎 0

Differential DX

Neuroleptic malignant syndrome

Kloss and Bruce - YouTube 👍 0 👎 1

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Score: **24.8%**

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A 56-year-old female was admitted to the Emergency Department with a 90-minute history of new onset weakness in her left arm and leg. She stated that she was out shopping when the weakness suddenly came on. Since the onset of the weakness she has noticed some regained strength but still felt definite weakness. She had a past medical history comprising new atrial fibrillation for which she was undergoing investigation, hypertension, hypercholesterolaemia and asthma. She also had a perforated gastric ulcer 16 years ago. She was prescribed amlodipine 5mg OD, bisoprolol 2.5mg OD, atorvastatin 20mg OD, Clenil modulite 200mcg BD and aspirin 75mg OD. She declined formal anticoagulation regarding her atrial fibrillation. When questioned specifically, she denied the presence of any visual loss, headache, vomiting, loss of consciousness or cardiac symptoms. She smoked 20 cigarettes per day and did not consume alcohol.

On examination she was alert and walking with a hemiplegic gait. Her blood pressure was 168/74 mmHg, heart rate 78bpm, respiratory rate of 18/min, temperature of 37.2 C and oxygen saturations of 99% on air. Other than an irregularly irregular pulse, examination of the cardiovascular, respiratory and gastrointestinal systems were unremarkable.

Examination of the central neurological system revealed normal cranial nerves 2-12, with equal and reactive pupils, normal fundoscopy and a GCS of 15. Examination of the peripheral nervous system revealed the presence of power 3/5 in all muscles of the left upper and lower limbs, with decreased tone and absent deep reflexes. Power was otherwise 5/5 in all other muscle groups, with downgoing plantar reflexes. Sensation and coordination testing was unremarkable.

Initial investigations revealed the following results:

Hb	179 g/l
MCV	99 fl
Platelets	452 * 10^9/l
WBC	12.2 * 10^9/l

ECG: atrial fibrillation 74 bpm no acute changes
CT head scan: no evidence of intracranial haemorrhage, mass shift or space occupying lesions

What is the next best management step?

○ Commence intravenous heparin ×

○ Commence thrombolysis therapy ×

○ Arrange urgent haematology consult ×

○ Commence aspirin 300mg and admit to stroke unit ×

○ Commence immediate amlodipine 5mg OD ×

Submit answer

Reference ranges

Score: **28%**

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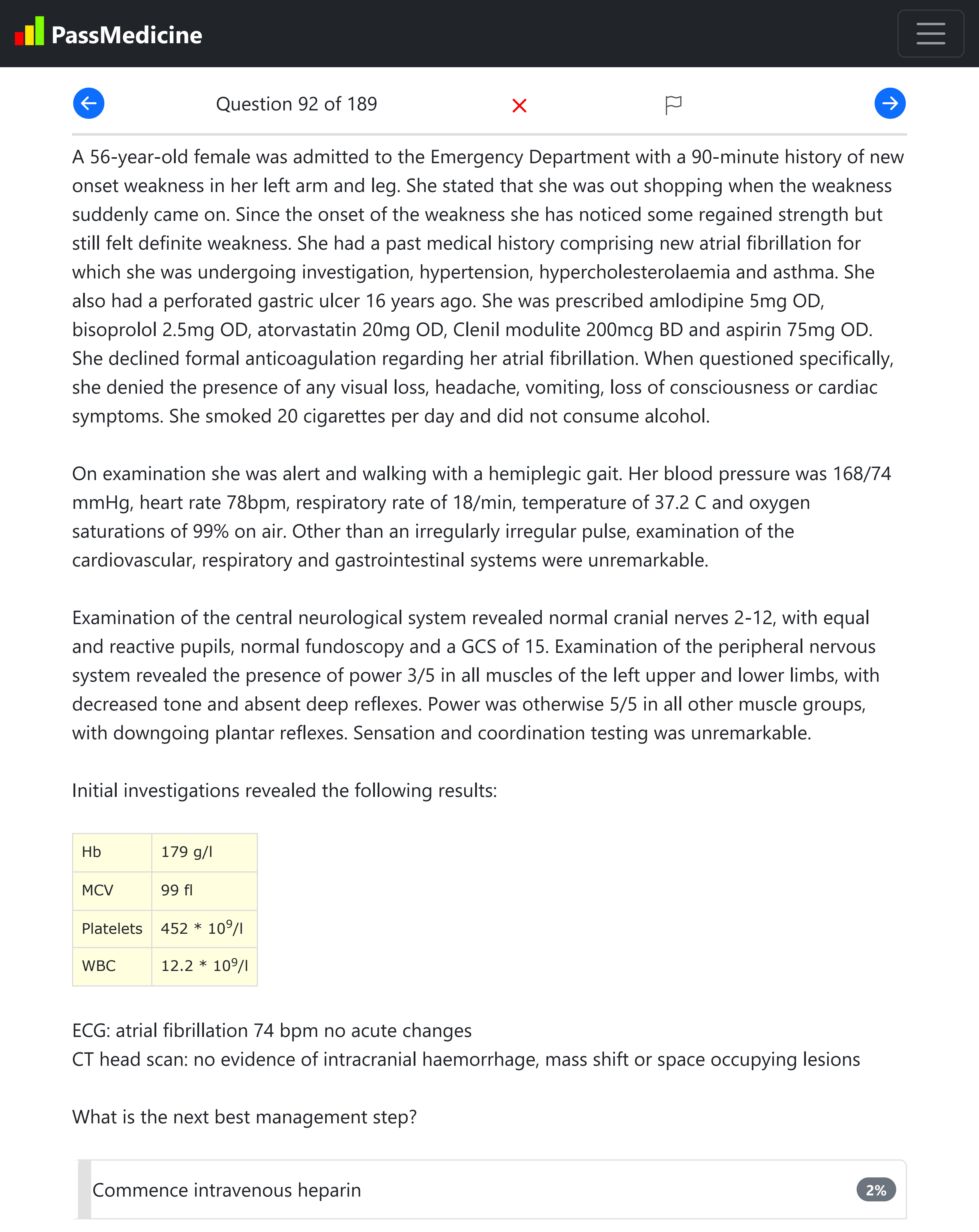

Question 92 of 189

A 56-year-old female was admitted to the Emergency Department with a 90-minute history of new onset weakness in her left arm and leg. She stated that she was out shopping when the weakness suddenly came on. Since the onset of the weakness she has noticed some regained strength but still felt definite weakness. She had a past medical history comprising new atrial fibrillation for which she was undergoing investigation, hypertension, hypercholesterolaemia and asthma. She also had a perforated gastric ulcer 16 years ago. She was prescribed amlodipine 5mg OD, bisoprolol 2.5mg OD, atorvastatin 20mg OD, Clenil modulite 200mcg BD and aspirin 75mg OD. She declined formal anticoagulation regarding her atrial fibrillation. When questioned specifically, she denied the presence of any visual loss, headache, vomiting, loss of consciousness or cardiac symptoms. She smoked 20 cigarettes per day and did not consume alcohol.

On examination she was alert and walking with a hemiplegic gait. Her blood pressure was 168/74 mmHg, heart rate 78bpm, respiratory rate of 18/min, temperature of 37.2 C and oxygen saturations of 99% on air. Other than an irregularly irregular pulse, examination of the cardiovascular, respiratory and gastrointestinal systems were unremarkable.

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Hb	179 g/l
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Platelets	452 * 10^9/l
WBC	12.2 * 10^9/l

ECG: atrial fibrillation 74 bpm no acute changes
CT head scan: no evidence of intracranial haemorrhage, mass shift or space occupying lesions

What is the next best management step?

Commence intravenous heparin 2%

Commence thrombolysis therapy	62%
Arrange urgent haematology consult	6%
Commence aspirin 300mg and admit to stroke unit	30%
Commence immediate amlodipine 5mg OD	1%

This lady has suffered an ischaemic stroke and fulfils the criteria for urgent thrombolysis. There are no absolute contraindications to thrombolysis and this will maximize the probability of regaining full function of the affected limbs. The other management options may serve a role but in the hyperacute setting of the above options initiating thrombolysis is the most appropriate management option.

👍 👎 Discuss (10) Improve

Next question ›

Stroke: management of acute ischaemic stroke ★

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
    - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
    - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'

- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:
- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.
- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
    - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
    - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

+ Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 11 👎 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 15

Stroke and TIA guidelines

Age and Ageing 👍 20 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

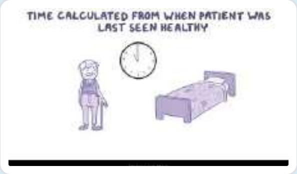
2023 National Clinical Guideline for Stroke

NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia
Acute ischaemic stroke

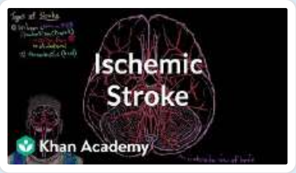
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic
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Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic
Strokes
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Haemorrhagic stroke

Khan Academy - YouTube 👍 1 👎 1

Report broken media

Score: **24.8%**

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Back to top

A 69-year-old retired accountant was seen in the neurology clinic having been referred by his GP. He presented with a three-month history of malaise and weakness. The weakness initially affected his legs, and at times he felt that his legs would not support him standing up. Over the course of the last month, however, he also noticed weakness in his hands, especially his left hand. This was particularly noticeable when he rode his motorbike when he felt that his fingers were not strong enough to apply the brakes. In addition, his wife remarked that his speech had changed in tone though he did not have any difficulties with understanding or expressing speech. His past medical history comprised hypertension, hypercholesterolaemia, depression and hypothyroidism. His medication regimen included amlodipine 5mg OD, atorvastatin 20mg ON, citalopram 20mg OD and levothyroxine 125mcg OD. He also remarked that he had been referred by his GP to the fast track gastroscopy clinic and was pending an appointment for new-onset difficulty in swallowing food over the last month. He did not smoke and did not consume alcohol. Upon a systems review, he denied the presence of other symptoms though he did admit to losing 3kg of weight since the onset of his symptoms.

On examination, he appeared cachectic with obvious dysarthria. His blood pressure was 128/78 mmHg and his heart rate was 78bpm. Examination of his cranial nerves revealed the presence of tongue fasciculations with tremulous lips and an absent jaw jerk reflex but was otherwise unremarkable. Examination of his peripheral nervous system revealed small muscle intrinsic wasting of both hands, as well as wasting of his thigh muscles. The tone was reduced in his lower limbs but increased in his upper limbs. He had obvious weakness of all muscle groups, with a grade 4/5 weakness in his lower limbs and proximal upper limb and a grade 3/5 weakness in his distal upper limbs. Fasciculations were present in his thighs and forearms. Both upper and lower limb reflexes were brisk with downgoing plantars. Examination of sensation was normal. Examination of the cardiovascular, respiratory and gastrointestinal systems was unremarkable.

Investigations conducted by the GP prior to referral are as follows:

Hb	111 g/l
Platelets	99 * 10^9/l
WBC	12.2 * 10^9/l
ESR	18 mm/hr
CRP	6 mg/l
ANA	positive
Rheumatoid factor	negative

Which one of the following interventions is most likely to be of benefit?

- ○ Mitoxantrone
- ○ Riluzole
- ○ Rivastigmine
- ○ Methotrexate
- ○ Azathioprine

Submit answer

Reference ranges

Score: **28%**

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WBC	12.2 * 10^9/l
ESR	18 mm/hr
CRP	6 mg/l
ANA	positive
Rheumatoid factor	negative

Which one of the following interventions is most likely to be of benefit?

Mitoxantrone	3%
Riluzole	86%
Rivastigmine	6%
Methotrexate	2%
Azathioprine	3%

This patient has motor neurone disease (MND), with clinical evidence of amyotrophic lateral sclerosis and bulbar palsy. Of the above options only riluzole, an anti-glutamate, has been shown to be of any benefit and is suitable for the amyotrophic lateral sclerosis variant of MND. The presence of ANA antibodies is a red herring; it is thought to be present in up to 15% of healthy individuals and is not associated with MND.

Discuss (4) | Improve

Next question ›

Motor neuron disease: management ★

Motor neuron disease is a neurological condition of unknown cause which can present with both upper and lower motor neuron signs. It rarely presents before 40 years and various patterns of disease are recognised including amyotrophic lateral sclerosis, progressive muscular atrophy and bulbar palsy

Riluzole
- prevents stimulation of glutamate receptors
- used mainly in amyotrophic lateral sclerosis
- prolongs life by about 3 months

Respiratory care
- non-invasive ventilation (usually BIPAP) is used at night
- studies have shown a survival benefit of around 7 months

Nutrition

- percutaneous gastrostomy tube (PEG) is the preferred way to support nutrition and has been associated with prolonged survival

Prognosis

- poor: 50% of patients die within 3 years

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 6 👎 5

2016 Motor neurone disease: assessment and management

Suggest link Report broken link

Media

MOTOR NEURONE DISEASE

Motor neuron disease

Zero To Finals - YouTube

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Report broken media

Score: **24.8%**

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101 ✓

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A 60-year-old male with a history of hypercholesterolaemia is brought to the emergency department with a 3-hour history of right sided body weakness and an inability to talk. The weakness was gradual in onset and has been progressing thereafter.

The patient has a history of peptic ulcer disease secondary to H.Pylori infection. He was treated with omeprazole, clarithromycin and amoxicillin-clavulanic acid. A recent upper GI endoscopy done 2 months ago was normal.

His medication includes omeprazole 20mg daily, atorvastatin 40mg daily and he takes paracetamol occasionally for joint pains.

On examination, he has a blood pressure of 140/80mmHg. He is aphasic, the tone on the right side is increased with the muscle power being 2/5 in the right upper limb proximally and distally while it is 1/5 in the lower limb both proximally and distally. There is no evidence of meningeal irritation and his pupils are normal.

Lab investigations reveal

Hb	150 g/l
Platelets	450 * 10^9/l
WBC	13.0 * 10^9/l

Na$^+$	138 mmol/l
K$^+$	4.4 mmol/l
Urea	6.9 mmol/l
Creatinine	118 µmol/l

ECG: Sinus tachycardia with occasional ventricular ectopics

CT scan brain: no evidence of cerebral haemorrhage, suspected left middle cerebral artery infarction.

What is the most appropriate management option for this patient?

○ Aspirin, clopidogrel, IV fluids and bowel and bladder care

- ○ Thrombolysis with alteplase ×
- ○ Modified release dipyridamole ×
- ○ Anticoagulation with LMWH ×
- ○ Anticoagulation with apixaban ×

Submit answer

Reference ranges ⌄

Score: **28%**

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What is the most appropriate management option for this patient?

Aspirin, clopidogrel, IV fluids and bowel and bladder care 6%

Thrombolysis with alteplase	91%
Modified release dipyridamole	1%
Anticoagulation with LMWH	1%
Anticoagulation with apixaban	1%

The patient has developed an ischaemic stroke and the question assesses the most appropriate management options. Evidence supports the role of thrombolysis in patients with a proven ischaemic stroke of less than 4.5 hours duration.

Guidelines are available at http://www.rcplondon.ac.uk/resources/stroke-guidelines

Eligibility for thrombolysis:

- Age >18 years
- Clinical diagnosis of acute ischaemic stroke
- Assessed by an experienced team
- Measurable neurological deficit
- Report of blood tests available
- CT/MRI consistent with acute ischaemic stroke
- Timing of onset well established
- Thrombolysis must commence as soon as possible within 4.5 hours of acute ischaemic stroke

Exclusion Criteria:

History

- Stroke or head trauma within the last 3 months
- Prior history of intracranial haemorrhage
- Major surgery within 14 days
- Gastrointestinal or genitourinary bleeding within 21 days
- MI in the last 3 months
- Lumbar puncture within the last 7 days
- Arterial puncture at non-compressible site in the last 7 days

Clinical

- Rapidly improving stroke syndrome
- Minor/isolated neurological deficit
- Seizure at the onset of stroke
- Symptoms suggestive of sub arachnoid haemorrhage even if imaging is normal
- Acute MI or post-MI pericarditis

- BP > 185 mmHg systolic or >110mmHg diastolic or aggressive therapy required to control BP
- Pregnancy or lactation
- Active bleeding or acute trauma

Laboratory
- Platelets <100,000/mm3
- Serum glucose <2.8 mmol/l or >21.2 mmol/l
- INR >1.7 if on warfarin
- Elevated APTT if on heparin

👍 👎 Discuss (8) Improve

Next question >

Stroke: management of acute ischaemic stroke

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:
- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
  - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
  - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

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- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
    - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
    - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
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Contraindications to thrombolysis

Absolute	**Relative**
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Absolute	Relative
- Oesophageal varices - Uncontrolled hypertension >200/120mmHg	

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

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Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 11 👎 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 15

Stroke and TIA guidelines

Age and Ageing 👍 20 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

2023 National Clinical Guideline for Stroke

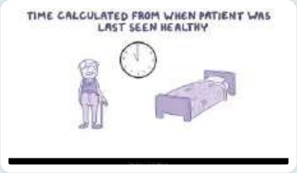
NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link

Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia.org
Acute ischaemic stroke

GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1

Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

Khan Academy - YouTube 👍 1 👎 1

Report broken media

Score: **24.8%**

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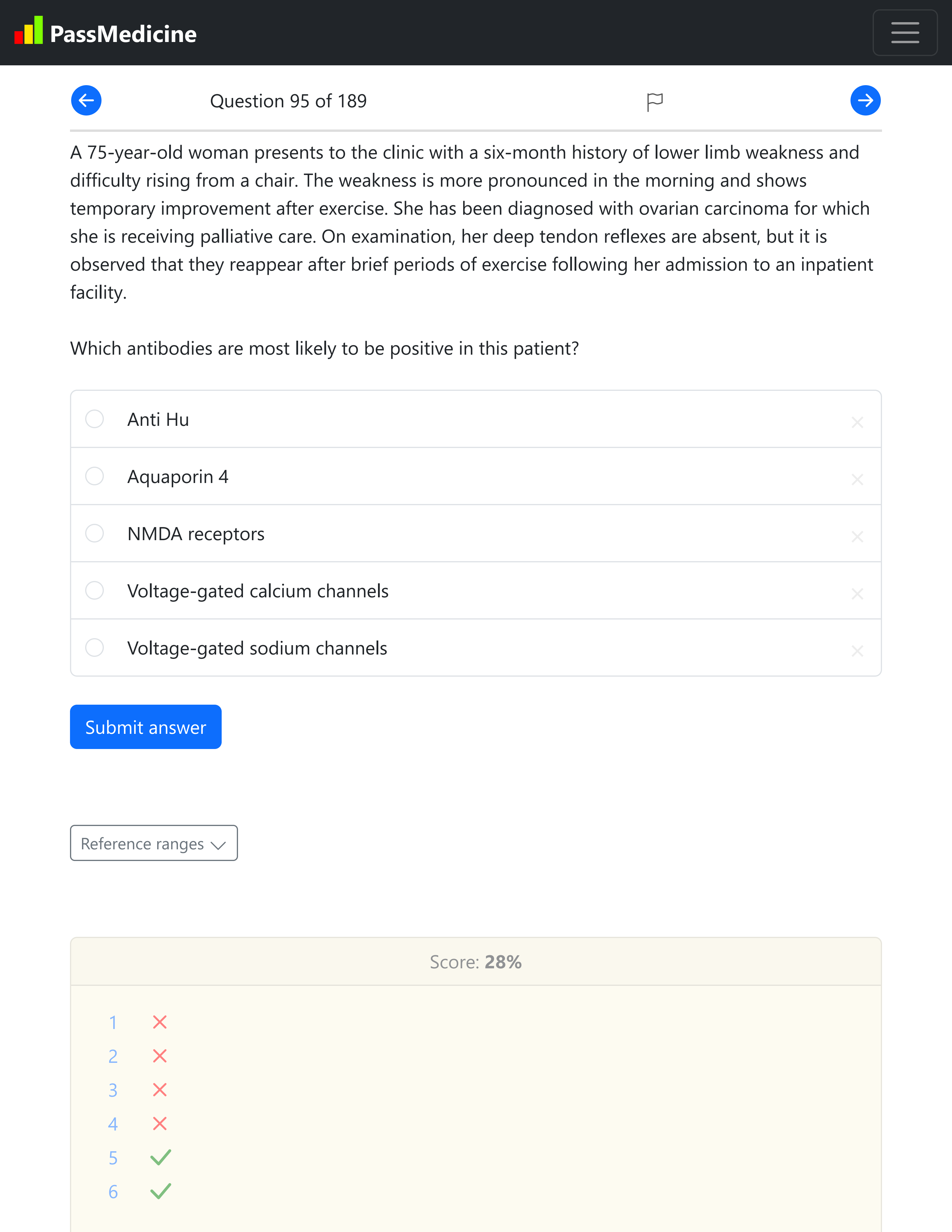

Question 95 of 189

A 75-year-old woman presents to the clinic with a six-month history of lower limb weakness and difficulty rising from a chair. The weakness is more pronounced in the morning and shows temporary improvement after exercise. She has been diagnosed with ovarian carcinoma for which she is receiving palliative care. On examination, her deep tendon reflexes are absent, but it is observed that they reappear after brief periods of exercise following her admission to an inpatient facility.

Which antibodies are most likely to be positive in this patient?

- ○ Anti Hu
- ○ Aquaporin 4
- ○ NMDA receptors
- ○ Voltage-gated calcium channels
- ○ Voltage-gated sodium channels

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Question 95 of 189

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Which antibodies are most likely to be positive in this patient?

Anti Hu	8%
Aquaporin 4	1%
NMDA receptors	14%
Voltage-gated calcium channels	71%
Voltage-gated sodium channels	6%

Lambert-Eaton syndrome - antibodies against the voltage-gated Ca2+ channels

Important for me Less important

Voltage-gated calcium channels is the correct answer. The clinical presentation of this patient suggests Lambert-Eaton myasthenic syndrome (LEMS), which is often associated with malignancies, most commonly small cell lung cancer and, in very rare instances, ovarian cancer as highlighted by a case report. The pathogenesis of LEMS involves autoantibody production against presynaptic voltage-gated calcium channels within the peripheral nervous system. This autoimmune response leads to reduced acetylcholine release and subsequent muscle weakness. A characteristic feature of LEMS is post-tetanic facilitation, an improvement in reflexes following exercise, which serves as a strong diagnostic indicator for the syndrome. The antibodies in question specifically target voltage-gated calcium channels, thereby disrupting normal calcium influx and diminishing acetylcholine secretion at nerve terminals.

The role of **voltage-gated sodium channels** is not relevant in this scenario since it is the calcium channels that are targeted by autoantibodies in cases of LEMS.

Although the patient's history of ovarian cancer might raise suspicion for **anti-NMDA receptor** encephalitis, this condition usually presents with acute psychiatric symptoms and behavioural changes that are inconsistent with the clinical presentation observed here; therefore, NMDA receptors are an incorrect choice. Moreover, post-exercise facilitation of deep tendon reflexes is not a feature of NMDA encephalitis.

Anti-Hu antibodies serve as markers for various paraneoplastic neurological syndromes such as limbic encephalopathy and sensory neuronopathy. These conditions do not correspond with the clinical findings discussed here. Anti-Hu antibodies are predominantly linked to small-cell lung cancer, with approximately 85% of positive cases being associated with this type of malignancy.

Lastly, neuromyelitis optica (NMO) is characterised by **aquaporin 4** antibodies; however, there are no clinical signs or symptoms indicative of NMO present in this patient's case. Therefore, selecting aquaporin 4 as an answer would be incorrect.

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Next question >

Lambert-Eaton syndrome ★

Lambert-Eaton myasthenic syndrome is seen in association with small cell lung cancer and to a lesser extent breast and ovarian cancer. It may also occur independently as an autoimmune disorder. Lambert-Eaton myasthenic syndrome is caused by an antibody directed against presynaptic voltage-gated calcium channel in the peripheral nervous system.

Features
- repeated muscle contractions lead to increased muscle strength (in contrast to myasthenia gravis)
  - in reality, this is seen in only 50% of patients and following prolonged muscle use muscle strength will eventually decrease
- limb-girdle weakness (affects lower limbs first)
- hyporeflexia
- autonomic symptoms: dry mouth, impotence, difficulty micturating
- ophthalmoplegia and ptosis not commonly a feature (unlike in myasthenia gravis)

EMG
- incremental response to repetitive electrical stimulation

Management
- treatment of underlying cancer

- immunosuppression, for example with prednisolone and/or azathioprine
- 3,4-diaminopyridine is currently being trialled
    - works by blocking potassium channel efflux in the nerve terminal so that the action potential duration is increased. Calcium channels can then be open for a longer time and allow greater acetylcholine release to the stimulate muscle at the end plate
- intravenous immunoglobulin therapy and plasma exchange may be beneficial

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Next question ›

Textbooks

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Media

NEUROLOGY
LAMBERT-EATON MYASTHENIC SYNDROME

Lambert-Eaton syndrome

Armando Hasudungan - YouTube 👍 1 👎 0

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Score: **24.8%**

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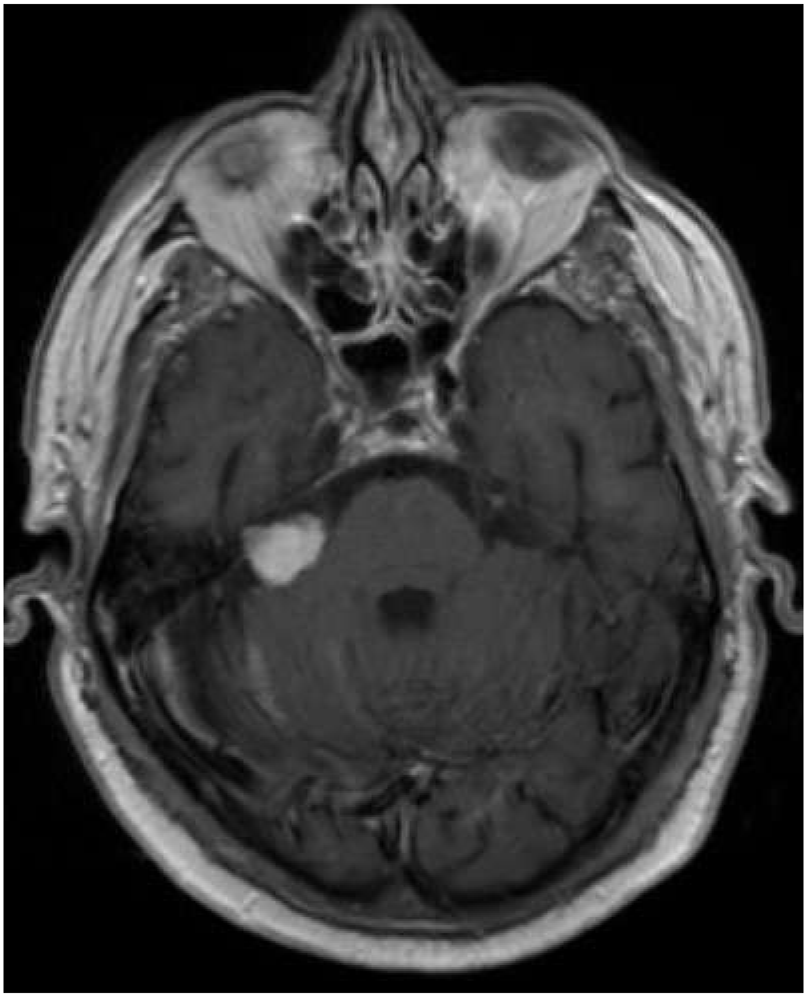
A 60-year-old schoolteacher is referred to the neurology clinic by his General Practitioner to assess his symptoms further. He has an MRI brain before review in the neurology clinic. The MRI brain showed:



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What is the most likely diagnosis?

- ○ Cavernous hemangioma
- ○ Facial nerve schwannoma
- ○ Infratentorial meningioma
- ○ Trigeminal schwannoma
- ○ Vestibular schwannoma

Submit answer

Reference ranges ⌵

Score: **28%**

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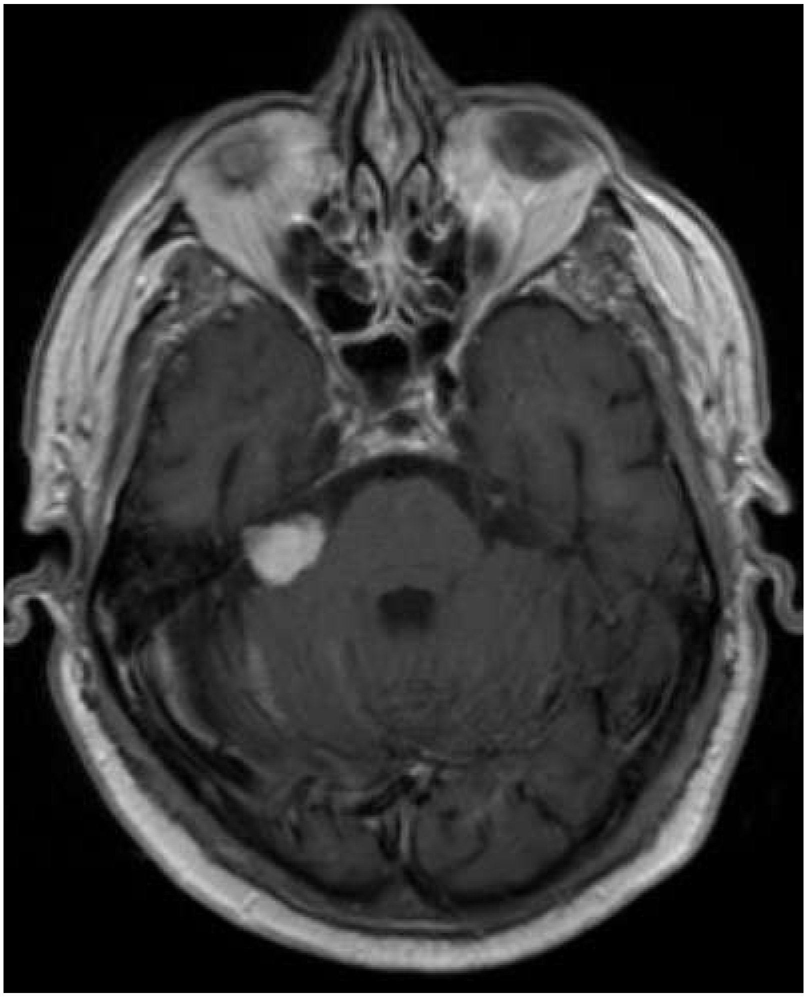
A 60-year-old schoolteacher is referred to the neurology clinic by his General Practitioner to assess his symptoms further. He has an MRI brain before review in the neurology clinic. The MRI brain showed:



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What is the most likely diagnosis?

Cavernous hemangioma	11%
Facial nerve schwannoma	2%
Infratentorial meningioma	15%
Trigeminal schwannoma	4%
Vestibular schwannoma	68%

The MRI brain shows an intracanalicular solid nodular mass with an intense contrast enhancement in the left internal auditory meatus (IAM) suggestive of vestibular schwannoma. Occasionally, cystic degeneration or haemorrhagic areas may also be seen, but calcification is typically not present.

Vestibular schwannoma is the correct answer. Vestibular schwannomas, or acoustic neuromas, are relatively common tumours arising from the vestibulocochlear nerve (CN VIII) and represent ~80% of cerebellopontine angle (CPA) masses. The classical presentation for vestibular schwannoma includes tinnitus, sensorineural hearing loss, vertigo and absent corneal reflex. Cranial nerves V and VII may also be affected.

Cavernous hemangiomas, also commonly known as cerebral cavernous venous malformations or cavernomas, are common cerebral vascular malformations, usually with characteristic appearances on MRI. MRI often demonstrates a characteristic 'popcorn' or 'berry' appearance with a rim of signal loss due to hemosiderin. The patients usually remain asymptomatic but may present with headaches and focal neurological deficits from intracranial haemorrhage.

Facial nerve schwannoma, or facial nerve neuroma, is a rare schwannoma that arises from the facial nerve. They are generally uncommon and make up less than 1% of all temporal bone tumours when involving the temporal bone.

Infratentorial meningiomas are rare even though meningiomas, in general, are the second most common cause of a cerebellopontine mass. They typically appear as extra-axial masses with a broad dural base and are usually homogeneous and well-circumscribed. Patients often present with headaches, paresis and changes in mental status.

Trigeminal schwannomas are uncommon slow-growing encapsulated tumours of Schwann cells. They are the second most common intracranial schwannoma, far less common than vestibular schwannoma, and have a predominantly benign growth.

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Next question >

Vestibular schwannoma (acoustic neuroma) ★

Vestibular schwannomas (sometimes referred to as acoustic neuromas) account for approximately 5% of intracranial tumours and 90% of cerebellopontine angle tumours.

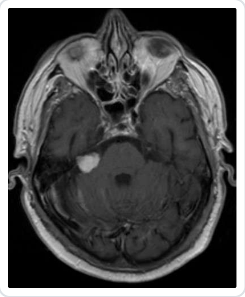
The classical history of vestibular schwannoma includes a combination of vertigo, hearing loss, tinnitus and an absent corneal reflex. Features can be predicted by the affected cranial nerves:

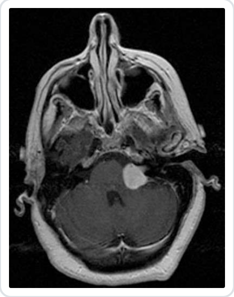
- cranial nerve VIII: vertigo, unilateral sensorineural hearing loss, unilateral tinnitus
- cranial nerve V: absent corneal reflex
- cranial nerve VII: facial palsy

Bilateral vestibular schwannomas are seen in neurofibromatosis type 2.

Patients with a suspected vestibular schwannoma should be referred urgently to ENT. It should be noted though that the tumours are often slow growing, benign and often observed initially.

MRI of the cerebellopontine angle is the investigation of choice. Audiometry is also important as only 5% of patients will have a normal audiogram.





Management is with either surgery, radiotherapy or observation.

123

Next question

Textbooks

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Extended textbook

Links

Patient.info 👍 17 👎 7

Acoustic neuroma

Suggest link Report broken link

Media

AUDITORY NERVE
UNILATERAL SYMPTOMS
CEREBELLO-PONTINE ANGLE
ACOUSTIC NEUROMAS
DIZZINESS
SCHWANN CELLS
FACIAL WEAKNESS

Understanding Acoustic Neuromas

Zero To Finals - YouTube 👍 0 👎 0

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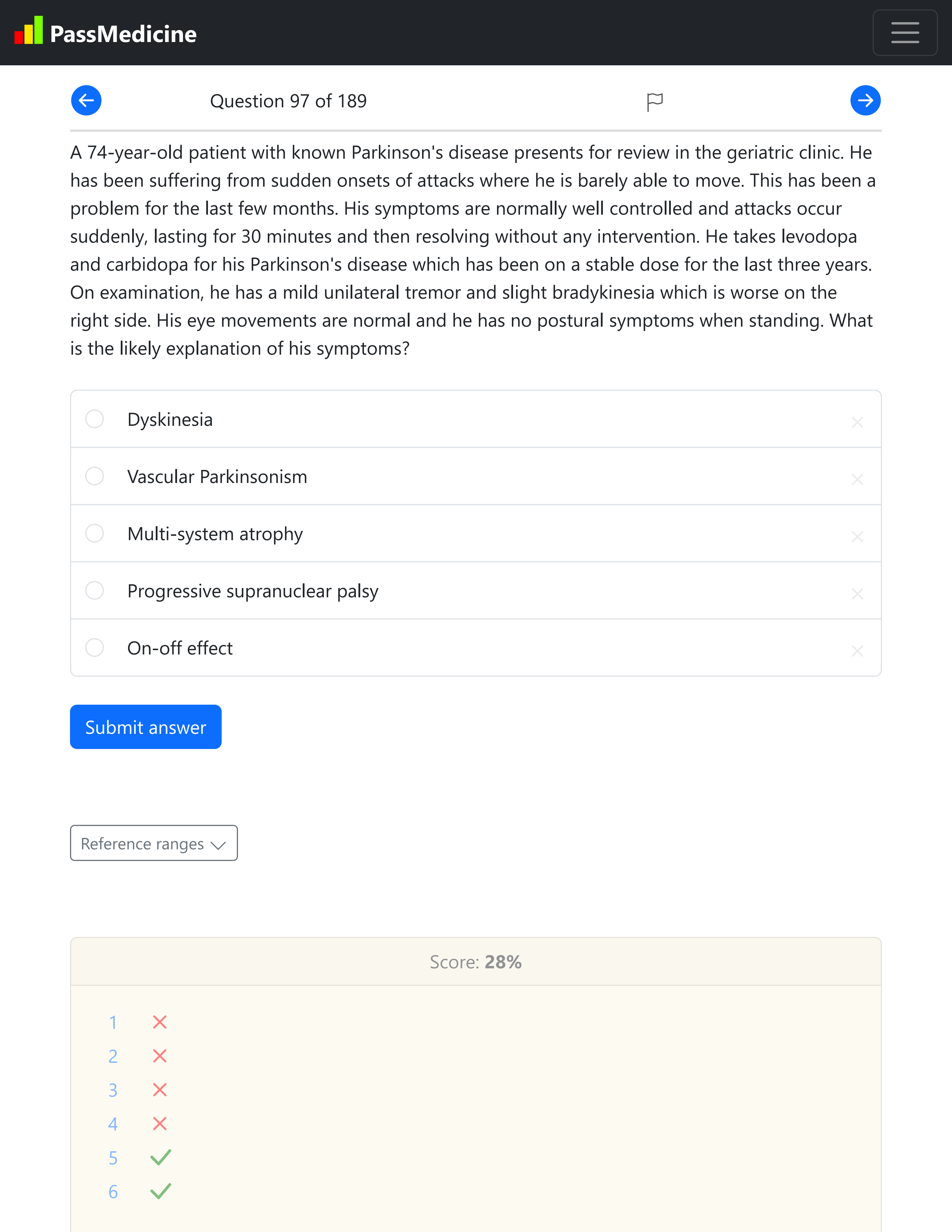

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A 74-year-old patient with known Parkinson's disease presents for review in the geriatric clinic. He has been suffering from sudden onsets of attacks where he is barely able to move. This has been a problem for the last few months. His symptoms are normally well controlled and attacks occur suddenly, lasting for 30 minutes and then resolving without any intervention. He takes levodopa and carbidopa for his Parkinson's disease which has been on a stable dose for the last three years. On examination, he has a mild unilateral tremor and slight bradykinesia which is worse on the right side. His eye movements are normal and he has no postural symptoms when standing. What is the likely explanation of his symptoms?

○ Dyskinesia

○ Vascular Parkinsonism

○ Multi-system atrophy

○ Progressive supranuclear palsy

○ On-off effect

Submit answer

Reference ranges

Score: **28%**

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A 74-year-old patient with known Parkinson's disease presents for review in the geriatric clinic. He has been suffering from sudden onsets of attacks where he is barely able to move. This has been a problem for the last few months. His symptoms are normally well controlled and attacks occur suddenly, lasting for 30 minutes and then resolving without any intervention. He takes levodopa and carbidopa for his Parkinson's disease which has been on a stable dose for the last three years. On examination, he has a mild unilateral tremor and slight bradykinesia which is worse on the right side. His eye movements are normal and he has no postural symptoms when standing. What is the likely explanation of his symptoms?

Dyskinesia	11%
Vascular Parkinsonism	3%
Multi-system atrophy	2%
Progressive supranuclear palsy	3%
On-off effect	82%

The correct answer is on-off effect. Over time, levodopa becomes less effective. Patients can experience after a few years of treatment that their symptoms are well-controlled parts of the day and other times their mobility is heavily affected. This is the most likely explanation given the fact that these symptoms started over two years after treatment and the medication regime will need to be adjusted. A Parkinson-plus syndrome is unlikely as these would not have originally responded to levodopa, and would be associated with other features. For example, progressive supranuclear palsy is associated with gaze palsy whilst multi-system atrophy is associated with postural hypotension. Dyskinesia is continuous writhing movements which occur as a side effect of levodopa.

Discuss (4) Improve

Next question

Parkinson's disease: management

Parkinsons disease should only be diagnosed, and management initiated, by a specialist with expertise in movement disorders. However, it is important for all doctors to be aware of the medications used in Parkinson's given the prevalence of this condition. NICE published guidelines in 2017 regarding the management of Parkinson's disease.

For first-line treatment:

- if the motor symptoms are affecting the patient's quality of life: levodopa
- if the motor symptoms are not affecting the patient's quality of life: dopamine agonist (non-ergot derived), levodopa or monoamine oxidase B (MAO-B) inhibitor

Whilst all drugs used to treat Parkinson's can cause a wide variety of side-effects NICE produced tables to help with decision making:

	Levodopa	**Dopamine agonists**	**MAO-B inhibitors**
Motor symptoms	More improvement in motor symptoms	Less improvement in motor symptoms	Less improvement in motor symptoms
Activities of daily living	More improvement in activities of daily living	Less improvement in activities of daily living	Less improvement in activities of daily living
Motor complications	More motor complications	Fewer motor complications	Fewer motor complications
Adverse events	Fewer specified adverse events*	More specified adverse events*	Fewer specified adverse events*

* excessive sleepiness, hallucinations and impulse control disorders

If a patient continues to have symptoms despite optimal levodopa treatment or has developed dyskinesia then NICE recommend the addition of a dopamine agonist, MAO-B inhibitor or catechol-O-methyl transferase (COMT) inhibitor as an adjunct. Again, NICE summarise the main points in terms of decision making:

	Dopamine agonists	**MAO-B inhibitors**	**COMT inhibitors**	**Amantadine**
Motor symptoms	Improvement in motor symptoms	Improvement in motor symptoms	Improvement in motor symptoms	No evidence of improvement in motor symptoms

	Dopamine agonists	MAO-B inhibitors	COMT inhibitors	Amantadine
Activities of daily living	Improvement in activities of daily living	Improvement in activities of daily living	Improvement in activities of daily living	No evidence of improvement in activities of daily living
Off time	More off-time reduction	Off-time reduction	Off-time reduction	No studies reporting this outcome
Adverse events	Intermediate risk of adverse events	Fewer adverse events	More adverse events	No studies reporting this outcome
Hallucinations	More risk of hallucinations	Lower risk of hallucinations	Lower risk of hallucinations	No studies reporting this outcome

Specific points regarding Parkinson's medication

NICE reminds us of the risk of acute akinesia or neuroleptic malignant syndrome if medication is not taken/absorbed (for example due to gastroenteritis) and advise against giving patients a 'drug holiday' for the same reason.

Impulse control disorders have become a significant issue in recent years. These can occur with any dopaminergic therapy but are more common with:

- dopamine agonist therapy
- a history of previous impulsive behaviours
- a history of alcohol consumption and/or smoking

If excessive daytime sleepiness develops then patients should not drive. Medication should be adjusted to control symptoms. Modafinil can be considered if alternative strategies fail.

If orthostatic hypotension develops then a medication review looking at potential causes should be done. If symptoms persist then midodrine (acts on peripheral alpha-adrenergic receptors to increase arterial resistance) can be considered.

Consider glycopyrronium bromide to manage drooling of saliva in people with Parkinson's

disease.

Further information regarding specific anti-Parkinson's medication

Levodopa

- nearly always combined with a decarboxylase inhibitor (e.g. carbidopa or benserazide)
    - this prevents the peripheral metabolism of levodopa to dopamine outside of the brain and hence can reduce side effects
- common adverse effects:
    - dry mouth
    - anorexia
    - palpitations
    - postural hypotension
    - psychosis
- some adverse effects are due to the difficulty in achieving a steady dose of levodopa
    - end-of-dose wearing off: symptoms often worsen towards the end of dosage interval. This results in a decline of motor activity
    - 'on-off' phenomenon: large variations in motor performance, with normal function during the 'on' period, and weakness and restricted mobility during the 'off' period
    - dyskinesias at peak dose: dystonia, chorea and athetosis (involuntary writhing movements)
    - these effects may worsen over time with - clinicians therefore may limit doses until necessary
- it is important not to acutely stop levodopa, for example, if a patient is admitted to hospital
    - if a patient with Parkinson's disease cannot take levodopa orally, they can be given a dopamine agonist patch as rescue medication to prevent acute dystonia

Dopamine receptor agonists

- e.g. bromocriptine, ropinirole, cabergoline, apomorphine
- ergot-derived dopamine receptor agonists (bromocriptine, cabergoline) have been associated with pulmonary, retroperitoneal and cardiac fibrosis. The Committee on Safety of Medicines advice that an echocardiogram, ESR, creatinine and chest x-ray should be obtained prior to treatment and patients should be closely monitored
- patients should be warned about the potential for dopamine receptor agonists to cause impulse control disorders and excessive daytime somnolence
- more likely than levodopa to cause hallucinations in older patients. Nasal congestion and postural hypotension are also seen in some patients

MAO-B (Monoamine Oxidase-B) inhibitors

- e.g. selegiline
- inhibits the breakdown of dopamine secreted by the dopaminergic neurons

Amantadine

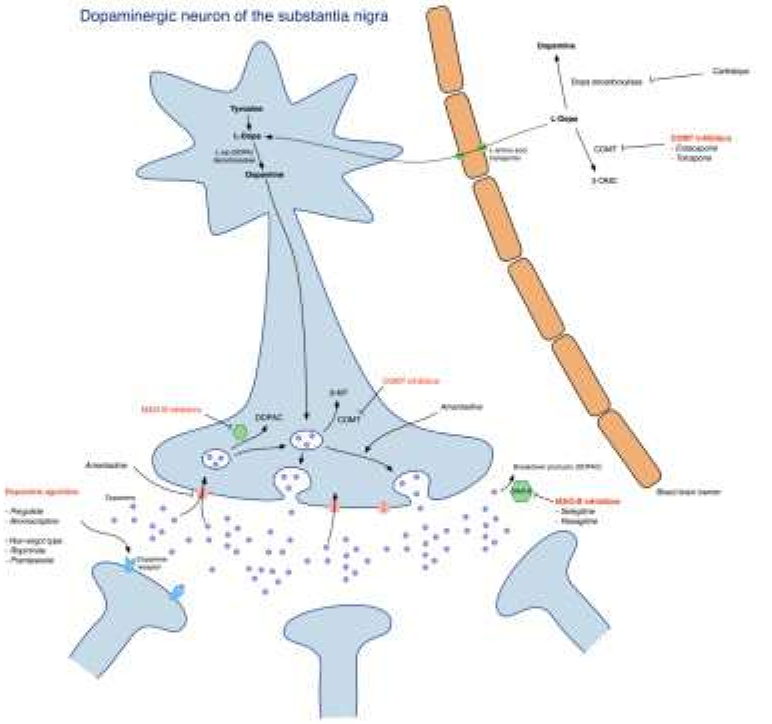
- mechanism is not fully understood, probably increases dopamine release and inhibits its uptake at dopaminergic synapses
- side-effects include ataxia, slurred speech, confusion, dizziness and livedo reticularis

COMT (Catechol-O-Methyl Transferase) inhibitors

- e.g. entacapone, tolcapone
- COMT is an enzyme involved in the breakdown of dopamine, and hence may be used as an adjunct to levodopa therapy
- used in conjunction with levodopa in patients with established PD

Antimuscarinics

- block cholinergic receptors
- now used more to treat drug-induced parkinsonism rather than idiopathic Parkinson's disease
- help tremor and rigidity
- e.g. procyclidine, benzotropine, trihexyphenidyl (benzhexol)


Dopaminergic neuron of the substantia nigra
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COMT
Amantadine
Amantadine

Image sourced from Wikipedia

Diagram showing the mechanism of action of Parkinson's drugs

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Next question

Textbooks

High-yield textbook

Extended textbook

Links

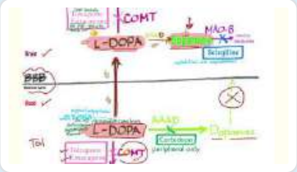
NICE 👍 23 👎 12

[2017 Parkinson's disease guidelines]

Suggest link

Report broken link

Media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The use of Levodopa/ Carbidopa in the treatment of Parkinson's disease

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PARKINSON'S
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Parkinson's disease pharmacology

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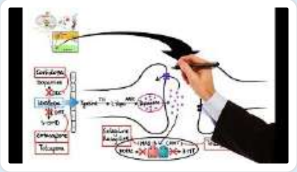

Pharmacology - DRUGS FOR PARKINSON'S DISEASE

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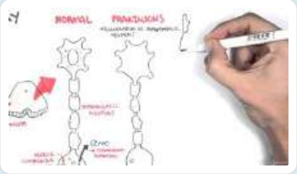
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Pharmacology - Parkinson's Disease

Armando Hasudungan - YouTube 👍 16 👎 2

PARKINSON'S
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Parkinson's disease

Osmosis - YouTube 👍 4 👎 1

PARKINSON'S
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Understanding Parkinson's Disease

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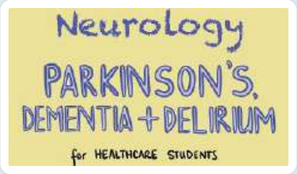
Neurology
PARKINSON'S,
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for HEALTHCARE STUDENTS

Parkinson's disease

MedFlix - YouTube 👍 1 👎 2

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Score: **24.8%**

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A 35-year-old man is brought into the Emergency Department after 3 successive tonic-clonic seizures. He was given 10mg of rectal diazepam and has since stabilised.

A brief history from his girlfriend states that he was usually fit and well apparent from recurrent sinusitis. Over the past month, however, he had been complaining of increasing headaches and had received a course of antibiotics from his GP.

He takes no regular medication but his partner states that they do occasionally take ecstasy whilst on nights out.

On examination, he is drowsy with a Glasgow Coma Scale (GCS) of 12. His temperature is 38.8 degrees Celsius, his pulse is 57 bpm and regular, blood pressure 150/90 mmHg, oxygen saturations 97% on 15L oxygen via a non-rebreathe mask.

Cardiovascular examination reveals normal heart sounds, capillary refill time of 3 seconds. His calves were soft and non-tender. His chest was clear with no signs of consolidation. Abdominal examination was unremarkable.

Neurological examination was difficult in view of the patients low GCS but no focal abnormality could be detected. On attempt to passively flex his neck he became agitated and obviously uncomfortable. Pupils were equal and reactive to light. Fundoscopy demonstrated bilateral oedematous optic discs.

Bloods were as follows:

Na+	130 mmol/L
K+	3.9 mmol/L
Urea	5 mmol/L
Creatinine	80 µmol/L
Hb	160 g/L
WBC	25.0x10^9/L
Neutrophils	91%
LFTs	Normal
CRP	90 mg/L

Based on the findings above, what is the most likely diagnosis?

- ○ Intracerebral abscess ×
- ○ Sepsis secondary to sinusitis ×
- ○ Ecstasy overdose ×
- ○ Meningitis ×
- ○ Cerebral lymphoma ×

Submit answer

Reference ranges ⌄

Score: **28%**

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WBC	25.0x10^9/L
Neutrophils	91%
LFTs	Normal
CRP	90 mg/L

Based on the findings above, what is the most likely diagnosis?

Intracerebral abscess	69%
Sepsis secondary to sinusitis	3%
Ecstasy overdose	4%
Meningitis	23%
Cerebral lymphoma	1%

This is a 30-year-old man who presented with a history of headaches, seizures and a fever. The history of headaches and a course of antibiotics could suggest partially treated sinusitis. This has likely extended posteriorly resulting in an intracranial abscess as indicated by the inflammatory markers, signs of raised intracranial pressure and seizures.

Meningitis can sometimes present with seizures if this has progressed to meningoencephalitis but the history of recurrent sinusitis and headaches with raised intracranial pressure are more suggestive of a space occupying abscess.

Although ecstasy overdose can cause pyrexia and seizures, it is not in keeping with the chronic progression of this patient's symptoms.

Seizures have been associated with severe sepsis but it is not in keeping with all of the signs and symptoms demonstrated here.

👍 👎 Discuss (7) Improve

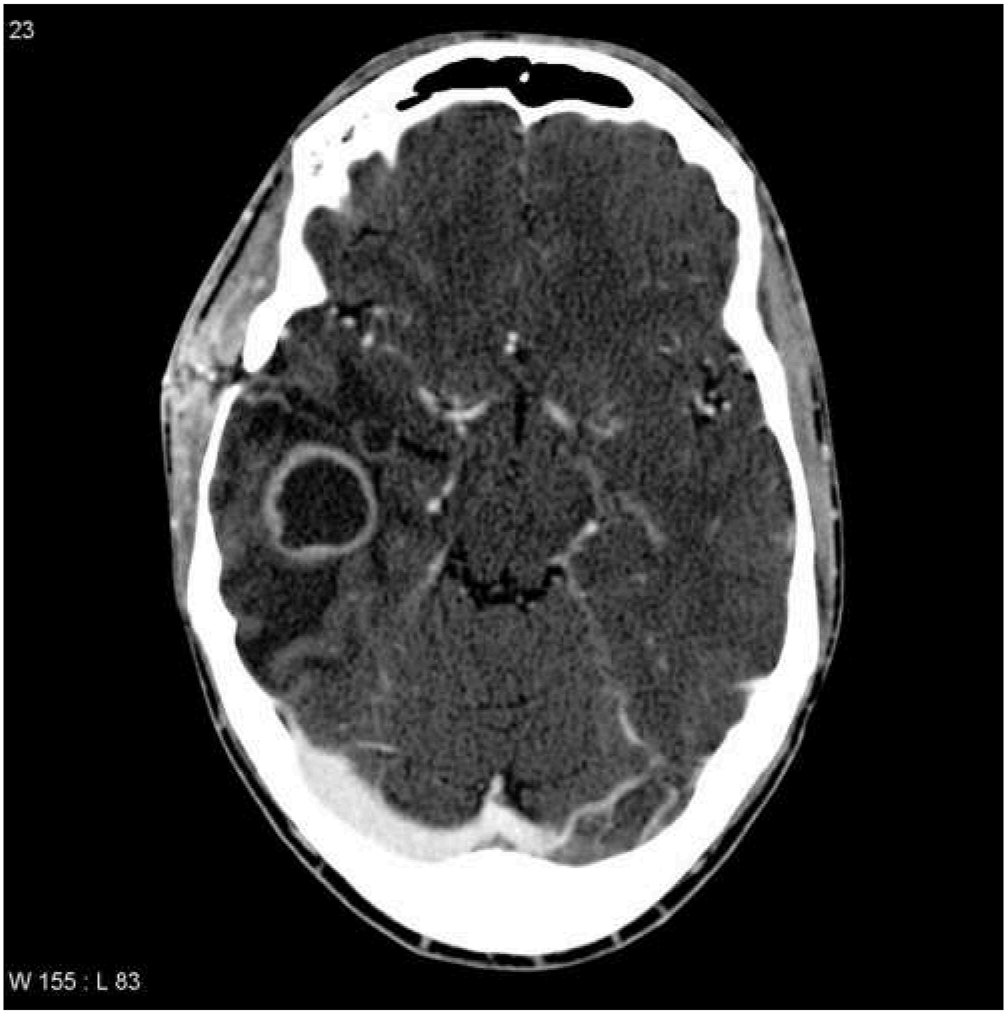
Next question ›

Intracerebral abscess ★

Features
- fever
- headache
- seizures
- signs of raised intracranial pressure
- focal neurological deficits

Management

- surgical drainage: supratentorial abscesses may be drained via a burr hole
- antibiotic therapy



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CT demonstrating a cerebral abscess. CT demonstrates a rounded lesions with vast amounts of surrounding vasogenic oedema resulting in mass effect and distortion of the cerebral peduncle. Contrast confirms the presence of a smooth ring of enhancement, characteristic of an abscess.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **24.8%**

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Question 99 of 189

A 79-year-old is brought into hospital with muscle cramps, fevers and passing dark urine. He is known to have previously had Parkinson's disease and takes Sinemet 125 five times a day. His daughter, who normally picks up his medicine for him from the pharmacy has been called away on business for the past 5 days. As a result, the patient's supplies ran out and he has not taken his PD meds for 3 days. His blood pressure is fluctuant, from 77/52 mmHg to 150/88mm Hg. On examination, his temperature is 39.2 degrees, his heart sounds are quiet but present and chest auscultation is unremarkable. You note rigid muscles in four all limbs, no obvious superficial evidence of head injury and new confusion, with abbreviated mental test 0/10. You start intravenous fluids, intravenous broad spectrum antibiotics, catheterise the patient and insert a nasogastric tube to administer his regular medications. What is the underlying diagnosis?

- ○ Urosepsis
- ○ Restrictive pericarditis
- ○ Idiopathic Parkinson's disease progression
- ○ L-dopa dyskinesia
- ○ Neuroleptic malignant syndrome

Submit answer

Reference ranges

Score: **28%**

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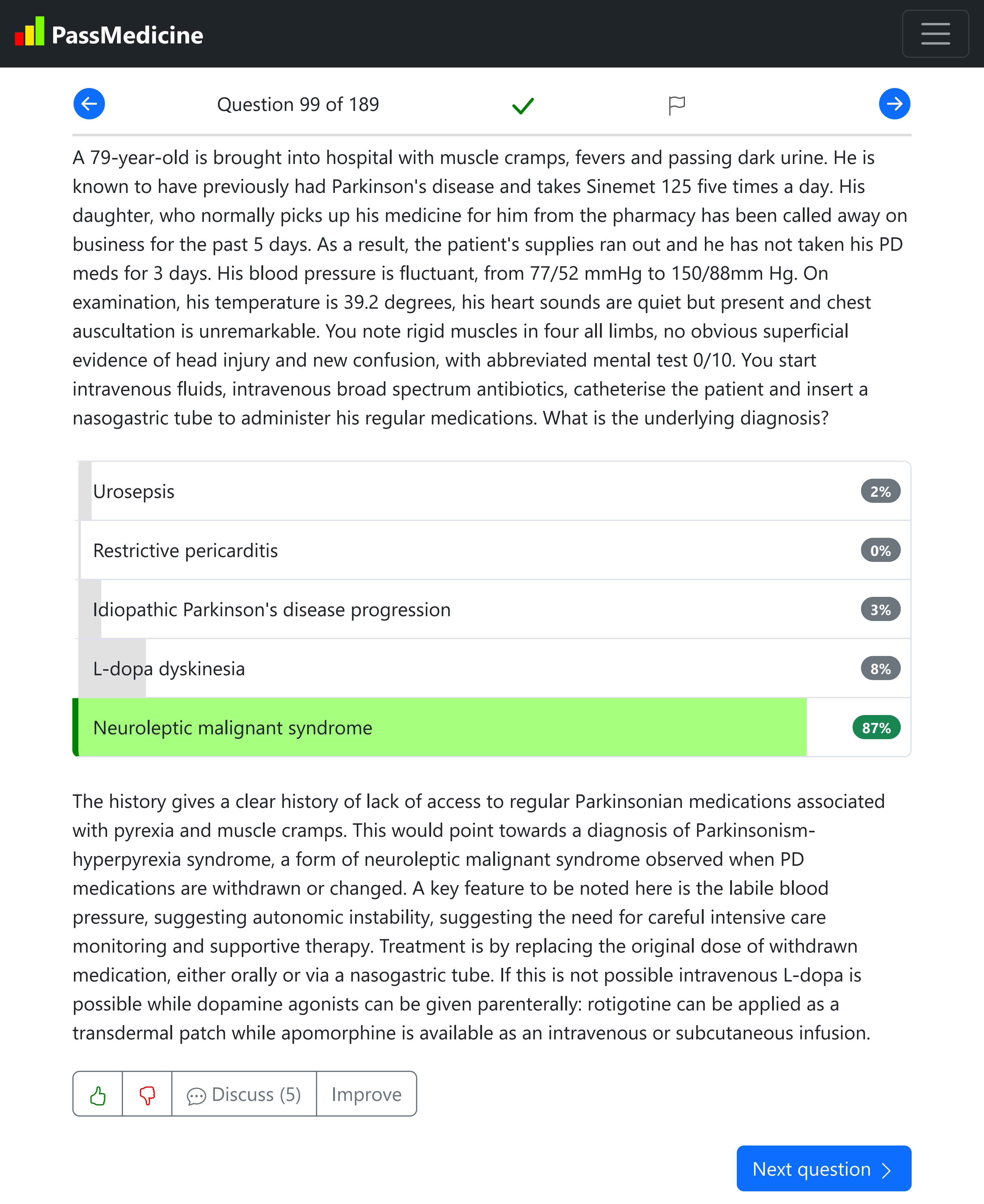

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Urosepsis	2%
Restrictive pericarditis	0%
Idiopathic Parkinson's disease progression	3%
L-dopa dyskinesia	8%
Neuroleptic malignant syndrome	87%

The history gives a clear history of lack of access to regular Parkinsonian medications associated with pyrexia and muscle cramps. This would point towards a diagnosis of Parkinsonism-hyperpyrexia syndrome, a form of neuroleptic malignant syndrome observed when PD medications are withdrawn or changed. A key feature to be noted here is the labile blood pressure, suggesting autonomic instability, suggesting the need for careful intensive care monitoring and supportive therapy. Treatment is by replacing the original dose of withdrawn medication, either orally or via a nasogastric tube. If this is not possible intravenous L-dopa is possible while dopamine agonists can be given parenterally: rotigotine can be applied as a transdermal patch while apomorphine is available as an intravenous or subcutaneous infusion.

Neuroleptic malignant syndrome

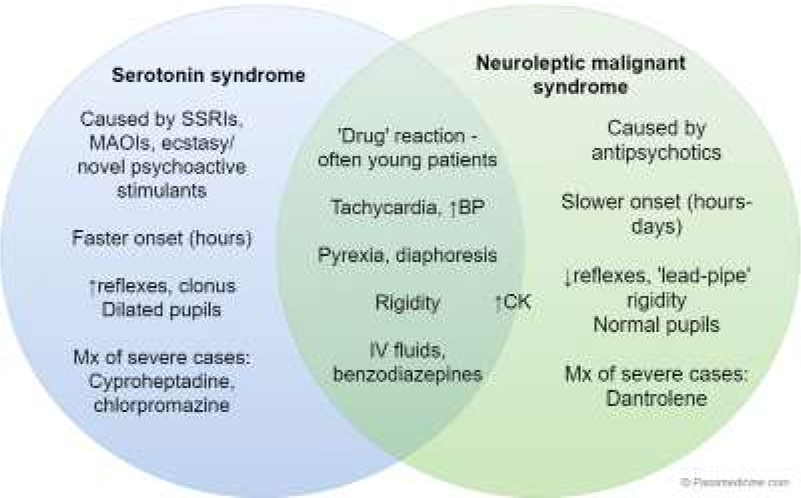
Neuroleptic malignant syndrome is a rare but dangerous condition seen in patients taking antipsychotic medication. It carries a mortality of up to 10% and can also occur with atypical antipsychotics. It may also occur with dopaminergic drugs (such as levodopa) for Parkinson's disease, usually when the drug is suddenly stopped or the dose reduced.

The pathophysiology is unknown but one theory is that the dopamine blockade induced by antipsychotics triggers massive glutamate release and subsequent neurotoxicity and muscle damage.

It occurs within hours to days of starting an antipsychotic (antipsychotics are also known as neuroleptics, hence the name) and the typical features are:
- pyrexia
- muscle rigidity
- autonomic lability: typical features include hypertension, tachycardia and tachypnoea
- agitated delirium with confusion

A raised creatine kinase is present in most cases. Acute kidney injury (secondary to rhabdomyolysis) may develop in severe cases. A leukocytosis may also be seen

Management
- stop antipsychotic
- patients should be transferred to a medical ward if they are on a psychiatric ward and often they are nursed in intensive care units
- IV fluids to prevent renal failure
- dantrolene may be useful in selected cases
    - thought to work by decreasing excitation-contraction coupling in skeletal muscle by binding to the ryanodine receptor, and decreasing the release of calcium from the sarcoplasmic reticulum
- bromocriptine, dopamine agonist, may also be used


Serotonin syndrome
Caused by SSRIs, MAOIs, ecstasy/ novel psychoactive stimulants
Faster onset (hours)
↑reflexes, clonus
Dilated pupils
Mx of severe cases: Cyproheptadine, chlorpromazine
Neuroleptic malignant syndrome
'Drug' reaction - often young patients
Tachycardia, ↑BP
Pyrexia, diaphoresis
Rigidity
↑CK
IV fluids, benzodiazepines
Caused by antipsychotics
Slower onset (hours-days)
↓reflexes, 'lead-pipe' rigidity
Normal pupils
Mx of severe cases: Dantrolene
© Passmedicine.com

Venn diagram showing contrasting serotonin syndrome with neuroleptic malignant syndrome. Note that both conditions can cause a raised creatine kinase (CK) but it tends to be more associated with NMS.

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 8 5

Neuroleptic Malignant Syndrome

Medsafe 15 4

Neuroleptice malignant syndrome Vs serotonin syndrome

Suggest link Report broken link

Media

DOPAMINE ANTAGONISM
AUTONOMIC INSTABILITY
NEUROLEPTIC MALIGNANT SYNDROME
FEVER
ANTIPSYCHOTICS

Neuroleptic malignant syndrome

Rhesus Medicine - YouTube 👍 1 👎 0

NEUROLEPTIC MALIGNANT SYNDROME

Neuroleptic malignant syndrome

Townsend Teaching - YouTube 👍 0 👎 0

Differential DX

Neuroleptic malignant syndrome

Kloss and Bruce - YouTube 👍 0 👎 1

Report broken media

Score: **24.8%**

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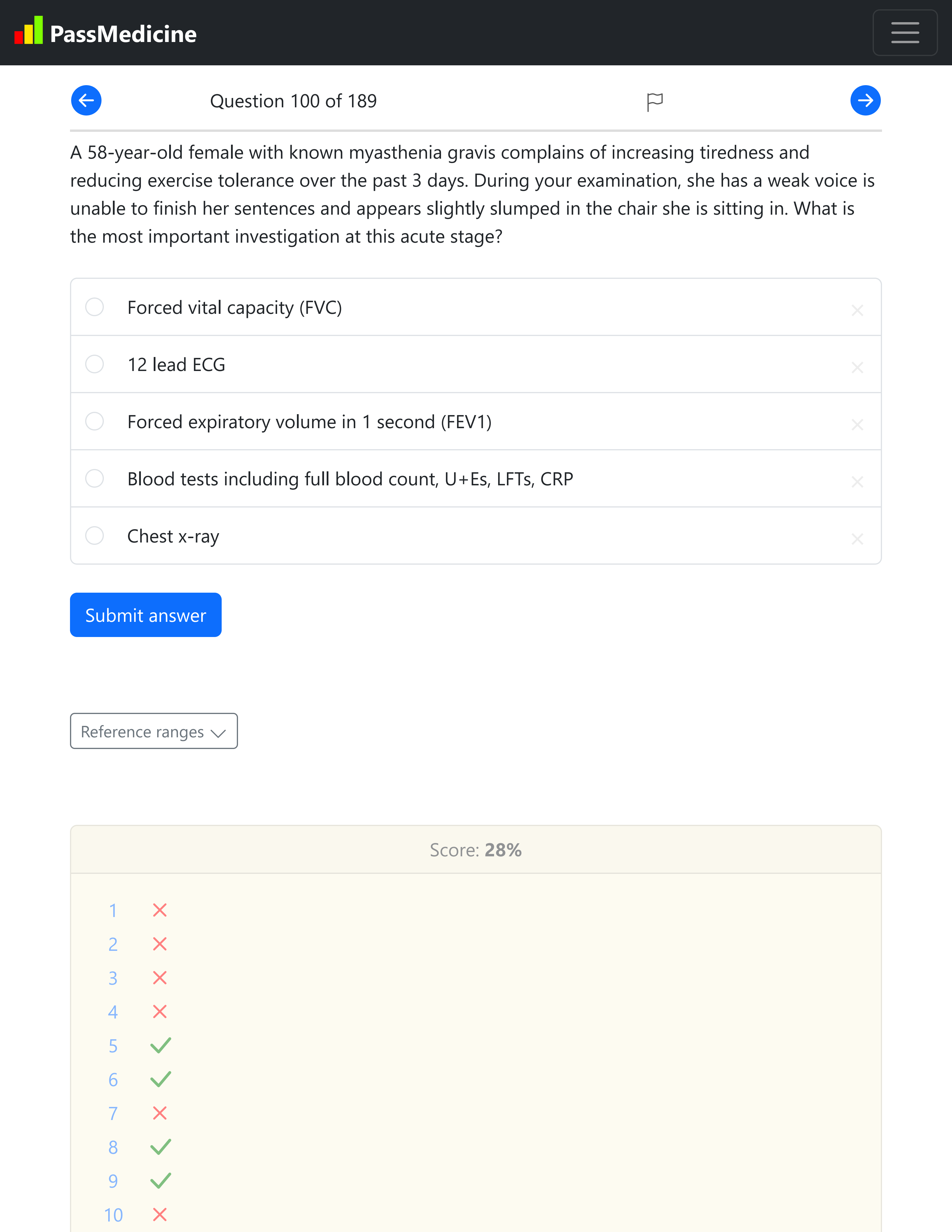

Question 100 of 189

A 58-year-old female with known myasthenia gravis complains of increasing tiredness and reducing exercise tolerance over the past 3 days. During your examination, she has a weak voice is unable to finish her sentences and appears slightly slumped in the chair she is sitting in. What is the most important investigation at this acute stage?

- ○ Forced vital capacity (FVC)
- ○ 12 lead ECG
- ○ Forced expiratory volume in 1 second (FEV1)
- ○ Blood tests including full blood count, U+Es, LFTs, CRP
- ○ Chest x-ray

Score: **28%**

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Question 100 of 189

A 58-year-old female with known myasthenia gravis complains of increasing tiredness and reducing exercise tolerance over the past 3 days. During your examination, she has a weak voice is unable to finish her sentences and appears slightly slumped in the chair she is sitting in. What is the most important investigation at this acute stage?

Forced vital capacity (FVC)	81%
12 lead ECG	0%
Forced expiratory volume in 1 second (FEV1)	14%
Blood tests including full blood count, U+Es, LFTs, CRP	2%
Chest x-ray	3%

The patient is approaching a myasthenic crisis. One of the most acute dangers to be aware of is impending respiratory distress, caused by a failure of respiratory muscles. Other symptoms can include dysphonia, dysphagia and generalised weakness. Forced vital capacity is the most accurate assessment of inspiratory and expiratory mechanisms. FEV1 is better at demonstrating an obstructive than respiratory muscle strength. Although blood tests and chest radiography may be useful in identifying an underlying infective cause for the deterioration, the acute issue remains the patients possible progressive respiratory deterioration, which may warrant elective intubation.

Discuss (3) Improve

Next question ›

Myasthenia gravis ★

Myasthenia gravis is an autoimmune disorder resulting in insufficient functioning acetylcholine receptors. Antibodies to acetylcholine receptors are seen in 85-90% of cases*. Myasthenia is more common in women (2:1)

The key feature is muscle fatigability - muscles become progressively weaker during periods of activity and slowly improve after periods of rest:

- extraocular muscle weakness: diplopia
- proximal muscle weakness: face, neck, limb girdle
- ptosis
- dysphagia

Associations

- thymomas in 15%
- autoimmune disorders: pernicious anaemia, autoimmune thyroid disorders, rheumatoid, SLE
- thymic hyperplasia in 50-70%

Investigations

- single fibre electromyography: high sensitivity (92-100%)
- CT thorax to exclude thymoma
- CK normal
- antibodies to acetylcholine receptors
  - positive in around 85-90% of patients
  - n the remaining patients, about about 40% are positive for anti-muscle-specific tyrosine kinase antibodies
- Tensilon test: IV edrophonium reduces muscle weakness temporarily - not commonly used any more due to the risk of cardiac arrhythmia

Management

- long-acting acetylcholinesterase inhibitors
  - pyridostigmine is first-line
- immunosuppression is usually not started at diagnosis, but the majority of patients eventually require it in addition to long-acting acetylcholinesterase inhibitors:
  - prednisolone initially
  - azathioprine, cyclosporine, mycophenolate mofetil may also be used
- thymectomy

Management of myasthenic crisis

- plasmapheresis
- intravenous immunoglobulins

*antibodies are less commonly seen in disease limited to the ocular muscles

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

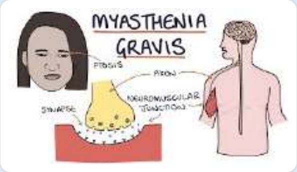
Postgraduate Medical Journal 👍 13 👎 3

Review of myasthenia gravis

Suggest link

Report broken link

Media


MYASTHENIA GRAVIS
PTOSIS
AXON
NEUROMUSCULAR JUNCTION
SYNAPSE

Myasthenia gravis

Zero to Finals - YouTube 👍 12 👎 1

NEUROLOGY
MYASTHENIA GRAVIS

Myasthenia gravis

Armando Hasudungan - YouTube 👍 1 👎 1

MYASTHENIA GRAVIS

Myasthenia gravis - causes, symptoms, treatment, pathology

Osmosis - YouTube 👍 2 👎 2

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101 ✓

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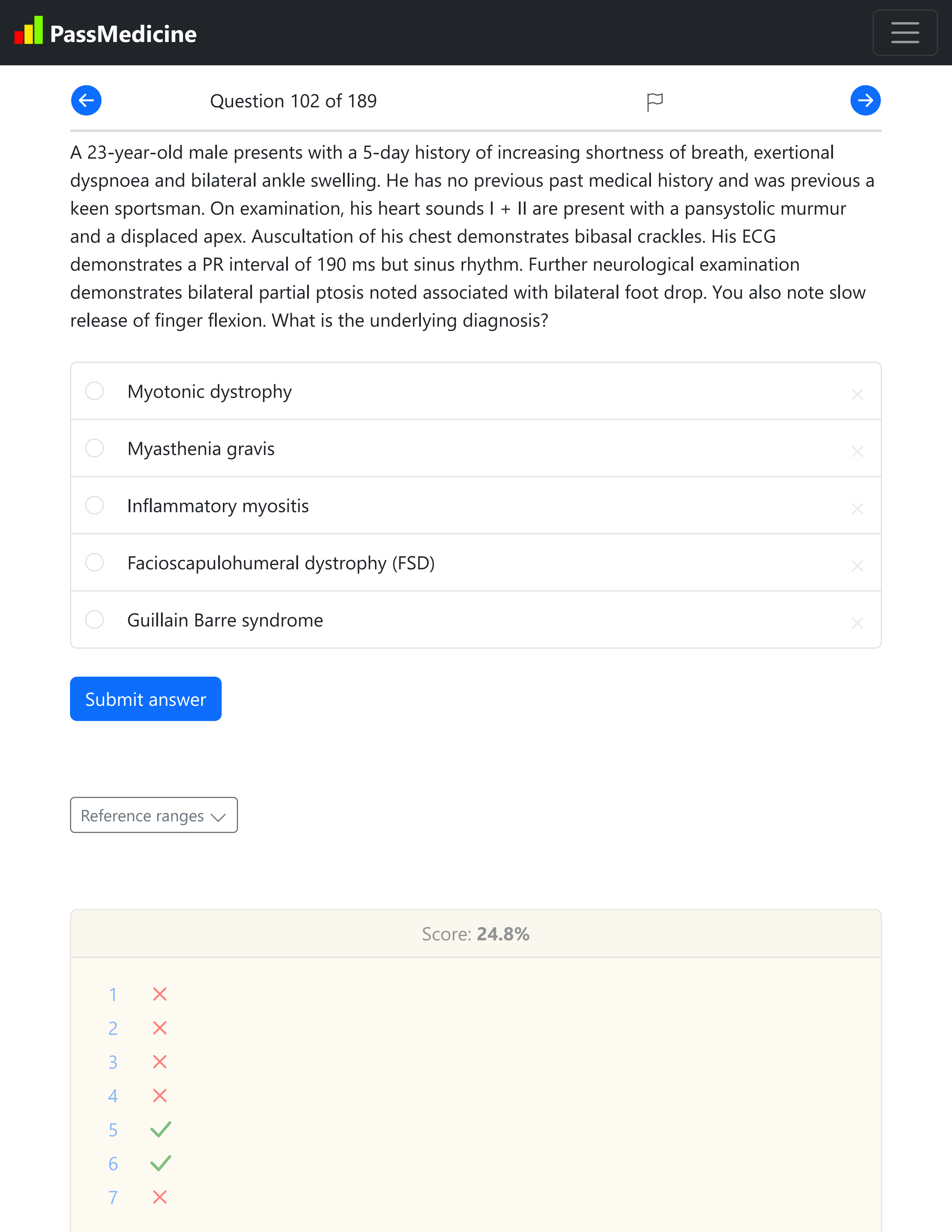

Question 102 of 189

A 23-year-old male presents with a 5-day history of increasing shortness of breath, exertional dyspnoea and bilateral ankle swelling. He has no previous past medical history and was previous a keen sportsman. On examination, his heart sounds I + II are present with a pansystolic murmur and a displaced apex. Auscultation of his chest demonstrates bibasal crackles. His ECG demonstrates a PR interval of 190 ms but sinus rhythm. Further neurological examination demonstrates bilateral partial ptosis noted associated with bilateral foot drop. You also note slow release of finger flexion. What is the underlying diagnosis?

- ○ Myotonic dystrophy
- ○ Myasthenia gravis
- ○ Inflammatory myositis
- ○ Facioscapulohumeral dystrophy (FSD)
- ○ Guillain Barre syndrome

Score: **24.8%**

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A 22-year-old male is referred to clinic by his GP. His initial complaint had been that his friends have been increasingly remarking on how his eyelids look increasing 'droopy' over the past 9 months. He denies any diplopia or muscle weakness. He has no past medical history, is a non-smoker and non-drinker. On examination, you note bilateral inability to abduct or adduct his eyes. Vertical gaze is also inconsistently impaired. His upper and lower limb neurological investigation was unremarkable except very mild finger-nose dysmetria, his bloods tests show no abnormalities. His ECG demonstrates sinus rhythm with a PR interval of 260ms. Fundoscopy reveals a pigmented retina. What is the diagnosis?

Myasthenia gravis	3%
Thyroid eye disease	1%
Oculopharyngeal musculodystrophy	5%
Extraocular muscle fibrosis	2%
Kearns-Sayre syndrome	90%

This young patient demonstrates a combination of retinitis pigmentosa, chronic progressive external ophthalmoplegia, bilateral ptosis, mild cerebellar signs and a cardiac conductive defect (first degree heart block). These features are consistent with a mitochondrial disorder, in particular Kearns-Sayre syndrome. Some patient may also display proximal myopathy, cognitive impairment or short stature. The underlying cause is a genetic mitochondrial disorder. Patients rarely live beyond their 40s and there are no therapeutics currently available.

Discuss (3) | Improve

Next question >

Mitochondrial diseases

Whilst most DNA is found in the cell nucleus, a small amount of double-stranded DNA is present in the mitochondria. It encodes protein components of the respiratory chain and some special types of RNA

Mitochondrial inheritance has the following characteristics:

- inheritance is only via the maternal line as the sperm contributes no cytoplasm to the zygote
- none of the children of an affected male will inherit the disease
- all of the children of an affected female will inherit the disease
- generally, encode rare neurological diseases
- poor genotype:phenotype correlation - within a tissue or cell there can be different mitochondrial populations - this is known as heteroplasmy

Histology

- muscle biopsy classically shows 'red, ragged fibres' due to increased number of mitochondria

Examples include:

- Leber's optic atrophy
  - symptoms typically develop at around the age of 30 years
  - central scotoma → loss of colour vision → rapid onset of significant visual impairment
- MELAS syndrome: mitochondrial encephalomyopathy lactic acidosis and stroke-like episodes
- MERRF syndrome: myoclonus epilepsy with ragged-red fibres
- Kearns-Sayre syndrome: onset in patients < 20 years old, external ophthalmoplegia, retinitis pigmentosa. Ptosis may be seen
- sensorineural hearing loss

AI + Q 123

Next question ›

B I A

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 5 👎 6

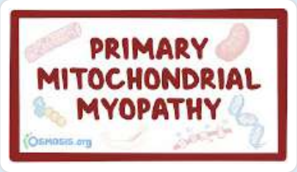
2013 How to spot mitochondrial disease in adults

Rare Disease Video Library - NORD 👍 3 👎 4

Primary Mitochondrial Myopathy

Suggest link Report broken link

Media


PRIMARY MITOCHONDRIAL MYOPATHY
Osmosis.org

Primary mitochondrial myopathy

Osmosis - YouTube 👍 6 👎 1

Report broken media

Score: **20.8%**

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Question 103 of 189

An 84-year-old male presents as a blue light ambulance call with a twelve hour history of sudden onset inability to move his right side. On examination, you note an expressive and receptive dysphasia associated with a dense right sensori-motor syndrome. Cardiovascular examination was unremarkable except for an irregular heartbeat at 80 per minute. A hyperacute CT head demonstrated a large area of ischaemia in a the left middle cerebral artery vascular territory. The patient was outside the window for thrombolysis and started on 300mg aspirin. Subsequent echocardiogram demonstrated 60% ejection fraction with no mural thrombus, 40% left and 35% right stenosis on carotid Doppler while a 24hr tape demonstrated new atrial fibrillation. How can the risk of subsequent strokes be reduced?

- ○ Subcutaneous low molecular heparin at 48 hours after stroke ×
- ○ Warfarinisation at 48 hours after stroke ×
- ○ Warfarinisation at 14 days after stroke ×
- ○ Referral to vascular surgery for left carotid endarectomy ×
- ○ Insertion of permanent pacemaker ×

Submit answer

Reference ranges

Score: **24.8%**

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A 23-year-old male presents with a 5-day history of increasing shortness of breath, exertional dyspnoea and bilateral ankle swelling. He has no previous past medical history and was previous a keen sportsman. On examination, his heart sounds I + II are present with a pansystolic murmur and a displaced apex. Auscultation of his chest demonstrates bibasal crackles. His ECG demonstrates a PR interval of 190 ms but sinus rhythm. Further neurological examination demonstrates bilateral partial ptosis noted associated with bilateral foot drop. You also note slow release of finger flexion. What is the underlying diagnosis?

Myotonic dystrophy	73%
Myasthenia gravis	4%
Inflammatory myositis	13%
Facioscapulohumeral dystrophy (FSD)	8%
Guillain Barre syndrome	2%

The patient has presented with clinical heart failure on a background of systemic disease, involving structural heart disease, conductive heart block, bilateral ptosis bilateral foot drop and slow finger flexion release suggestive of myotonia. It would not be possible to distinguish at this stage between DM1 or DM2 myotonic dystrophy, the latter being less severe than DM1. The diagnosis would be clinched with genetic testing for an expanded CTG repeat in the DMPK gene: normally, 5-34 CTG repeats are found. Greater than 50 alleles is strongly diagnostic of disease manifestations.

Discuss (5) | Improve

Next question ›

Myotonic dystrophy ★

Myotonic dystrophy (also called dystrophia myotonica) is an inherited myopathy with features developing at around 20-30 years old. It affects skeletal, cardiac and smooth muscle. There are two main types of myotonic dystrophy, DM1 and DM2.

Genetics

- autosomal dominant
- a trinucleotide repeat disorder
- DM1 is caused by a CTG repeat at the end of the DMPK (Dystrophia Myotonica-Protein Kinase) gene on chromosome 19
- DM2 is caused by a repeat expansion of the ZNF9 gene on chromosome 3

The key differences are listed in table below:

DM1	**DM2**
- DMPK gene on chromosome 19 - Distal weakness more prominent	- ZNF9 gene on chromosome 3 - Proximal weakness more prominent - Severe congenital form not seen

General features

- myotonic facies (long, 'haggard' appearance)
- frontal balding
- bilateral ptosis
- cataracts
- dysarthria

Other features

- myotonia (tonic spasm of muscle)
- weakness of arms and legs (distal initially)
- mild mental impairment
- diabetes mellitus
- testicular atrophy
- cardiac involvement: heart block, cardiomyopathy
- dysphagia

123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Myotonic dystrophy

Zero To Finals - YouTube 👍 4 👎 0

Report broken media

Score: **20.8%**

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A 55-year-old female presents with 3 weeks of bilateral tingling sensation in her medial one and half digits at night. She has noted a clawing of her 4th and 5th digits and she is particularly concerned by the cosmetic elements. She also complains of a left sided foot drop present over the past 8 months. Her past medical history includes type 2 diabetes mellitus, for which she take metformin 850mg TDS and she admits to occasional poor compliance. Her last HbA1c was 53 mmol/mol. She has also had multiple admissions for surgery to her feet at childhood but she is unaware of further details. She was adopted and is unaware of her birth family history. On examination, she clinically has a left common peroneal palsy with bilateral thin calves, and loss of sensation in bilateral ulnar nerve territories. What is the unifying diagnosis for her presenting paraesthesia and foot drop?

- ○ Hereditary neuropathy with liability to pressure palsies
- ○ Diabetic neuropathy
- ○ Chronic inflammation demyelinating polyneuropathy (CIDP)
- ○ Systemic lupus erythematosus (SLE)
- ○ Sarcoidosis

Submit answer

Reference ranges

Score: **24.8%**

1 ✗
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An 84-year-old male presents as a blue light ambulance call with a twelve hour history of sudden onset inability to move his right side. On examination, you note an expressive and receptive dysphasia associated with a dense right sensori-motor syndrome. Cardiovascular examination was unremarkable except for an irregular heartbeat at 80 per minute. A hyperacute CT head demonstrated a large area of ischaemia in a the left middle cerebral artery vascular territory. The patient was outside the window for thrombolysis and started on 300mg aspirin. Subsequent echocardiogram demonstrated 60% ejection fraction with no mural thrombus, 40% left and 35% right stenosis on carotid Doppler while a 24hr tape demonstrated new atrial fibrillation. How can the risk of subsequent strokes be reduced?

Subcutaneous low molecular heparin at 48 hours after stroke	8%
Warfarinisation at 48 hours after stroke	5%
Warfarinisation at 14 days after stroke	82%
Referral to vascular surgery for left carotid endarectomy	4%
Insertion of permanent pacemaker	1%

This patient demonstrates an ischaemic infarct secondary to likely new atrial fibrillation, for which the optimum treatment is anticoagulation. The degree of carotid stenosis is not sufficiently significant to justify carotid endarectomy. The patient has no indications for a pacemaker. The treatment of choice for atrial fibrillation in such a high-risk patient for further strokes is anticoagulation.

In the absence of intracerebral haemorrhages or haemorrhagic transformation of infarcts, NICE guidelines recommend starting anticoagulation at 14 days after the onset of stroke[1]. Due to the risk of haemorrhage as the infarcted brain matures, which is increased with the size of the initial infarct, early anticoagulation is not recommended. A recent meta-analysis collated the results of seven trials studying early anticoagulation in acute ischaemic strokes. Although the risk of further ischaemic strokes between days 7 and 14 is significantly reduced from 4.9 to 3%, the risk of symptomatic intracerebral haemorrhage was also significantly increased fro 0.7% to 2.5%[2]. As a result, early anticoagulation before 14 days is not indicated by NICE or the American Heart and Stroke Associations[3].

1. Atrial Fibrillation: the management of atrial fibrillation. NICE Guidline 2006

2. Paciaroni M, Agnelli G, Micheli S, Caso V. Efficacy and safety of anticoagulant treatment in acute cardioembolic stroke: a meta-analysis of randomized controlled trials. Stroke. 2007;38(2):423
3. Jauch EC, Saver JL, Adams HP Jr, Bruno A, Connors JJ, Demaerschalk BM, Khatri P, McMullan PW Jr, Qureshi AI, Rosenfield K, Scott PA, Summers DR, Wang DZ, Wintermark M, Yonas H, American Heart Association Stroke Council, Council on Cardiovascular Nursing, Council on Peripheral Vascular Disease, Council on Clinical Cardiology. Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2013;44(3):870.

Discuss (4) | Improve

Next question ›

Stroke: management of acute ischaemic stroke

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
    - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
    - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:
- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.
- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
  - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
  - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.
- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6 hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

+ Q 123

Next question >

B *I* A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 11 👎 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 15

Stroke and TIA guidelines

Age and Ageing 👍 20 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

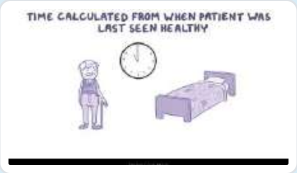
2023 National Clinical Guideline for Stroke

NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia
Acute Ischaemic Stroke

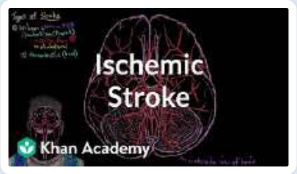
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

Khan Academy - YouTube

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Report broken media

Score: **20.8%**

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A 32-year-old female presents with four episodes of loss of consciousness within the past 4 weeks. She denies palpitations or chest pain but reports sudden onset binocular black dots in visual fields, occasional flashing lights, dysarthria and hearing loss, all of which resolves after about 60 minutes. She is unsure about the relevance of an occipital headache, onset with frequency of about three times per week for the past year. She denies any limb weakness, altered sensation or facial droop. She has no past medical history or family history of epilepsy. Your neurological examination, including fundoscopy is unremarkable. An EEG is unremarkable. What is the likely diagnosis?

- ○ Cluster headache ×
- ○ Transient ischaemic attacks ×
- ○ Basilar migraine ×
- ○ Bilateral retinal detachment ×
- ○ Guillain-Barre syndrome ×

Submit answer

Reference ranges

Score: **24.8%**

1 ✗
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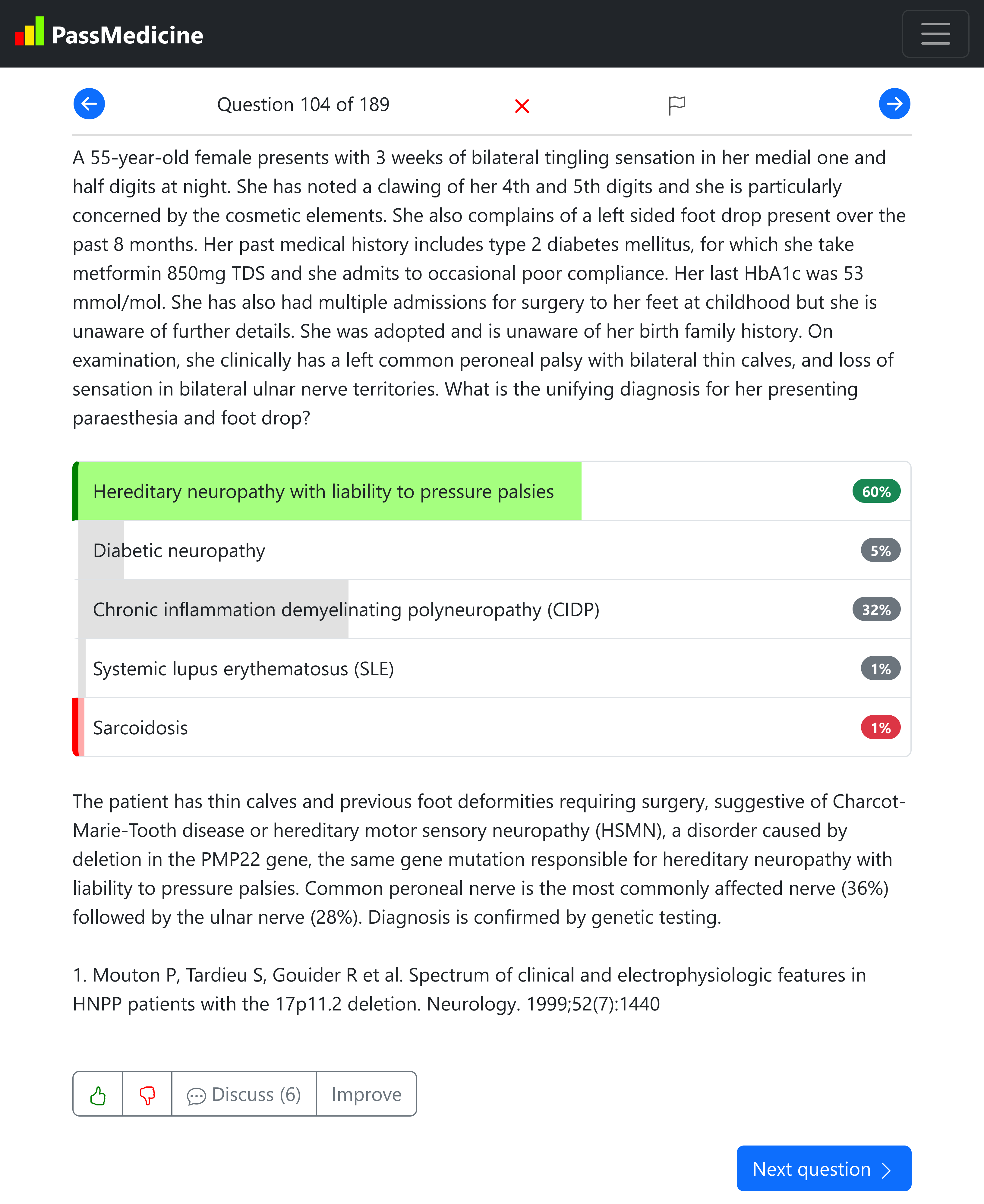

A 55-year-old female presents with 3 weeks of bilateral tingling sensation in her medial one and half digits at night. She has noted a clawing of her 4th and 5th digits and she is particularly concerned by the cosmetic elements. She also complains of a left sided foot drop present over the past 8 months. Her past medical history includes type 2 diabetes mellitus, for which she take metformin 850mg TDS and she admits to occasional poor compliance. Her last HbA1c was 53 mmol/mol. She has also had multiple admissions for surgery to her feet at childhood but she is unaware of further details. She was adopted and is unaware of her birth family history. On examination, she clinically has a left common peroneal palsy with bilateral thin calves, and loss of sensation in bilateral ulnar nerve territories. What is the unifying diagnosis for her presenting paraesthesia and foot drop?

Hereditary neuropathy with liability to pressure palsies	60%
Diabetic neuropathy	5%
Chronic inflammation demyelinating polyneuropathy (CIDP)	32%
Systemic lupus erythematosus (SLE)	1%
Sarcoidosis	1%

The patient has thin calves and previous foot deformities requiring surgery, suggestive of Charcot-Marie-Tooth disease or hereditary motor sensory neuropathy (HSMN), a disorder caused by deletion in the PMP22 gene, the same gene mutation responsible for hereditary neuropathy with liability to pressure palsies. Common peroneal nerve is the most commonly affected nerve (36%) followed by the ulnar nerve (28%). Diagnosis is confirmed by genetic testing.

1. Mouton P, Tardieu S, Gouider R et al. Spectrum of clinical and electrophysiologic features in HNPP patients with the 17p11.2 deletion. Neurology. 1999;52(7):1440

HSMN ★

Hereditary sensorimotor neuropathy (HSMN) is a relatively new term which encompasses Charcot-Marie-Tooth disease (also known as peroneal muscular atrophy). Over 7 types have been characterised - however only 2 are common to clinical practice

- HSMN type I: primarily due to demyelinating pathology
- HSMN type II: primarily due to axonal pathology

HSMN type I

- autosomal dominant
- due to defect in PMP-22 gene (which codes for myelin)
- features often start at puberty
- motor symptoms predominate
- distal muscle wasting, pes cavus, clawed toes
- foot drop, leg weakness often first features

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20.8%**

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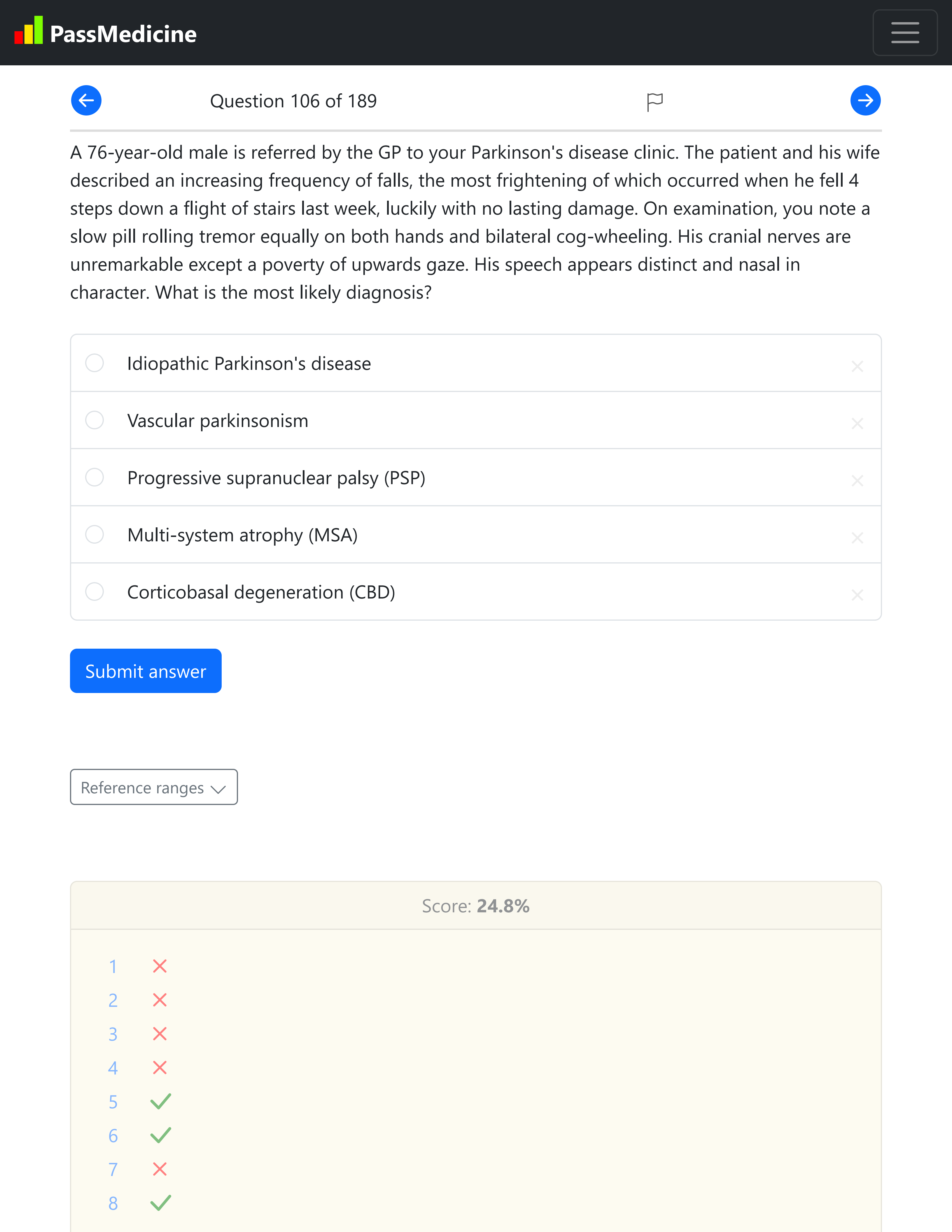

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A 76-year-old male is referred by the GP to your Parkinson's disease clinic. The patient and his wife described an increasing frequency of falls, the most frightening of which occurred when he fell 4 steps down a flight of stairs last week, luckily with no lasting damage. On examination, you note a slow pill rolling tremor equally on both hands and bilateral cog-wheeling. His cranial nerves are unremarkable except a poverty of upwards gaze. His speech appears distinct and nasal in character. What is the most likely diagnosis?

- ○ Idiopathic Parkinson's disease
- ○ Vascular parkinsonism
- ○ Progressive supranuclear palsy (PSP)
- ○ Multi-system atrophy (MSA)
- ○ Corticobasal degeneration (CBD)

Score: **24.8%**

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Question 105 of 189

A 32-year-old female presents with four episodes of loss of consciousness within the past 4 weeks. She denies palpitations or chest pain but reports sudden onset binocular black dots in visual fields, occasional flashing lights, dysarthria and hearing loss, all of which resolves after about 60 minutes. She is unsure about the relevance of an occipital headache, onset with frequency of about three times per week for the past year. She denies any limb weakness, altered sensation or facial droop. She has no past medical history or family history of epilepsy. Your neurological examination, including fundoscopy is unremarkable. An EEG is unremarkable. What is the likely diagnosis?

Cluster headache	3%
Transient ischaemic attacks	8%
Basilar migraine	87%
Bilateral retinal detachment	2%
Guillain-Barre syndrome	1%

The symptoms are most likely to represent a rare migraine subtype with aura known as basilar migraine. A large Danish study identified up to 10% of all non-hemiplegic migraineurs as basilar migraines, most of whom report a brainstem-associated range of symptoms lasting for around one hour[1]. 61% of patients report vertigo and over half dysarthria. One in four report episodes of loss of consciousness. Investigations include 24 hour tape and echocardiogram to exclude cardiac syncope and MRI to exclude posterior fossa space occupying lesion. An EEG may not exclude posterior epileptiform activity. Prevention can be offered with verapamil or topiramate.

1. Kirchmann M, Thomsen LL, Olesen J. Basilar-type migraine: clinical, epidemiologic, and genetic features. Neurology. 2006;66(6):880

Discuss (4) Improve

Next question ›

Migraine: diagnostic criteria

The International Headache Society has produced the following diagnostic criteria for migraine without aura:

Point	Criteria
A	At least 5 attacks fulfilling criteria B-D
B	Headache attacks lasting 4-72 hours* (untreated or unsuccessfully treated)
C	Headache has at least two of the following characteristics: - 1. unilateral location* - 2. pulsating quality (i.e., varying with the heartbeat) - 3. moderate or severe pain intensity - 4. aggravation by or causing avoidance of routine physical activity (e.g., walking or climbing stairs)
D	During headache at least one of the following: - 1. nausea and/or vomiting* - 2. photophobia and phonophobia
E	Not attributed to another disorder (history and examination do not suggest a secondary headache disorder or, if they do, it is ruled out by appropriate investigations or headache attacks do not occur for the first time in close temporal relation to the other disorder)

*In children, attacks may be shorter-lasting, headache is more commonly bilateral, and gastrointestinal disturbance is more prominent.

Migraine with aura (seen in around 25% of migraine patients) tends to be easier to diagnose with a typical aura being progressive in nature and may occur hours prior to the headache. Typical aura include a transient hemianopic disturbance or a spreading scintillating scotoma ('jagged crescent'). Sensory symptoms may also occur

If we compare these guidelines to the **NICE criteria** the following points are noted:
- NICE suggests migraines may be unilateral or bilateral
- NICE also give more detail about typical auras:

Auras may occur with or without headache and:
- are fully reversible
- develop over at least 5 minutes
- last 5-60 minutes

The following aura symptoms are atypical and may prompt further investigation/referral;

- motor weakness
- double vision
- visual symptoms affecting only one eye
- poor balance
- decreased level of consciousness.

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

International Headache Society 👍 7 👎 5

2019 The International Classification of Headache Disorders

Suggest link Report broken link

Media

Management of Migraine
Podmedics

Migraine

Podmedics - YouTube 👍 4 👎 0

Migraine

Townsend Teaching - YouTube 👍 0 👎 0

Report broken media

Score: **20.8%**

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A 32-year-old male presents with a sudden onset left sided weakness with new onset expressive dysphasia and dress apraxia. He has a seven year history of progressive cognitive impairment and seizures, lives in sheltered accommodation and was brought in after his relatives, who visit him on a weekly basis, noted a change from his baseline. The patient is a poor historian and could not remember the duration of symptoms. He reports a recent history of burning sensation when passing urine associated with increased frequency and reduced oral intake for the past four days. An MRI head demonstrates multiple areas of ischaemia within left and right cortex inconsistent with one single vascular territory. A urine dip is positive for leucocytes and nitrites, negative for ketones. A venous blood gas is taken:

pH	7.15
$PaCO_2$	2.4 kPa
Bicarbonate	6 mmol/l
Lactate	18 mmol/l
Anion gap	16 mmol/l

What is the unifying diagnosis?

○ MELAS

○ Diabetic ketoacidosis

○ Ingestion of antifreeze

○ Severe dehydration secondary to urosepsis

○ Metformin induced lactic acidosis

Submit answer

Reference ranges

Score: **24.8%**

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Question 106 of 189

A 76-year-old male is referred by the GP to your Parkinson's disease clinic. The patient and his wife described an increasing frequency of falls, the most frightening of which occurred when he fell 4 steps down a flight of stairs last week, luckily with no lasting damage. On examination, you note a slow pill rolling tremor equally on both hands and bilateral cog-wheeling. His cranial nerves are unremarkable except a poverty of upwards gaze. His speech appears distinct and nasal in character. What is the most likely diagnosis?

Idiopathic Parkinson's disease	4%
Vascular parkinsonism	1%
Progressive supranuclear palsy (PSP)	88%
Multi-system atrophy (MSA)	6%
Corticobasal degeneration (CBD)	2%

PSP is most likely in this scenario. A number of features should alert you to a Parkinson's-plus syndrome instead of idiopathic Parkinson's disease: bilateral, symmetrical symptoms are uncommon, particularly in early PD. In addition, eye signs are very unusual. PSP patients classically present with increasing falls near the stairs due to an impaired vertical gaze and a nasal 'Donald-Duck' voice secondary to pseudobulbar palsy. MSA patients classically present with significant cerebellar or autonomic symptoms. CBD present with higher order dysfunction such as apraxia and aphasia, but classically with 'alien hand syndrome', thought to be as prevalent as 60% of CBD patients, when the patient has no control over their own hand movements.

Discuss (4) Improve

Next question >

Progressive supranuclear palsy

Overview

- aka Steele-Richardson-Olszewski syndrome

- a 'Parkinson Plus' syndrome

Features

- postural instability and falls
  - patients tend to have a stiff, broad-based gait
- impairment of vertical gaze (down gaze worse than up gaze - patients may complain of difficultly reading or descending stairs)
- parkinsonism
  - bradykinesia is prominent
- cognitive impairment
  - primarily frontal lobe dysfunction

Management

- poor response to L-dopa

+ Q 123

Next question >

B *I* A

Textbooks

High-yield textbook

Extended textbook

Score: **20.8%**

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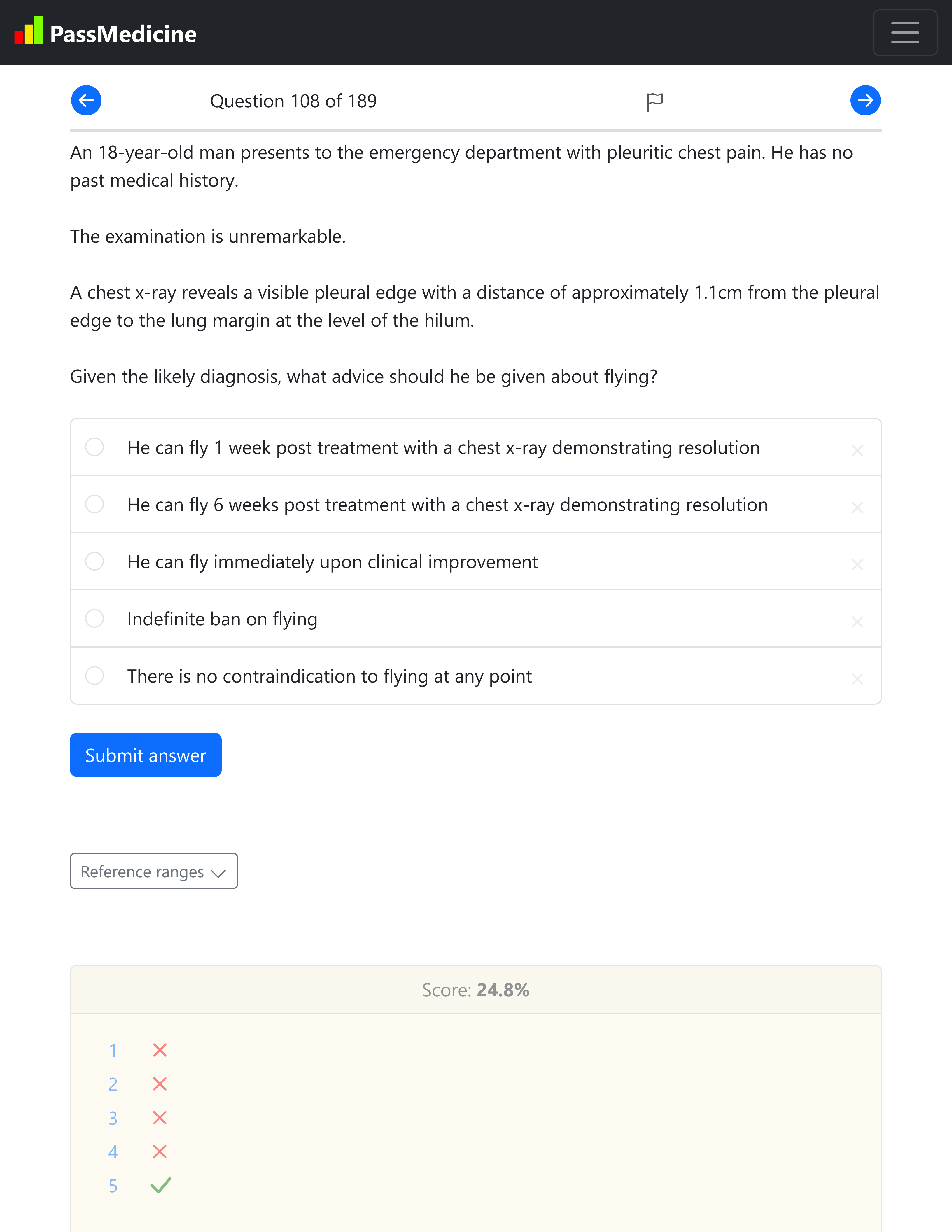

An 18-year-old man presents to the emergency department with pleuritic chest pain. He has no past medical history.

The examination is unremarkable.

A chest x-ray reveals a visible pleural edge with a distance of approximately 1.1cm from the pleural edge to the lung margin at the level of the hilum.

Given the likely diagnosis, what advice should he be given about flying?

- ○ He can fly 1 week post treatment with a chest x-ray demonstrating resolution
- ○ He can fly 6 weeks post treatment with a chest x-ray demonstrating resolution
- ○ He can fly immediately upon clinical improvement
- ○ Indefinite ban on flying
- ○ There is no contraindication to flying at any point

Score: **24.8%**

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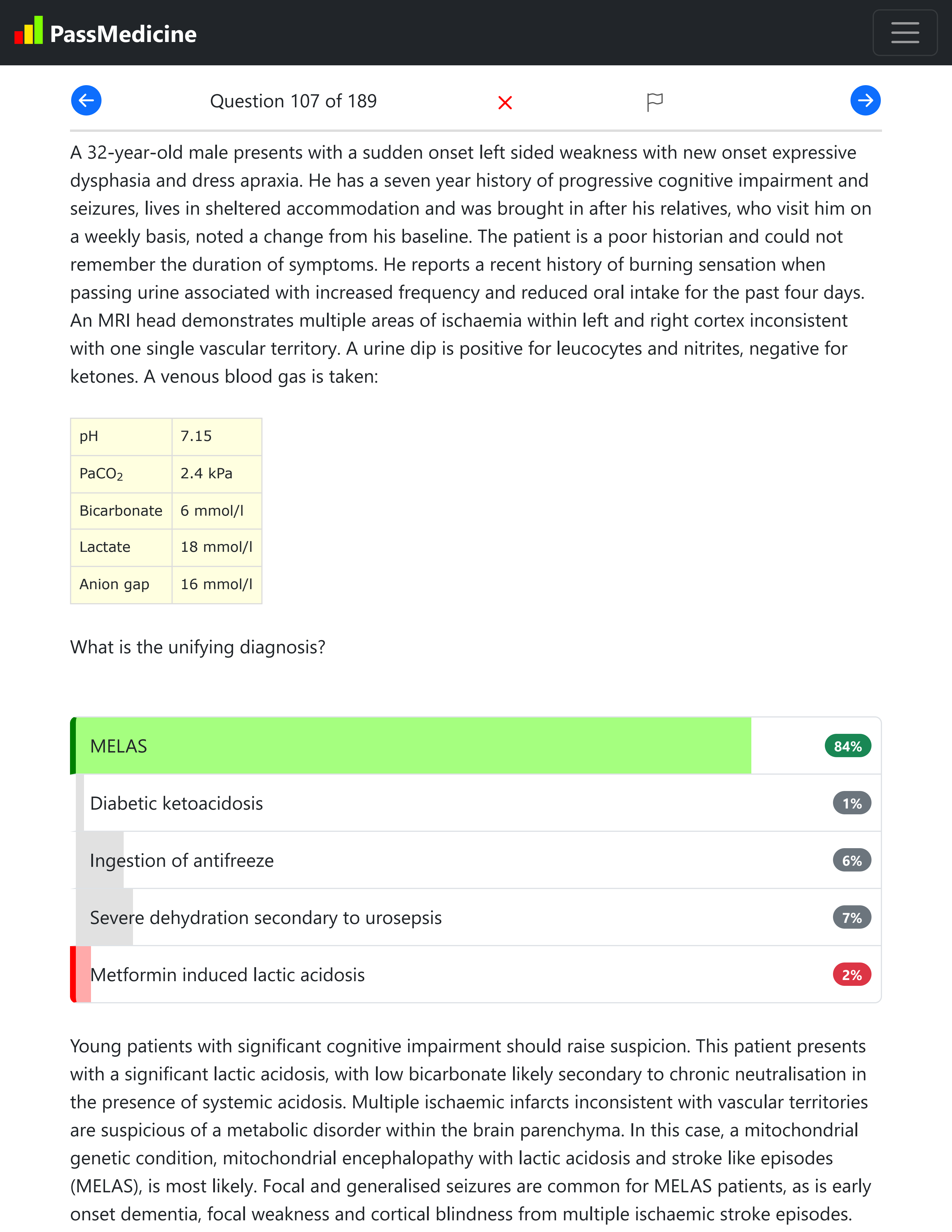


A 32-year-old male presents with a sudden onset left sided weakness with new onset expressive dysphasia and dress apraxia. He has a seven year history of progressive cognitive impairment and seizures, lives in sheltered accommodation and was brought in after his relatives, who visit him on a weekly basis, noted a change from his baseline. The patient is a poor historian and could not remember the duration of symptoms. He reports a recent history of burning sensation when passing urine associated with increased frequency and reduced oral intake for the past four days. An MRI head demonstrates multiple areas of ischaemia within left and right cortex inconsistent with one single vascular territory. A urine dip is positive for leucocytes and nitrites, negative for ketones. A venous blood gas is taken:

pH	7.15
$PaCO_2$	2.4 kPa
Bicarbonate	6 mmol/l
Lactate	18 mmol/l
Anion gap	16 mmol/l

What is the unifying diagnosis?

MELAS	84%
Diabetic ketoacidosis	1%
Ingestion of antifreeze	6%
Severe dehydration secondary to urosepsis	7%
Metformin induced lactic acidosis	2%

Young patients with significant cognitive impairment should raise suspicion. This patient presents with a significant lactic acidosis, with low bicarbonate likely secondary to chronic neutralisation in the presence of systemic acidosis. Multiple ischaemic infarcts inconsistent with vascular territories are suspicious of a metabolic disorder within the brain parenchyma. In this case, a mitochondrial genetic condition, mitochondrial encephalopathy with lactic acidosis and stroke like episodes (MELAS), is most likely. Focal and generalised seizures are common for MELAS patients, as is early onset dementia, focal weakness and cortical blindness from multiple ischaemic stroke episodes.

Symptoms normally present at late childhood and early adulthood, with patients develop normally until then. Diagnosis is made through lactic acidosis, a progressive neurological and dementing clinical course and muscle biopsy for ragged red fibres. The majority of patients have been linked to an underlying mutation in transfer RNA[1].

In this case, the UTI is a red herring. While the patient may have a UTI, it does not produce a unifying diagnosis of cerebral and metabolic abnormalities. A normal anion gap rules out ingestion of inorganic acids or DKA. The patient is negative for urinary ketones. There is no suggestion he takes metformin.

1. Montagna P, Gallassi R, Medori R, Govoni E, Zeviani M, Di Mauro S, Lugaresi E, Andermann F. MELAS syndrome: characteristic migrainous and epileptic features and maternal transmission. Neurology. 1988;38(5):751

Discuss (7) | Improve

Next question ›

Mitochondrial diseases

Whilst most DNA is found in the cell nucleus, a small amount of double-stranded DNA is present in the mitochondria. It encodes protein components of the respiratory chain and some special types of RNA

Mitochondrial inheritance has the following characteristics:
- inheritance is only via the maternal line as the sperm contributes no cytoplasm to the zygote
- none of the children of an affected male will inherit the disease
- all of the children of an affected female will inherit the disease
- generally, encode rare neurological diseases
- poor genotype:phenotype correlation - within a tissue or cell there can be different mitochondrial populations - this is known as heteroplasmy

Histology
- muscle biopsy classically shows 'red, ragged fibres' due to increased number of mitochondria

Examples include:
- Leber's optic atrophy
    - symptoms typically develop at around the age of 30 years

    - central scotoma → loss of colour vision → rapid onset of significant visual impairment
- MELAS syndrome: mitochondrial encephalomyopathy lactic acidosis and stroke-like episodes
- MERRF syndrome: myoclonus epilepsy with ragged-red fibres
- Kearns-Sayre syndrome: onset in patients < 20 years old, external ophthalmoplegia, retinitis pigmentosa. Ptosis may be seen
- sensorineural hearing loss

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 5 👎 6

2013 How to spot mitochondrial disease in adults

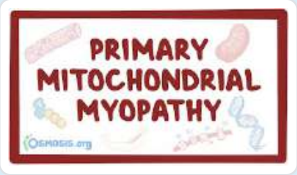
Rare Disease Video Library - NORD 👍 3 👎 4

Primary Mitochondrial Myopathy

Suggest link

Report broken link

Media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Primary mitochondrial myopathy

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A 45 year old male presented with right shoulder pain and weakness. Three weeks ago he had been brought to the emergency department (ER) with the same complaint. The pain was so severe that it awoke him from sleep. At that time he took two paracetamol tablets but that gave him no relief. Eventually the ER team had been forced to give him morphine to relieve his agony having tried less potent analgesia. On further questioning he described the pain as sharp, exacerbated by movement of the shoulder, with numbness at the shoulder tip.

On examination, he was holding his right arm with his left one in adduction and internal rotation. Wasting of the deltoid muscle had been noted. The biceps tendon jerk was absent. Sensations were intact except in a small region over deltoid muscle. Lower limb examination was normal. One month ago he had a flu. The past medical history was otherwise of no significance.

He admitted to drinking alcohol at least four times a week. He has a ten year old son who is an insulin dependent diabetic.

The following investigations were ordered:

Hb	14 g/dl
Platelets	180 * 10^9/l
WBC	6 * 10^9/l
MCV	85 fl
MCH	0.6 fmol/cell
MCHC	21 mmol/l
Na+	135 mmol/l
K+	4 mmol/l
Creatinine	85 µmol/l
Urea	3.2 mmol/l
ESR	4 mm/hr
ANA	negative

What is the most likely diagnosis?

○ Syringomyelia

○ Brachial neuritis ×

○ Adhesive capsulitis ×

○ Polymyalgia rheumatica ×

○ Cervical radiculopathy ×

Submit answer

Reference ranges ⌵

Score: **24.8%**

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An 18-year-old man presents to the emergency department with pleuritic chest pain. He has no past medical history.

The examination is unremarkable.

A chest x-ray reveals a visible pleural edge with a distance of approximately 1.1cm from the pleural edge to the lung margin at the level of the hilum.

Given the likely diagnosis, what advice should he be given about flying?

He can fly 1 week post treatment with a chest x-ray demonstrating resolution	70%
He can fly 6 weeks post treatment with a chest x-ray demonstrating resolution	22%
He can fly immediately upon clinical improvement	3%
Indefinite ban on flying	3%
There is no contraindication to flying at any point	3%

Pneumothorax -> can fly 1 week after treatment if chest x-ray shows resolution

Important for me Less important

He can fly 1-week post-treatment with a chest x-ray demonstrating resolution is the correct answer. The patient has a small PTX < 2cm without any lung disease history. He is likely to be suitable for discharge and outpatient review. He can be advised that he can fly one week post any required treatment if a chest x-ray demonstrates resolution.

He can fly 6 weeks post-treatment with a chest x-ray demonstrating resolution is incorrect. This was previous BTS advice but has now been replaced by the above advice.

He can fly immediately upon clinical improvement is incorrect. We need to see chest x-ray evidence of resolution prior to flying. This would be more suitable advice if he had a pneumonia.

Indefinite ban on flying is not correct. A prior history of resolved PTX is not a contraindication to flying.

There is no contraindication to flying at any point is incorrect. An active, unresolved PTX is an absolute contraindication to air travel.

👍 👎 Discuss (1) Improve

Next question >

Fitness to fly

The Civil Aviation Authority (CAA) has issued guidelines on air travel for people with medical conditions; please see the link provided.

Cardiovascular disease

- unstable angina, uncontrolled hypertension, uncontrolled cardiac arrhythmia, decompensated heart failure, severe symptomatic valvular disease: should not fly
- uncomplicated myocardial infarction: may fly after 7-10 days
- complicated myocardial infarction: after 4-6 weeks
- coronary artery bypass graft: after 10-14 days
- percutaneous coronary intervention: after 3 days
- stroke: patients are advised to wait 10 days following an event, although if stable may be carried within 3 days of the event

Respiratory disease

- pneumonia: should be 'clinically improved with no residual infection'
- pneumothorax: absolute contraindication, the CAA suggest patients may travel 2 weeks after successful drainage if there is no residual air. The British Thoracic Society used to recommend not travelling by air for a period of 6 weeks but this has now been changed to 1 week post check x-ray

Pregnancy

- most airlines do not allow travel after 36 weeks for a single pregnancy and after 32 weeks for a multiple pregnancy
- most airlines require a certificate after 28 weeks confirming that the pregnancy is progressing normally

Surgery

- travel should be avoided for 10 days following abdominal surgery
- laparoscopic surgery: after 24 hours
- colonoscopy: after 24 hours

- following the application of a plaster cast, the majority of airlines restrict flying for 24 hours on flights of less than 2 hours or 48 hours for longer flights

Haematological disorders

- patients with a haemoglobin of greater than 8 g/dl may travel without problems (assuming there is no coexisting condition such as cardiovascular or respiratory disease)

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Civil Aviation Authority 👍 18 👎 7

Fitness to fly guidelines

Suggest link Report broken link

Score: **20.8%**

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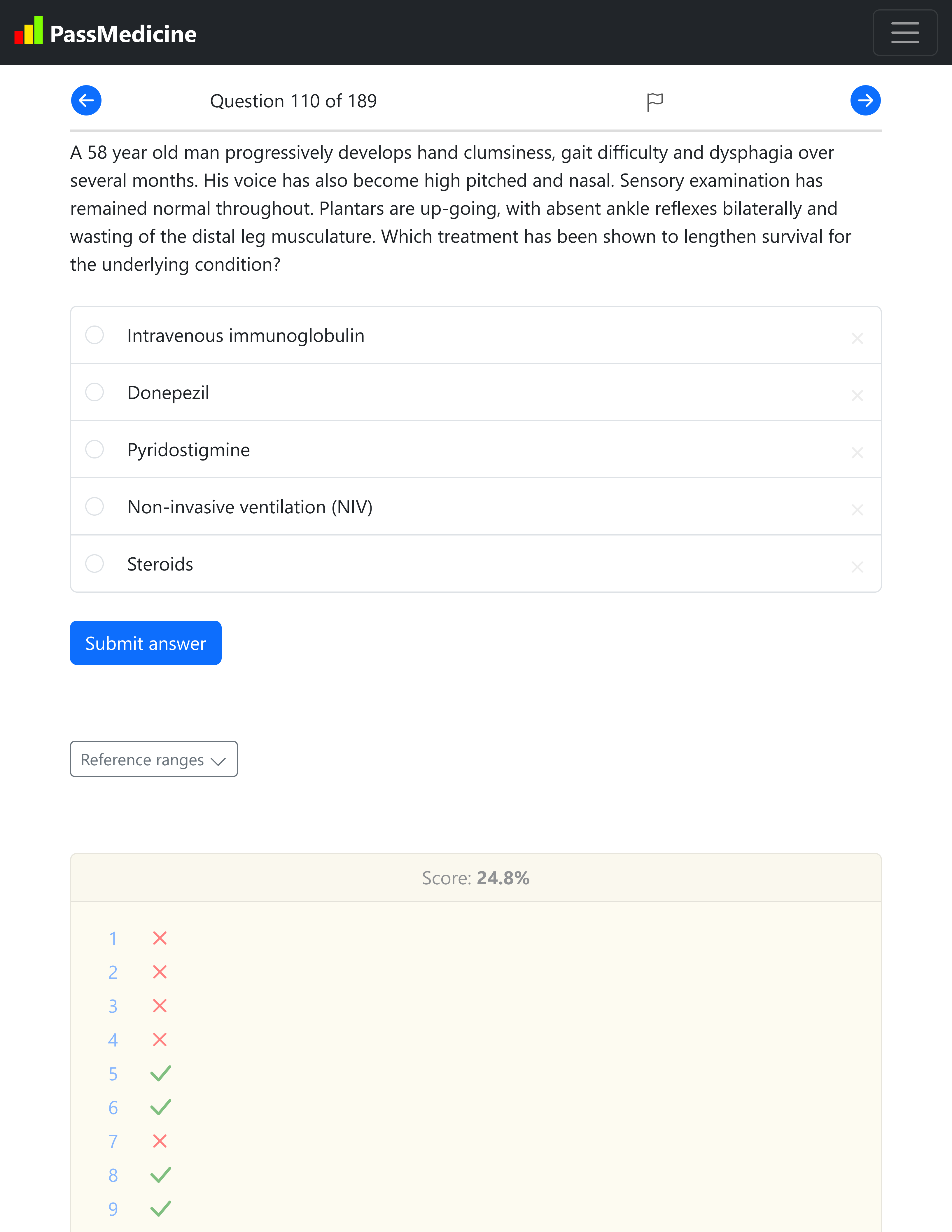

A 58 year old man progressively develops hand clumsiness, gait difficulty and dysphagia over several months. His voice has also become high pitched and nasal. Sensory examination has remained normal throughout. Plantars are up-going, with absent ankle reflexes bilaterally and wasting of the distal leg musculature. Which treatment has been shown to lengthen survival for the underlying condition?

- ○ Intravenous immunoglobulin
- ○ Donepezil
- ○ Pyridostigmine
- ○ Non-invasive ventilation (NIV)
- ○ Steroids

Score: **24.8%**

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Na+	135 mmol/l
K+	4 mmol/l
Creatinine	85 µmol/l
Urea	3.2 mmol/l
ESR	4 mm/hr
ANA	negative

What is the most likely diagnosis?

Syringomyelia 4%

Brachial neuritis	72%
Adhesive capsulitis	11%
Polymyalgia rheumatica	1%
Cervical radiculopathy	13%

Adhesive capsulitis usually presents with shoulder pain and restrictions of movement. It would not cause wasting nor neither lower motor neurological signs. Adhesive capsulitis is usually associated with diabetes and thyroid disorders.

Discuss (8) Improve

Next question

Brachial neuritis

Brachial neuritis is characterized by acute onset of unilateral (occasionally bilateral) severe pain, followed by shoulder and scapular weakness several days later. Sensory changes are usually minimal. There may be subsequent rapid wasting of the arm muscles in accordance to which nerve is involved. Precipitating factors include recent trauma, infection, surgery, or even vaccination. Rarely it may be hereditary. The prognosis is usually good except when the phrenic nerve is involved since this can result in significant breathlessness.

AI + Q 123

Next question

B I A

Textbooks

High-yield textbook

Extended textbook

Score: **20.8%**

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Question 110 of 189

A 58 year old man progressively develops hand clumsiness, gait difficulty and dysphagia over several months. His voice has also become high pitched and nasal. Sensory examination has remained normal throughout. Plantars are up-going, with absent ankle reflexes bilaterally and wasting of the distal leg musculature. Which treatment has been shown to lengthen survival for the underlying condition?

- ○ Intravenous immunoglobulin ×
- ○ Donepezil ×
- ○ Pyridostigmine ×
- ○ Non-invasive ventilation (NIV) ×
- ○ Steroids ×

Submit answer

Reference ranges

Score: **24.8%**

1 ✗
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9 ✓

10	✗
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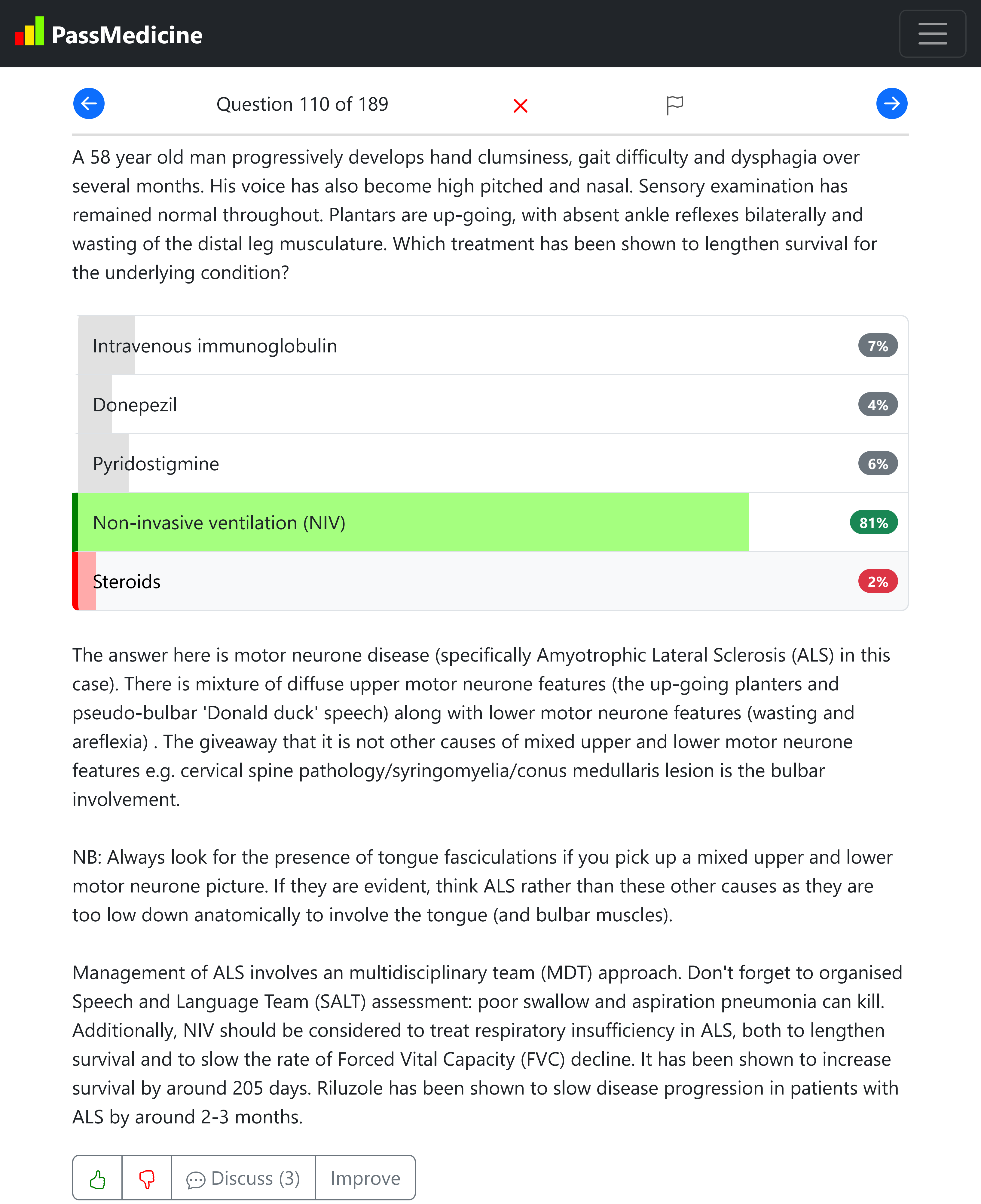

A 58 year old man progressively develops hand clumsiness, gait difficulty and dysphagia over several months. His voice has also become high pitched and nasal. Sensory examination has remained normal throughout. Plantars are up-going, with absent ankle reflexes bilaterally and wasting of the distal leg musculature. Which treatment has been shown to lengthen survival for the underlying condition?

Intravenous immunoglobulin	7%
Donepezil	4%
Pyridostigmine	6%
Non-invasive ventilation (NIV)	81%
Steroids	2%

The answer here is motor neurone disease (specifically Amyotrophic Lateral Sclerosis (ALS) in this case). There is mixture of diffuse upper motor neurone features (the up-going planters and pseudo-bulbar 'Donald duck' speech) along with lower motor neurone features (wasting and areflexia) . The giveaway that it is not other causes of mixed upper and lower motor neurone features e.g. cervical spine pathology/syringomyelia/conus medullaris lesion is the bulbar involvement.

NB: Always look for the presence of tongue fasciculations if you pick up a mixed upper and lower motor neurone picture. If they are evident, think ALS rather than these other causes as they are too low down anatomically to involve the tongue (and bulbar muscles).

Management of ALS involves an multidisciplinary team (MDT) approach. Don't forget to organised Speech and Language Team (SALT) assessment: poor swallow and aspiration pneumonia can kill. Additionally, NIV should be considered to treat respiratory insufficiency in ALS, both to lengthen survival and to slow the rate of Forced Vital Capacity (FVC) decline. It has been shown to increase survival by around 205 days. Riluzole has been shown to slow disease progression in patients with ALS by around 2-3 months.

Next question >

Motor neuron disease: management ★

Motor neuron disease is a neurological condition of unknown cause which can present with both upper and lower motor neuron signs. It rarely presents before 40 years and various patterns of disease are recognised including amyotrophic lateral sclerosis, progressive muscular atrophy and bulbar palsy

Riluzole
- prevents stimulation of glutamate receptors
- used mainly in amyotrophic lateral sclerosis
- prolongs life by about 3 months

Respiratory care
- non-invasive ventilation (usually BIPAP) is used at night
- studies have shown a survival benefit of around 7 months

Nutrition
- percutaneous gastrostomy tube (PEG) is the preferred way to support nutrition and has been associated with prolonged survival

Prognosis
- poor: 50% of patients die within 3 years

AI + Q 123

Next question >

B *I* A T↕

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 6 👎 5

2016 Motor neurone disease: assessment and management

Suggest link

Report broken link

Media

SPEECH
TRUNK
MOTOR NEURONE
DISEASE
LIMBS
TONGUE WASTING
BRISK REFLEXES

Motor neuron disease

Zero To Finals - YouTube 👍 0 👎 0

Report broken media

Score: **20.8%**

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Question 111 of 189

A 41 year old lady with a background of vitiligo and pernicious anaemia presents with weakness largely affecting her shoulders and hip muscles. The problem is variable throughout the day dependent on how much activity she has done. She does feel that symptoms are at their worst at the end of the day, when indeed her family also say her voice becomes quieter. Clinical examination demonstrates proximal muscle weakness after repeated testing. Electromyography demonstrates a decline in the amplitude of successive potentials (decremental response). Antibodies to the acetylcholine receptor (AChR) are negative on serum testing. Her CT thorax is normal. Which of the following is likely to be most helpful in establishing a diagnosis?

- ○ Anti-smooth muscle antibodies ×
- ○ Anti-muscle specific kinase (MuSK) antibodies ×
- ○ Anti-striated muscle antibody ×
- ○ Anticardiolipin antibodies ×
- ○ Anti-transglutaminase antibodies ×

Submit answer

Reference ranges

Score: **24.8%**

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A 41 year old lady with a background of vitiligo and pernicious anaemia presents with weakness largely affecting her shoulders and hip muscles. The problem is variable throughout the day dependent on how much activity she has done. She does feel that symptoms are at their worst at the end of the day, when indeed her family also say her voice becomes quieter. Clinical examination demonstrates proximal muscle weakness after repeated testing. Electromyography demonstrates a decline in the amplitude of successive potentials (decremental response). Antibodies to the acetylcholine receptor (AChR) are negative on serum testing. Her CT thorax is normal. Which of the following is likely to be most helpful in establishing a diagnosis?

Anti-smooth muscle antibodies	3%
Anti-muscle specific kinase (MuSK) antibodies	93%
Anti-striated muscle antibody	2%
Anticardiolipin antibodies	1%
Anti-transglutaminase antibodies	1%

The diagnosis here is Myasthenia Gravis (MG). Most (but not all) patients with MG are seropositive for the acetylcholine receptor (AChR) antibody. Antibodies to muscle-specific receptor tyrosine kinase (MuSK), a surface membrane component essential in the development of the neuromuscular junction, have recently been identified and are found in up to 50% of MG patients who are seronegative for AChR antibodies. Emerging data suggests that the patterns of weakness and response to certain treatments may be different from those with AChR positive MG. Anti-striated muscle antibody usually positive in MG patients that have a thymoma.

Don't forget to recognise the autoimmune past medical history in this patient. It often gives useful diagnostic clues that the underlying condition is also an autoimmune one.

Ref: www.myasthenia.org/healthprofessionals/clinicaloverviewofmg.aspx#sthash.dCo1qtr5.dpuf

Discuss (2) Improve

Next question

Myasthenia gravis ★

Myasthenia gravis is an autoimmune disorder resulting in insufficient functioning acetylcholine receptors. Antibodies to acetylcholine receptors are seen in 85-90% of cases*. Myasthenia is more common in women (2:1)

The key feature is muscle fatigability - muscles become progressively weaker during periods of activity and slowly improve after periods of rest:

- extraocular muscle weakness: diplopia
- proximal muscle weakness: face, neck, limb girdle
- ptosis
- dysphagia

Associations

- thymomas in 15%
- autoimmune disorders: pernicious anaemia, autoimmune thyroid disorders, rheumatoid, SLE
- thymic hyperplasia in 50-70%

Investigations

- single fibre electromyography: high sensitivity (92-100%)
- CT thorax to exclude thymoma
- CK normal
- antibodies to acetylcholine receptors
    - positive in around 85-90% of patients
    - n the remaining patients, about about 40% are positive for anti-muscle-specific tyrosine kinase antibodies
- Tensilon test: IV edrophonium reduces muscle weakness temporarily - not commonly used any more due to the risk of cardiac arrhythmia

Management

- long-acting acetylcholinesterase inhibitors
    - pyridostigmine is first-line
- immunosuppression is usually not started at diagnosis, but the majority of patients eventually require it in addition to long-acting acetylcholinesterase inhibitors:
    - prednisolone initially
    - azathioprine, cyclosporine, mycophenolate mofetil may also be used
- thymectomy

Management of myasthenic crisis

- plasmapheresis
- intravenous immunoglobulins

*antibodies are less commonly seen in disease limited to the ocular muscles

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

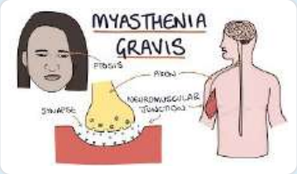
Postgraduate Medical Journal 13 3

Review of myasthenia gravis

Suggest link

Report broken link

Media


MYASTHENIA GRAVIS
PTOSIS
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NEUROMUSCULAR JUNCTION
SYNAPSE

Myasthenia gravis

Zero to Finals - YouTube 12 1

NEUROLOGY
MYASTHENIA GRAVIS

Myasthenia gravis

Armando Hasudungan - YouTube 👍 1 👎 1

MYASTHENIA GRAVIS

Myasthenia gravis - causes, symptoms, treatment, pathology

Osmosis - YouTube 👍 2 👎 2

Report broken media

Score: **20.8%**

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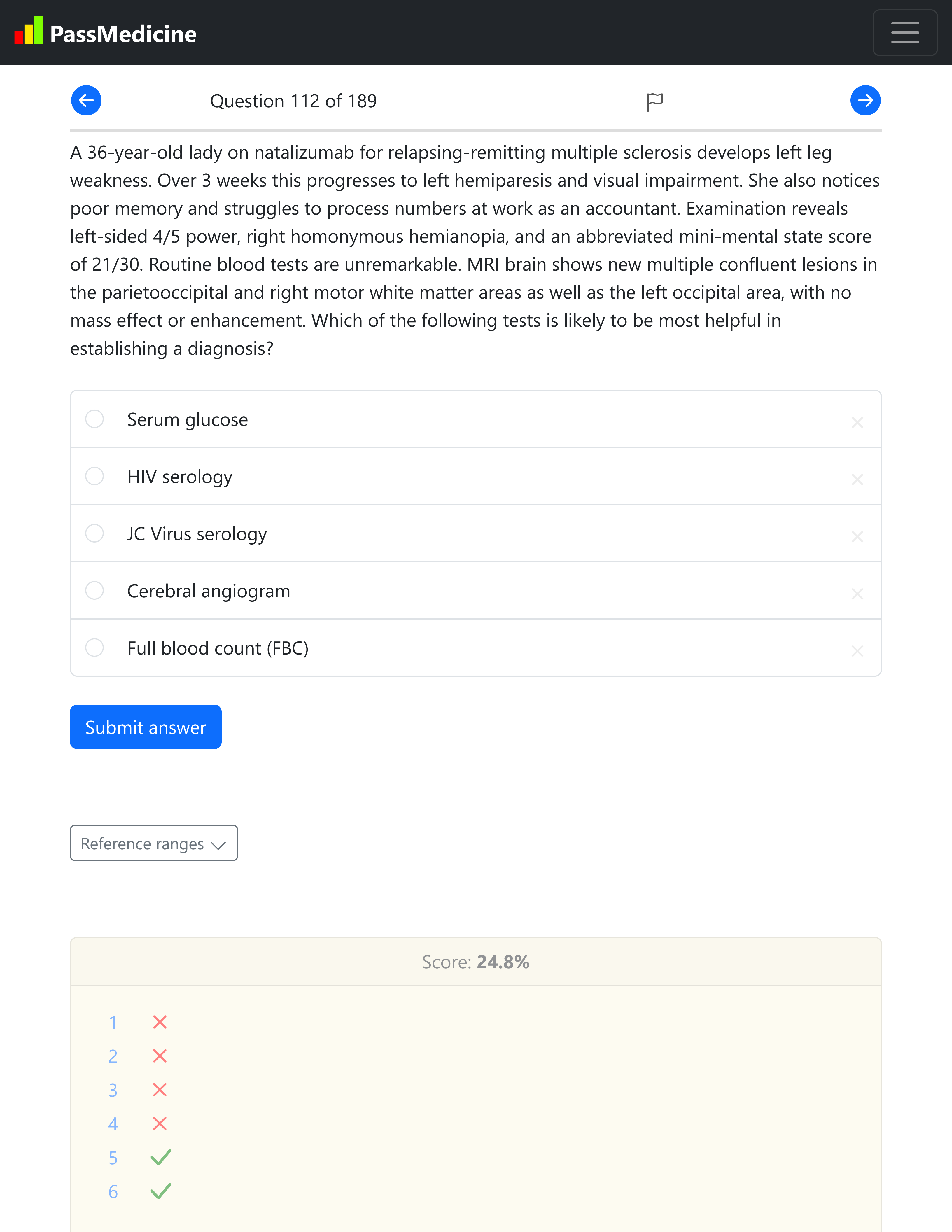

Question 112 of 189

A 36-year-old lady on natalizumab for relapsing-remitting multiple sclerosis develops left leg weakness. Over 3 weeks this progresses to left hemiparesis and visual impairment. She also notices poor memory and struggles to process numbers at work as an accountant. Examination reveals left-sided 4/5 power, right homonymous hemianopia, and an abbreviated mini-mental state score of 21/30. Routine blood tests are unremarkable. MRI brain shows new multiple confluent lesions in the parietooccipital and right motor white matter areas as well as the left occipital area, with no mass effect or enhancement. Which of the following tests is likely to be most helpful in establishing a diagnosis?

- ○ Serum glucose
- ○ HIV serology
- ○ JC Virus serology
- ○ Cerebral angiogram
- ○ Full blood count (FBC)

Score: **24.8%**

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A 36-year-old lady on natalizumab for relapsing-remitting multiple sclerosis develops left leg weakness. Over 3 weeks this progresses to left hemiparesis and visual impairment. She also notices poor memory and struggles to process numbers at work as an accountant. Examination reveals left-sided 4/5 power, right homonymous hemianopia, and an abbreviated mini-mental state score of 21/30. Routine blood tests are unremarkable. MRI brain shows new multiple confluent lesions in the parietooccipital and right motor white matter areas as well as the left occipital area, with no mass effect or enhancement. Which of the following tests is likely to be most helpful in establishing a diagnosis?

Serum glucose	1%
HIV serology	3%
JC Virus serology	90%
Cerebral angiogram	5%
Full blood count (FBC)	2%

The diagnosis here is progressive multifocal leukoencephalopathy (PML). This is a demyelinating disease of the CNS characterised by widespread lesions due to brain infection of oligodendrocytes by the JC Virus (JCV). These are visible on an MRI and have a characteristic appearance. The signs correspond to the areas occupied by the lesions. PML occurs almost exclusively in immunocompromised patients such as those with HIV or on immunotherapy. You should consider PML in your differential in such patients when they present with new odd neurology and an abnormal MRI scan.

This question's other aim is to introduce to you one of the newer therapies in multiple sclerosis (MS) called nataluzumab. It is actually very frequently used in MS and indeed now approved for the treatment of relapsing-remitting MS (RRMS) in more than 50 countries. It is a monoclonal antibody that inhibits the migration of leucocytes into the central nervous system, hence reducing inflammation and demyelination. It is given as an IV infusion once a month. It is indicated in patients getting relapses despite treatment with interferon-beta or patients who just have rapidly evolving severe RRMS. It has been shown to decrease the risk of progression of disability by 42-54% and the annualised rate of relapse by 68%. The two principle side-effects are hepatotoxicity and increased PML risk, and we monitor patients for these with monthly liver function tests (LFTs) and 6-monthly JCV serology. It would not be unreasonable of them to expect you to know something about nataluzumab in the exam (especially to recognise PML as a side-effect) given that the drug is now in such common use.

👍 👎 Discuss (8) Improve

Next question >

Multiple sclerosis: management ★

Treatment in multiple sclerosis is focused on reducing the frequency and duration of relapses. There is no cure.

Acute relapse

High-dose steroids (e.g. oral or IV methylprednisolone) may be given for 5 days to shorten the length of an acute relapse. It should be noted that steroids shorten the duration of a relapse and do not alter the degree of recovery (i.e. whether a patient returns to baseline function)

Disease modifying drugs

A number of drugs have been shown to reduce the risk of relapse in patients with MS. Typical indications for disease-modifying drugs include:

- relapsing-remitting disease + 2 relapses in past 2 years + able to walk 100m unaided
- secondary progressive disease + 2 relapses in past 2 years + able to walk 10m (aided or unaided)

Drug options for reducing the risk of relapse in MS

- natalizumab
    - a recombinant monoclonal antibody that antagonises alpha-4 beta-1-integrin found on the surface of leucocytes
    - inhibit migration of leucocytes across the endothelium across the blood-brain barrier
    - generally considered to have the strongest evidence base for preventing relapse of the disease-modifying and hence is often used first-line
    - given intravenously
- ocrelizumab
    - humanized anti-CD20 monoclonal antibody
    - like natalizumab, it is considered a high-efficacy drug that is often used first-line
    - given intravenously
- fingolimod

    - sphingosine 1-phosphate (S1P) receptor modulator
    - prevents lymphocytes from leaving lymph nodes
    - oral formulations are available
- beta-interferon
    - not considered to be as effective as alternative disease-modifying drugs
    - given subcutaneously/intramuscularly
- glatiramer acetate
    - immunomodulating drug - acts as an 'immune decoy'
    - given subcutaneously
    - along with beta-interferon considered an 'older drug' with less effectiveness compared to monoclonal antibodies and S1P) receptor modulators

Some specific problems

Fatigue

- once other problems (e.g. anaemia, thyroid or depression) have been excluded NICE recommend a trial of amantadine
- other options include mindfulness training and CBT

Spasticity

- baclofen and gabapentin are first-line. Other options include diazepam, dantrolene and tizanidine
- physiotherapy is important
- cannabis and botox are undergoing evaluation

Bladder dysfunction

- may take the form of urgency, incontinence, overflow etc
- guidelines stress the importance of getting an ultrasound first to assess bladder emptying - anticholinergics may worsen symptoms in some patients
- if significant residual volume → intermittent self-catheterisation
- if no significant residual volume → anticholinergics may improve urinary frequency

Oscillopsia (visual fields appear to oscillate)

- gabapentin is first-line

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

Multiple Sclerosis Society 👍 6 👎 15

Primary care guidelines

NICE 👍 15 👎 7

2014 Multiple Sclerosis guidelines

Suggest link Report broken link

Media

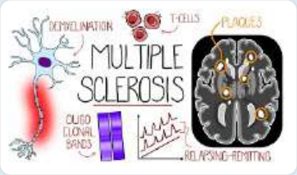
Multiple sclerosis

Podmedics - YouTube 👍 4 👎 0

Multiple Sclerosis

Multiple sclerosis

Soton Brain Hub - YouTube 👍 3 👎 0


DEMYELINATION
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MULTIPLE SCLEROSIS
OLIGO CLONAL BANDS
RELAPSING-REMITTING

Multiple sclerosis

Rhesus Medicine - YouTube 👍 1 👎 0

MULTIPLE SCLEROSIS

Multiple sclerosis - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 7 👎 1

Report broken media

Score: **20.8%**

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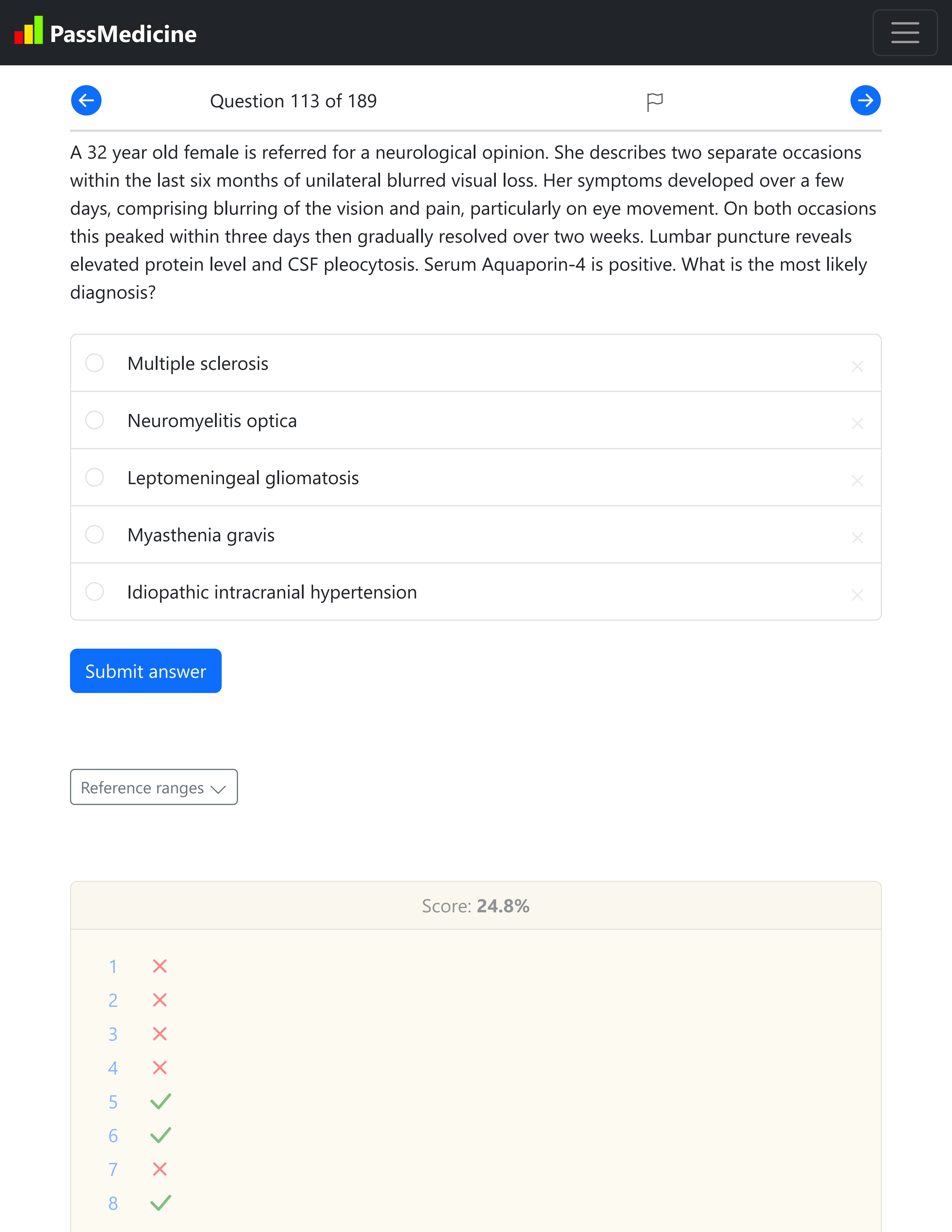

Question 113 of 189

A 32 year old female is referred for a neurological opinion. She describes two separate occasions within the last six months of unilateral blurred visual loss. Her symptoms developed over a few days, comprising blurring of the vision and pain, particularly on eye movement. On both occasions this peaked within three days then gradually resolved over two weeks. Lumbar puncture reveals elevated protein level and CSF pleocytosis. Serum Aquaporin-4 is positive. What is the most likely diagnosis?

- ○ Multiple sclerosis
- ○ Neuromyelitis optica
- ○ Leptomeningeal gliomatosis
- ○ Myasthenia gravis
- ○ Idiopathic intracranial hypertension

Score: **24.8%**

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A 32 year old female is referred for a neurological opinion. She describes two separate occasions within the last six months of unilateral blurred visual loss. Her symptoms developed over a few days, comprising blurring of the vision and pain, particularly on eye movement. On both occasions this peaked within three days then gradually resolved over two weeks. Lumbar puncture reveals elevated protein level and CSF pleocytosis. Serum Aquaporin-4 is positive. What is the most likely diagnosis?

Multiple sclerosis	7%
Neuromyelitis optica	90%
Leptomeningeal gliomatosis	1%
Myasthenia gravis	1%
Idiopathic intracranial hypertension	1%

Her symptoms are highly suggestive of Neuromyelitis optica (NMO, also known as Devic disease). NMO causes demyelination of the optic nerves or spinal cord due to autoimmune inflammation. Aquaporin-4 (AQP-4) is a water channel protein located in the astrocytic foot processes; NMO-IgG antibody attacks AQP-4 causing demyelination.

NMO typically presents with episodes of optic neuritis or transverse myelitis. The optic neuritis can be clinically indistinguishable from that seen in multiple sclerosis (MS), whereas the transverse myelitis is commonly more of a complete picture than that of MS. This is a result of the fact that the resulting spinal cord demyelination seen in NMO is longer and more extensive.

Discuss (8) | Improve

Next question >

Neuromyelitis optica ★

Neuromyelitis optica (NMO) is a monophasic or relapsing-remitting demyelinating CNS disorder Although previously thought to be a variant of multiple sclerosis, it is now recognised to be a distinct disease, particularly prevalent in Asian populations. It typically involves the optic nerves and cervical spine, with imaging of the brain frequently normal. Vomiting is also a common presenting complaint.

Diagnosis requires bilateral optic neuritis, myelitis and 2 of the following 3 criteria:

- 1. Spinal cord lesion involving 3 or more spinal levels
- 2. Initially normal MRI brain
- 3. Aquaporin 4 positive serum antibody

Management

- immunosuppressant e.g. with anti-CD20 agent rituximab)

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20.8%**

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A 25 year old man presents to A+E in a comatose state. He was found by his university flatmate collapsed on the floor. His flatmate states that the man had been behaving bizarrely in the last 24 hours and had been quite agitated and aggressive at times. When you examine him he has a GCS of 8 (E 2 V 1 M 5). He has a temp of 39.4ºC, heart rate 120/min, blood pressure 178/89 mmHg, sats 98% on room air and respiratory rate 20/min. Chest clear abdomen soft and non-tender, bowel sounds present. Globally greatly increased tone in all 4 limbs.

When looking at his previous electronic notes the only information that you can find is a recent admission to a psychiatric hospital where a diagnosis of paranoid schizophrenia was established.

CT Brain: normal

Hb	15.4 g/dl
Platelets	232 * 10^9/l
WBC	11.5 * 10^9/l

Na^+	143 mmol/l
K^+	4.1 mmol/l
Urea	8.1 mmol/l
Creatinine	101 µmol/l

Bilirubin	14 µmol/l
ALP	63 u/l
ALT	28 u/l
Calcium	2.64 mmol/l
Albumin	41 g/l
Creatine kinase	21,000 iu/l
Serum glucose	6.4 mmol/l

Lumbar Puncture:

Glucose	4.9 mmol/l
Protein	0.3g/l

Culture	nil organisms found
Opening pressure	21 mmHg

What is the most likely diagnosis?

- ○ Viral meningitis
- ○ Serotonin syndrome
- ○ Neuroleptic malignant syndrome
- ○ Bacterial meningitis
- ○ Malignant hyperthermia

Submit answer

Reference ranges

Score: **24.8%**

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Serum glucose	6.4 mmol/l

Lumbar Puncture:

Glucose	4.9 mmol/l
Protein	0.3g/l

Culture	nil organisms found
Opening pressure	21 mmHg

What is the most likely diagnosis?

Viral meningitis	1%
Serotonin syndrome	11%
Neuroleptic malignant syndrome	83%
Bacterial meningitis	1%
Malignant hyperthermia	4%

The answer is neuroleptic malignant syndrome. NMS is characterised by a triad of altered conscious state, hyperpyrexia and raised CK in a person on neuroleptic medications. Young males recently started on a high dose of a high potency antipsychotic are most at risk from this potentially fatal complication. Autonomic instability is also a feature.

Serotonin syndrome shares many clinical features with NMS but normally you would expect to see GI features and cerebellar symptoms in addition and rigidity and hyperthermia are usually more mild. In addition serotonin syndrome is associated with antidepressant medication. This is therefore a less likely diagnosis in an individual recently diagnosed with schizophrenia.

Malignant hyperthermia also shares many clinical features with NMS but this is a genetic disorder provoked by the use of anaesethic agents and there is nothing in the history to suggest this. A normal Lumbar Puncture MC+S essentially rules out bacterial meningitis and viral meningitis is unlikely to cause such severe symptoms

👍 👎 Discuss (2) Improve

Next question >

Neuroleptic malignant syndrome ★

Neuroleptic malignant syndrome is a rare but dangerous condition seen in patients taking antipsychotic medication. It carries a mortality of up to 10% and can also occur with atypical antipsychotics. It may also occur with dopaminergic drugs (such as levodopa) for Parkinson's disease, usually when the drug is suddenly stopped or the dose reduced.

The pathophysiology is unknown but one theory is that the dopamine blockade induced by antipsychotics triggers massive glutamate release and subsequent neurotoxicity and muscle damage.

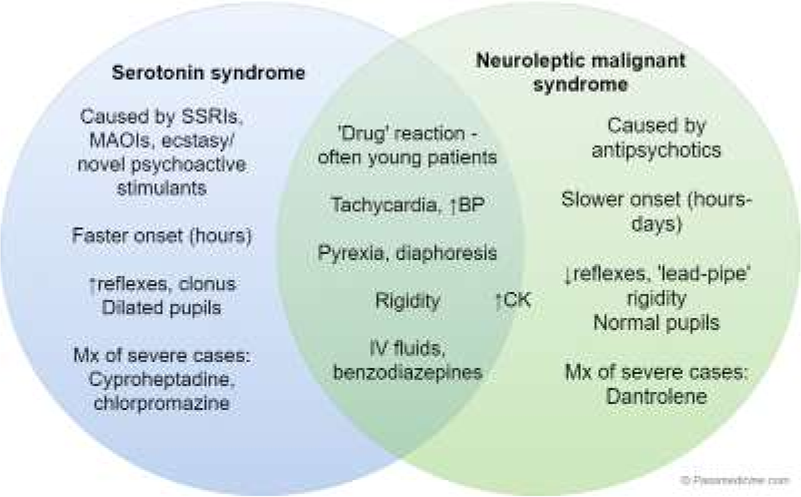
It occurs within hours to days of starting an antipsychotic (antipsychotics are also known as neuroleptics, hence the name) and the typical features are:

- pyrexia
- muscle rigidity
- autonomic lability: typical features include hypertension, tachycardia and tachypnoea
- agitated delirium with confusion

A raised creatine kinase is present in most cases. Acute kidney injury (secondary to rhabdomyolysis) may develop in severe cases. A leukocytosis may also be seen

Management

- stop antipsychotic
- patients should be transferred to a medical ward if they are on a psychiatric ward and often they are nursed in intensive care units
- IV fluids to prevent renal failure
- dantrolene may be useful in selected cases
    - thought to work by decreasing excitation-contraction coupling in skeletal muscle by binding to the ryanodine receptor, and decreasing the release of calcium from the sarcoplasmic reticulum
- bromocriptine, dopamine agonist, may also be used


Serotonin syndrome
Caused by SSRIs, MAOIs, ecstasy/ novel psychoactive stimulants
Faster onset (hours)
↑reflexes, clonus
Dilated pupils
Mx of severe cases: Cyproheptadine, chlorpromazine
Neuroleptic malignant syndrome
Caused by antipsychotics
Slower onset (hours-days)
↓reflexes, 'lead-pipe' rigidity
Normal pupils
Mx of severe cases: Dantrolene
'Drug' reaction - often young patients
Tachycardia, ↑BP
Pyrexia, diaphoresis
Rigidity
↑CK
IV fluids, benzodiazepines
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Venn diagram showing contrasting serotonin syndrome with neuroleptic malignant syndrome. Note that both conditions can cause a raised creatine kinase (CK) but it tends to be more associated with NMS.

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 8 👎 5

Neuroleptic Malignant Syndrome

Medsafe 👍 15 👎 4

Neuroleptice malignant syndrome Vs serotonin syndrome

Suggest link Report broken link

Media

DOPAMINE ANTAGONISM
AUTONOMIC INSTABILITY
NEUROLEPTIC MALIGNANT SYNDROME
FEVER
ANTIPSYCHOTICS

Neuroleptic malignant syndrome

Rhesus Medicine - YouTube 👍 1 👎 0

NEUROLEPTIC MALIGNANT SYNDROME

Neuroleptic malignant syndrome

Townsend Teaching - YouTube 👍 0 👎 0

Neuroleptic malignant syndrome

Kloss and Bruce - YouTube 👍 0 👎 1

Report broken media

Score: **20.8%**

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A 68-year-old female is admitted into the acute medical unit after suffering from a prolonged tonic-clonic seizure. She was diagnosed with status epilepticus which was terminated with lorazepam and a phenytoin infusion. Two days into the admission you are asked to review her due to ongoing confusion. She has a past medical history of depression which is currently treated with fluoxetine.

On examination of her cardiorespiratory system there is nothing of note. Her abdomen is soft and non tender. You note rapid blinking, which the nursing staff confirmed has been on-going over the past 24 hours.

Investigation results are as follows:

Hb	135 g/l	Na^+	137 mmol/l	Bilirubin	14 µmol/l
CRP	<4 mg/l	Lactate	1.2 mmol/l	Albumin	32 g/l
Platelets	385 * 10^9/l	K^+	4.2 mmol/l	ALP	88 u/l
WBC	6.6 * 10^9/l	Urea	4.4 mmol/l	ALT	44 u/l
Neuts	4.2 * 10^9/l	Creatinine	75 µmol/l	γGT	68 u/l
Lymphs	2.2 * 10^9/l				
Eosin	0.2 * 10^9/l				

What is the most likely diagnosis?

- ○ Non-Convulsive status epilepticus
- ○ Psychogenic seizure
- ○ Tardive dyskinesia
- ○ Convulsive status epilepticus
- ○ Delirium

Submit answer

Reference ranges

Score: **24.8%**

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Lymphs	2.2 * 10^9/l				
Eosin	0.2 * 10^9/l				

What is the most likely diagnosis?

Non-Convulsive status epilepticus	49%
Psychogenic seizure	12%
Tardive dyskinesia	32%
Convulsive status epilepticus	5%
Delirium	2%

Non-convulsive status epilepticus can present with subtle signs, such as twitching or blinking and/or fluctuating mental status

Important for me Less important

Non-convulsive status epilepticus (NCSE) is defined as a prolonged (>30 mins) electrographic seizure activity with non-convulsive signs such as twitching or blinking. It can be a challenging diagnosis to make which often relies on EEG confirmation. It requires a high index of suspicion in individuals with risk factors and suggestive clinical features. Early recognition and treatment are essential to optimize response to treatment and to prevent neurological and systemic sequelae.

NCSE is very common in patients who have suffered convulsive status epilepticus, comatosed patients and in the general ICU population.

In this case, the prolonged post-ictal confusion and subtle motor signs are very suggestive of NCSE, and an urgent EEG should be arranged.

There is nothing on examination to suggest infection, and this is reassured with the normal inflammatory markers.

Given the history of depression, a reasonable differential includes a psychogenic seizure, however NCSE would be more likely and more important to rule out.

Tardive dyskinesia can cause blepharospasm however it is rarely caused by SSRIs.

👍 👎 Discuss (9) Improve

Next question >

Status epilepticus ★

Status epilepticus is defined as:
- a single seizure lasting >5 minutes, or
- >= 2 seizures within a 5-minute period without the person returning to normal between them

This is a medical emergency. The priority is the termination of seizure activity, which if prolonged will lead to irreversible brain damage.

Management
- ABC
    - airway adjunct

    - oxygen
    - check blood glucose
- First-line drugs are benzodiazepines
    - in the prehospital setting PR diazepam or buccal midazolam may be given
    - in hospital IV lorazepam is generally used. This may be repeated once after 5-10minutes
- If ongoing (or 'established') status it is appropriate to start a second-line agent such as levetiracetam, phenytoin or sodium valproate
    - NICE state 'Take into account that levetiracetam may be quicker to administer and have fewer adverse effects than the alternative options.'
- If no response ('refractory status') within 45 minutes from onset, then the best way to achieve rapid control of seizure activity is induction of general anaesthesia or phenobarbital

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 14 👎 9

2022 Epilepsy guidelines

Suggest link Report broken link

Score: **20.8%**

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Question 116 of 189

A 34-year-old female attends for review. She has a background of schizophrenia which is currently managed with quetiapine having failed treatment with olanzapine and risperidone in the past. She informs that she has on-going derogatory third person auditory hallucinations. You elect to start her on clozapine.

You perform a baseline blood tests which are as follows:

Hb	145 g/l
Platelets	320 * 10^9/l
WBC	6.8 * 10^9/l
Neutrophils	3.8 * 10^9/l

What other investigation is important to perform before starting clozapine?

- ○ Abdominal x-ray
- ○ Cardiac echo
- ○ Liver function tests
- ○ ECG
- ○ Urea and electrolytes

Submit answer

Reference ranges

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Back to top

A 34-year-old female attends for review. She has a background of schizophrenia which is currently managed with quetiapine having failed treatment with olanzapine and risperidone in the past. She informs that she has on-going derogatory third person auditory hallucinations. You elect to start her on clozapine.

You perform a baseline blood tests which are as follows:

Hb	145 g/l
Platelets	320 * 10^9/l
WBC	6.8 * 10^9/l
Neutrophils	3.8 * 10^9/l

What other investigation is important to perform before starting clozapine?

Abdominal x-ray	9%
Cardiac echo	11%
Liver function tests	23%
ECG	52%
Urea and electrolytes	5%

Clozapine can cause myocarditis. A baseline ECG should be performed before initiating treatment

Important for me Less important

Clozapine can cause myocarditis. Therefore, a baseline ECG should be performed before initiating treatment.

Clozapine requires differential white blood cell monitoring weekly for 18 weeks, then fortnightly for up to one year, and then monthly as part of the clozapine patient monitoring service.

Prolactin, lipids, glucose, and body weight should also be regularly monitored.

It is important to be aware that clozapine can cause impairment of intestinal peristalsis, potentially resulting in constipation, intestinal obstruction, faecal impaction, and paralytic ileus. However there is no need to perform an abdominal x-ray as a baseline if not clinically indicated.

Discuss (6) Improve

Next question

Clozapine

Clozapine, one of the first atypical agents to be developed, carries a significant risk of agranulocytosis and full blood count monitoring is therefore essential during treatment. For this reason, clozapine should only be used in patients resistant to other antipsychotic medication. The BNF states:

> *Clozapine should be introduced if schizophrenia is not controlled despite the sequential use of two or more antipsychotic drugs (one of which should be a second-generation antipsychotic drug), each for at least 6-8 weeks.*

Adverse effects of clozapine
- agranulocytosis (1%), neutropaenia (3%)
- reduced seizure threshold - can induce seizures in up to 3% of patients
- constipation
- myocarditis: a baseline ECG should be taken before starting treatment
- hypersalivation

Dose adjustment of clozapine might be necessary if smoking is started or stopped during treatment.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BNF 👍 4 👎 2

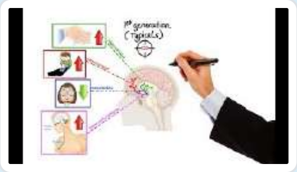
Clozapine

Clinical Knowledge Summaries 👍 3 👎 0

Schizophrenia > Prescribing Information > Adverse effects

Suggest link Report broken link

Media



Antipsychotics

Speed Pharmacology - YouTube 👍 2 👎 0

Report broken media

Score: **20.8%**

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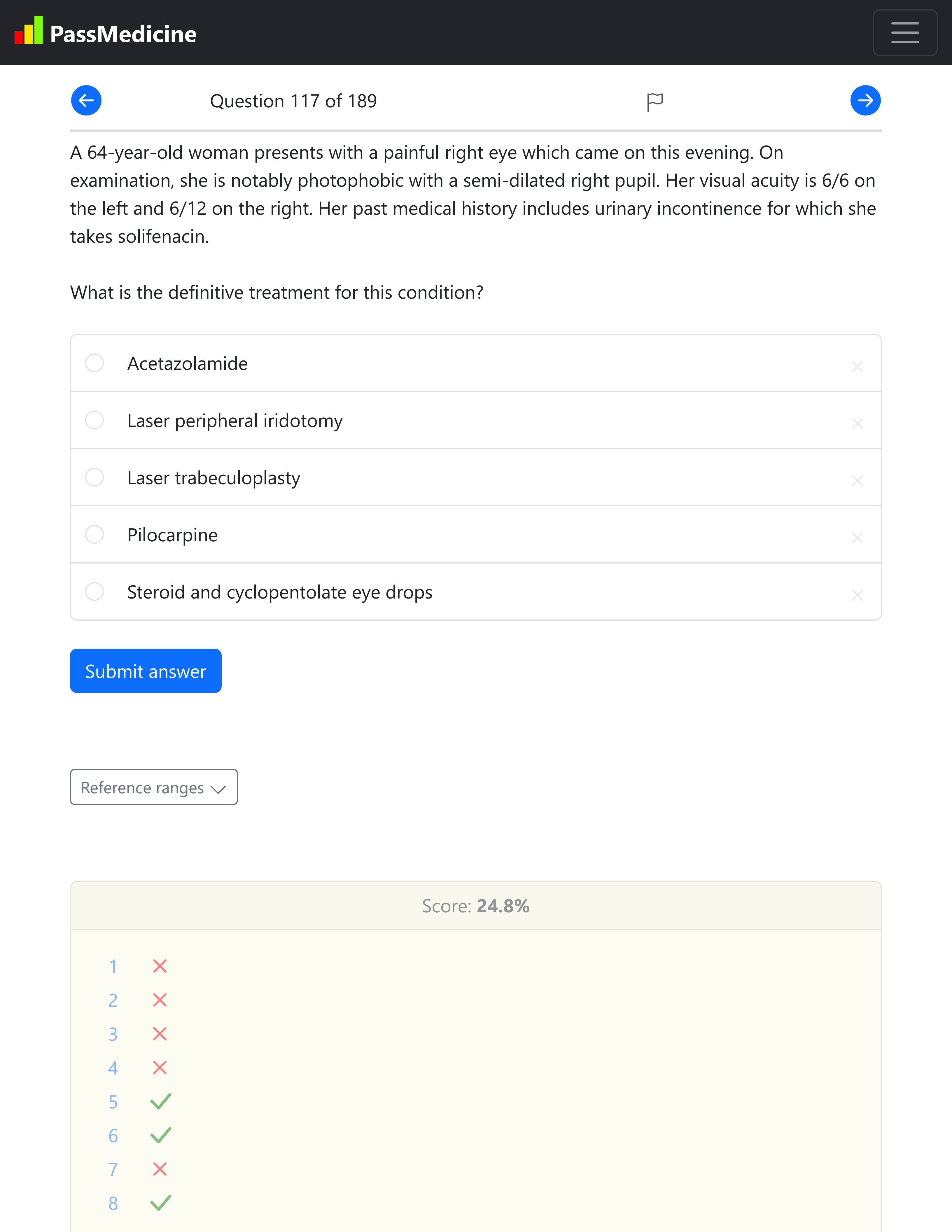

Question 117 of 189

A 64-year-old woman presents with a painful right eye which came on this evening. On examination, she is notably photophobic with a semi-dilated right pupil. Her visual acuity is 6/6 on the left and 6/12 on the right. Her past medical history includes urinary incontinence for which she takes solifenacin.

What is the definitive treatment for this condition?

- ○ Acetazolamide
- ○ Laser peripheral iridotomy
- ○ Laser trabeculoplasty
- ○ Pilocarpine
- ○ Steroid and cyclopentolate eye drops

Score: **24.8%**

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Question 117 of 189

A 64-year-old woman presents with a painful right eye which came on this evening. On examination, she is notably photophobic with a semi-dilated right pupil. Her visual acuity is 6/6 on the left and 6/12 on the right. Her past medical history includes urinary incontinence for which she takes solifenacin.

What is the definitive treatment for this condition?

Acetazolamide	13%
Laser peripheral iridotomy	56%
Laser trabeculoplasty	14%
Pilocarpine	9%
Steroid and cyclopentolate eye drops	8%

Laser peripheral iridotomy is the definitive treatment for acute angle-closure glaucoma

Important for me Less important

Laser peripheral iridotomy is correct. The clinical features are highly suggestive of acute angle closure glaucoma (AACG). The patient takes an anti-muscarinic (solifenacin) drug which can precipitate AACG. Definitive management of this condition is with laser peripheral iridotomy.

Acetazolamide is incorrect. This drug can be used in the emergency setting to lower intra-ocular pressures, however, the definitive treatment is with laser peripheral iridotomy.

Laser trabeculoplasty is incorrect. Laser trabeculoplasty is performed as an additional measure to lower the intraocular pressure in patients with open-angle glaucoma. It can also be performed in a closed-angle glaucoma patient with a patent iridotomy (e.g. following a laser peripheral iridotomy).

Pilocarpine is incorrect. This drug can be used in the emergency setting to lower intra-ocular pressures, however, the definitive treatment is with laser peripheral iridotomy.

Steroid eye drops and cyclopentolate is incorrect. This combination of medications can be used to treat anterior uveitis. Although this condition also presents with an acutely painful red eye, the presence of a semi-dilated pupil favours the diagnosis of AACG. In anterior uveitis, the pupil is classically oval and non-reactive.

Discuss (1) | Improve

Next question

Acute angle-closure glaucoma

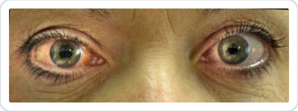
Glaucoma is a group of disorders characterised by optic neuropathy due, in the majority of patients, to raised intraocular pressure (IOP). It is now recognised that a minority of patients with raised IOP do not have glaucoma and vice versa.

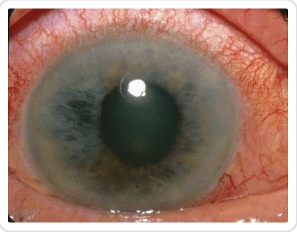
In acute angle-closure glaucoma (AACG) there is a rise in IOP secondary to an impairment of aqueous outflow. Factors predisposing to AACG include:

- hypermetropia (long-sightedness)
- pupillary dilatation
- lens growth associated with age

Features

- severe pain: may be ocular or headache
- decreased visual acuity
- symptoms worse with mydriasis (e.g. watching TV in a dark room)
- hard, red-eye
- haloes around lights
- semi-dilated non-reacting pupil
- corneal oedema results in dull or hazy cornea
- systemic upset may be seen, such as nausea and vomiting and even abdominal pain





Investigations

- tonometry to assess for elevated IOP
- gonioscopy (literally looking, *oscopy*, at the angle, *gonio*): a special lens for the slit lamp that allows visualisation of the angle

Management

The management of AACG is an emergency and should prompt urgent referral to an ophthalmologist. Emergency medical treatment is required to lower the IOP with more definitive surgical treatment given once the acute attack has settled.

There are no guidelines for the initial medical treatment emergency treatment. An example regime would be:
- combination of eye drops, for example:
  - a direct parasympathomimetic (e.g. pilocarpine, causes contraction of the ciliary muscle → opening the trabecular meshwork → increased outflow of the aqueous humour)
  - a beta-blocker (e.g. timolol, decreases aqueous humour production)
  - an alpha-2 agonist (e.g. apraclonidine, dual mechanism, decreasing aqueous humour production and increasing uveoscleral outflow)
- intravenous acetazolamide
  - reduces aqueous secretions
- some guidelines also recommend the use of topical steroids to reduce inflammation

Definitive management
- laser peripheral iridotomy
  - creates a tiny hole in the peripheral iris → aqueous humour flowing to the angle

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

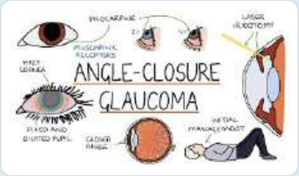
Patient.info 👍 27 👎 9

Acute angle closure glaucoma review

Suggest link

Report broken link

Media


ANGLE-CLOSURE
GLAUCOMA
PILOCARPINE
MUSCARINIC RECEPTORS
LASER IRIDOTOMY
HAZY CORNEA
FIXED AND DILATED PUPIL
INITIAL MANAGEMENT

Acute angle closure glaucoma

Zero To Finals - YouTube 👍 4 👎 0

GLAUCOMA
Osmosis.org

Glaucoma (open-angle, closed-angle, and normal-tension)

Osmosis - YouTube 👍 6 👎 3

Report broken media

Score: **20.8%**

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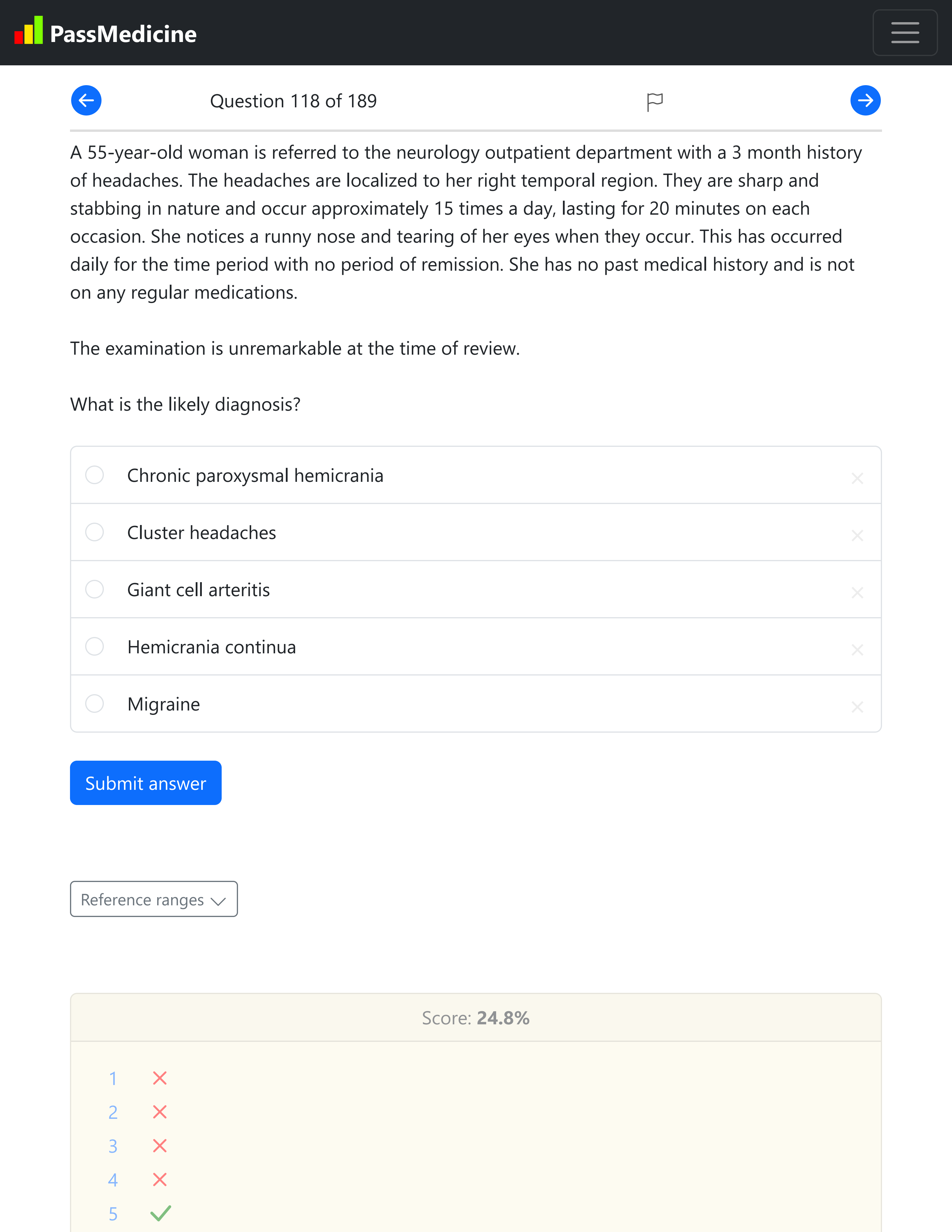
Back to top



Question 118 of 189

A 55-year-old woman is referred to the neurology outpatient department with a 3 month history of headaches. The headaches are localized to her right temporal region. They are sharp and stabbing in nature and occur approximately 15 times a day, lasting for 20 minutes on each occasion. She notices a runny nose and tearing of her eyes when they occur. This has occurred daily for the time period with no period of remission. She has no past medical history and is not on any regular medications.

The examination is unremarkable at the time of review.

What is the likely diagnosis?

- ○ Chronic paroxysmal hemicrania ×
- ○ Cluster headaches ×
- ○ Giant cell arteritis ×
- ○ Hemicrania continua ×
- ○ Migraine ×

Submit answer

Reference ranges

Score: **24.8%**

1 ✗
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Question 118 of 189 ✗

A 55-year-old woman is referred to the neurology outpatient department with a 3 month history of headaches. The headaches are localized to her right temporal region. They are sharp and stabbing in nature and occur approximately 15 times a day, lasting for 20 minutes on each occasion. She notices a runny nose and tearing of her eyes when they occur. This has occurred daily for the time period with no period of remission. She has no past medical history and is not on any regular medications.

The examination is unremarkable at the time of review.

What is the likely diagnosis?

Chronic paroxysmal hemicrania	39%
Cluster headaches	32%
Giant cell arteritis	1%
Hemicrania continua	27%
Migraine	1%

Chronic paroxysmal hemicrania is characterised by multiple (5+) short (5-30 min) headaches centered around the eye. CPH headaches are shorter and more frequent than cluster headaches

Important for me Less important

Chronic paroxysmal hemicrania is the correct answer. This headache disorder is characterized by multiple (5+) short (5-30 minutes) headaches localized to the orbital, supraorbital or temporal region that are associated with ipsilateral autonomic features (rhinorrhoea, lacrimation).

Giant cell arteritis is incorrect. This typically causes a persistent rather than intermittent headache and is associated with scalp tenderness and other clinical features such as jaw claudication and visual obscuration.

Migraine is incorrect. Migraines usually last from 4-72 hours and occur with less frequency than chronic paroxysmal hemicrania. There may be an aura that precedes the headaches.

Cluster headaches is incorrect. While this can also cause unilateral headaches with ipsilateral autonomic features, the headaches are typically longer (usually 1-3 hours) and less frequent (usually twice a day and rarely more than eight a day).

Hemicrania continua is incorrect. This is a continuous unilateral headache characterized by fluctuation in severity. There is no episodes of being headache free like in chronic paroxysmal hemicrania. It is also associated with ipsilateral autonomic features.

Discuss (4) Improve

Next question >

Trigeminal autonomic cephalalgias

Trigeminal autonomic cephalalgias (TACs)

- cluster headache
- paroxysmal hemicrania
- hemicrania continua
- short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT syndrome)
- short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms (SUNA)

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

NEUROLOGY
TRIGEMINAL AUTONOMIC CEPHALALGIAS

Severe headaches - Understanding Trigeminal Autonomic Cephalgias - types, pathophysiology, treatment

Armando Hasudungan - YouTube 👍 10 👎 0

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Score: **20.8%**

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A 45-year-old man is brought to the hospital via ambulance with a tonic-clonic seizure. He has a past medical history of epilepsy, gastro-oesophageal reflux and latent TB. His medications include sodium valproate, omeprazole, isoniazid and he commenced carbamazepine as a consequence of poor seizure control 4 weeks previously. He is known to drink moderate-large quantities of grapefruit juice.

At the time of review, he has recovered and his Glasgow coma scale is 15/15.

A CT head is unremarkable.

The ingestion of which substance has caused his seizures?

- ○ Carbamazepine ×
- ○ Grapefruit juice ×
- ○ Isoniazid ×
- ○ Omeprazole ×
- ○ Sodium valproate ×

Submit answer

Reference ranges

Score: **24.8%**

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A 45-year-old man is brought to the hospital via ambulance with a tonic-clonic seizure. He has a past medical history of epilepsy, gastro-oesophageal reflux and latent TB. His medications include sodium valproate, omeprazole, isoniazid and he commenced carbamazepine as a consequence of poor seizure control 4 weeks previously. He is known to drink moderate-large quantities of grapefruit juice.

At the time of review, he has recovered and his Glasgow coma scale is 15/15.

A CT head is unremarkable.

The ingestion of which substance has caused his seizures?

Carbamazepine	52%
Grapefruit juice	33%
Isoniazid	10%
Omeprazole	2%
Sodium valproate	2%

Carbamazepine is known to exhibit autoinduction. hence when patients start carbamazepine they may see a return of seizures after 3-4 weeks of treatment

Important for me   Less important

Carbamazepine is correct. This medication exhibits autoinduction, which means that it may become less effective as a consequence. Due to this loss of response, patients may see a return of seizures 3-4 weeks after commencing treatment.

Grapefruit juice is incorrect. This is a cytochrome P450 enzyme inhibitor and therefore would increase rather than decrease the concentration of another drug.

Isoniazid is incorrect. This is a cytochrome P450 enzyme inhibitor and therefore would increase rather than decrease the concentration of another drug.

Omeprazole is incorrect. This is a cytochrome P450 enzyme inhibitor and therefore would increase rather than decrease the concentration of another drug.

Sodium valproate is incorrect. This is a cytochrome P450 enzyme inhibitor and therefore would increase rather than decrease the concentration of another drug.

Discuss (5) Improve

Next question

Carbamazepine

Carbamazepine is chemically similar to the tricyclic antidepressant drugs. It is most commonly used in the treatment of epilepsy, particularly partial seizures, where carbamazepine remains a first-line medication. Other uses include

- trigeminal neuralgia
- bipolar disorder

Mechanism of action

- binds to sodium channels increases their refractory period

Adverse effects

- P450 enzyme inducer
- dizziness and ataxia
- drowsiness
- headache
- visual disturbances (especially diplopia)
- Steven-Johnson syndrome
- leucopenia and agranulocytosis
- hyponatraemia secondary to syndrome of inappropriate ADH secretion

Carbamazepine is known to exhibit autoinduction, hence when patients start carbamazepine they may see a return of seizures after 3-4 weeks of treatment.

Next question >

B *I*

Textbooks

High-yield textbook

Extended textbook

Links

BNF 👍 9 👎 8

Carbamazepine

Suggest link Report broken link

Score: **20.8%**

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124 ✗

125 ✗

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Question 120 of 189

You are asked for advice from a GP regarding a 52-year-old woman with a diagnosis of Generalised Anxiety Disorder. She is having symptoms of free-floating anxiety for the past 5 years and this is causing work and relationship difficulties. The GP tells you that 5 years ago she was admitted to hospital with epigastric pain and anaemia and required a therapeutic endoscopy. A repeat endoscopy showed a small ulcer at D2 with a small amount of ooze which was treated with adrenaline. She still suffers from occasional heartburn.

She also has a history of alcohol dependency. She drank 20 units of alcohol per day for 20 years but has now cut down to 1-2 units per day. An ultrasound scan of the abdomen showed mild portal hypertension, hepatomegaly and no gastric varices. There is no history of deliberate self-harm or overdoses. She takes Vitamin B Co strong and thiamine 100mg PO OD and omeprazole 20mg PO OD and is compliant with medication currently but has a history of non-compliance with medication.

Which would be the safest therapy for treatment of her generalised anxiety disorder?

○ Clonazepam

○ Diazepam

○ Sertraline

○ Citalopram

○ Imipramine

Submit answer

Reference ranges

Score: **24.8%**

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You are asked for advice from a GP regarding a 52-year-old woman with a diagnosis of Generalised Anxiety Disorder. She is having symptoms of free-floating anxiety for the past 5 years and this is causing work and relationship difficulties. The GP tells you that 5 years ago she was admitted to hospital with epigastric pain and anaemia and required a therapeutic endoscopy. A repeat endoscopy showed a small ulcer at D2 with a small amount of ooze which was treated with adrenaline. She still suffers from occasional heartburn.

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Which would be the safest therapy for treatment of her generalised anxiety disorder?

Clonazepam	9%
Diazepam	6%
Sertraline	32%
Citalopram	25%
Imipramine	27%

Clonazepam and Diazepam are benzodiazepines. They are not recommended in the treatment of generalised anxiety disorder due to the chronic course of the illness and the dangers of developing tolerance and dependence upon the drug. The risk is even higher in this lady, who has a history of alcohol dependence. In addition, benzodiazepines potentiate the effect of alcohol as they both act on the GABA receptor making it an unsafe medication in someone with a history of alcohol dependency who is still consuming alcohol.

Sertraline and Citalopram are SSRIs. SSRIs inhibit platelet aggregation and are known to increase the risk of upper GI bleeds. This individual has a history of UGI haemorrhage, with recent bleeding duodenal ulcer and continuing symptoms. She also has portal hypertension and continues to drink alcohol putting him at risk of varices in the future. Whilst being on omeprazole will reduce this risk, there is a history of non-compliance with medication.

Imipramine is a TCA. TCAs are recommended when SSRIs are contraindicated in the treatment of generalised anxiety disorder. There is no increased risk of GI bleeds when on TCAs. It is potentially dangerous in overdose. However, there is no documented history of overdose with this individual

Discuss (13) | Improve

Next question >

Tricyclic antidepressants

Tricyclic antidepressants (TCAs) are used less commonly now for depression due to their side-effects and toxicity in overdose. They are however used widely in the treatment of neuropathic pain, where smaller doses are typically required.

The primary mechanism by which TCAs exert their antidepressant effects is through the inhibition of the reuptake of neurotransmitters

- Serotonin (5-HT): This neurotransmitter has a pivotal role in mood regulation. Inhibition of its reuptake leads to increased concentrations in the synaptic cleft, enhancing serotonergic neurotransmission.
- Noradrenaline (NA): Similar to 5-HT, blocking the reuptake of NA increases its synaptic cleft concentration, intensifying noradrenergic neurotransmission.

As well as 5-HT and NA, tricyclics interact with number of other receptors that contribute to their side-effect profile:

- antagonism of histamine receptors
  - drowsiness
- antagonism of muscarinic receptors
  - dry mouth
  - blurred vision
  - constipation
  - urinary retention
- antagonism of adrenergic receptors
  - postural hypotension
- lengthening of QT interval

Choice of tricyclic

- low-dose amitriptyline is commonly used in the management of neuropathic pain and the prophylaxis of headache (both tension and migraine)
- lofepramine has a lower incidence of toxicity in overdose
- amitriptyline and dosulepin (dothiepin) are considered the most dangerous in overdose

More sedative	Less sedative
Amitriptyline Clomipramine Dosulepin Trazodone*	Imipramine Lofepramine Nortriptyline

*trazodone is technically a 'tricyclic-related antidepressant'

Next question ›

Textbooks

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Media

ANTIDEPRESSANTS
PHARMACOLOGY

Antidepressants Pharmacology

Soton Brain Hub - YouTube

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Antidepressants

Speed Pharmacology - YouTube 👍 1 👎 1

Report broken media

Score: **20.8%**

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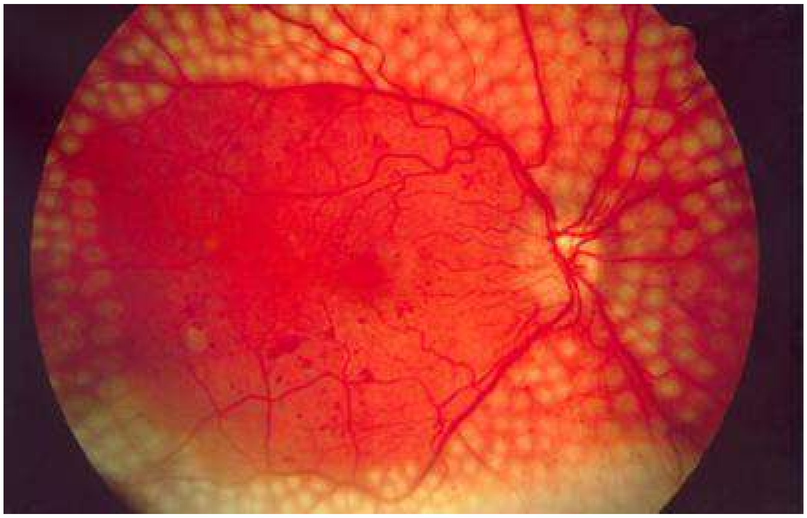
Question 121 of 189

A 67-year-old man with diabetes is attending the outpatient clinic complaining of a headache that has been bothering him for the last week. He reports he experienced some problems with the vision of his left eye two years ago, for which he was treated. Fundoscopic examination of his left eye appears as follows:



What treatment has this man taken for his previous visual problems?

- ○ Intravitreal ranibizumab
- ○ Intravitreal voriconazole
- ○ Laser photocoagulation
- ○ Oral voriconazole
- ○ Vitrectomy

Submit answer

Reference ranges

Score: **24.8%**

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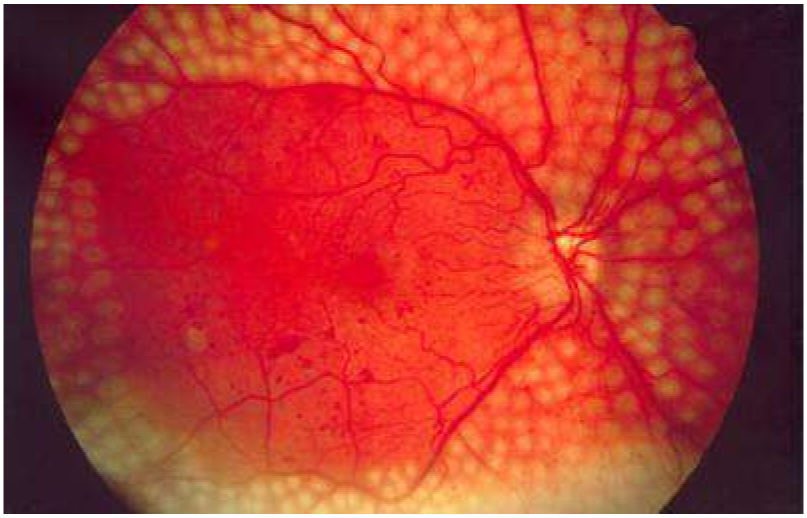


What treatment has this man taken for his previous visual problems?

Intravitreal ranibizumab	8%
Intravitreal voriconazole	2%
Laser photocoagulation	86%
Oral voriconazole	0%
Vitrectomy	4%

Laser photocoagulation is the correct answer. In this fundus picture are dot and blot haemorrhages and neovascularisation of the disc and retina, suggesting background proliferative diabetic retinopathy. There are laser scars in the peripheral retina, which are seen after laser photocoagulation. The pathophysiology, classification and management of diabetic retinopathy are explained in the notes linked below.

Intravitreal ranibizumab is incorrect. Ranibizumab is a VEGF inhibitor, and anti-VEGF agents are used widely to treat some forms of diabetic retinopathy. Indications of anti-VEGF agents in diabetic retinopathy include diabetic macular oedema with visual impairment and proliferative diabetic retinopathy. The patient in this scenario could have had intravitreal ranibizumab administered during his treatment. However, findings in the fundus picture should be considered critical to the solution, making laser photocoagulation the correct answer.

Intravitreal voriconazole is incorrect. This is administered in cases of fungal endophthalmitis involving the vitreous. Fundus examination in cases of fungal endophthalmitis reveals fluffy chorioretinal infiltrates with irregular margins. Diabetic patients have a higher predisposition to develop fungal infections. However, the fundus picture in this case is more suggestive of laser photocoagulation.

Oral voriconazole is incorrect. This is prescribed for conditions such as fungal keratitis. The fundus would not show any specific findings in a case of keratitis, making this an unlikely answer.

Vitrectomy is wrong. Vitrectomy is a procedure employed for treating complications of proliferative diabetic retinopathy like vitreous haemorrhage. Other indications for performing vitrectomy include tractional retinal detachment and progressive neovascularization that does not respond to photocoagulation. The fundus picture shows laser scars, which makes vitrectomy unlikely.

👍 👎 Discuss Improve

Next question >

Diabetic retinopathy ★

Diabetic retinopathy is the most common cause of blindness in adults aged 35-65 years-old.

Pathophysiology

Hyperglycaemia is thought to cause increased retinal blood flow and abnormal metabolism in the retinal vessel walls. This precipitates damage to endothelial cells and pericytes

Endothelial dysfunction leads to increased vascular permeability which causes the characteristic exudates seen on fundoscopy. Pericyte dysfunction predisposes to the formation of microaneurysms. Neovasculization is thought to be caused by the production of growth factors in response to retinal ischaemia

Classification

Patients are typically classified into those with non-proliferative diabetic retinopathy (NPDR), proliferative retinopathy (PDR) and maculopathy.

Non-proliferative diabetic retinopathy

Mild NPDR

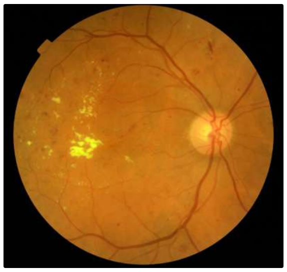
- 1 or more microaneurysm

Moderate NPDR

- microaneurysms
- blot haemorrhages
- hard exudates
- cotton wool spots ('soft exudates' - represent areas of retinal infarction), venous beading/looping and intraretinal microvascular abnormalities (IRMA) less severe than in severe NPDR

Severe NPDR

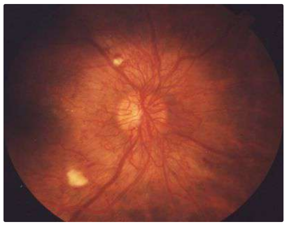
- blot haemorrhages and microaneurysms in 4 quadrants
- venous beading in at least 2 quadrants
- IRMA in at least 1 quadrant



Proliferative diabetic retinopathy

Key features

- retinal neovascularisation - may lead to vitrous haemorrhage
- fibrous tissue forming anterior to retinal disc
- more common in Type I DM, 50% blind in 5 years



Maculopathy

Key features

- based on location rather than severity, anything is potentially serious
- hard exudates and other 'background' changes on macula
- check visual acuity
- more common in Type II DM

Management

All patients

- optimise glycaemic control, blood pressure and hyperlipidemia
- regular review by ophthalmology

Maculopathy

- if there is a change in visual acuity then intravitreal vascular endothelial growth factor (VEGF) inhibitors

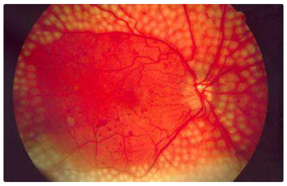
Non-proliferative retinopathy

- regular observation
- if severe/very severe consider panretinal laser photocoagulation

Proliferative retinopathy

- panretinal laser photocoagulation
  - following treatment around 50% of patients develop a noticeable reduction in their visual fields due to the scarring of peripheral retinal tissue
  - other complications include a decrease in night vision (rods are predominantly responsible for vision in low light conditions, the majority of rod cells are located in the peripheral retina), a generalised decrease in visual acuity and macular oedema
- intravitreal VEGF inhibitors
  - often now used in combination with panretinal laser photocoagulation
  - examples include ranibizumab
  - strong evidence base suggests they both slow progression of proliferative diabetic retinopathy and improve visual acuity

- if severe or vitreous haemorrhage: vitreoretinal surgery



123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20.8%**

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Question 122 of 189

A 26-year-old female presents with a 4-month history of continuous left sided facial pain and frontotemporal headache. She reports it to be constantly present and throbbing in nature, with exacerbations of worsened severity every 3 days. She reports exacerbations to typically be associated with injected left eye and left nasal congestion, with occasional teariness in her left eye.

She has no past medical history or drug history except the oral contraceptive pill. Her routine blood tests are unremarkable. A MRI head organised by her GP also demonstrated no intracranial pathology. A trial indomethacin you have organised is positive.

What is the most likely diagnosis?

- ○ Short-lasting unilateral neuralgiform attacks with conjunctival injection and tearing (SUNCT) ×
- ○ Hemicrania continua ×
- ○ Cluster headaches ×
- ○ Paroxysmal hemicrania ×
- ○ Migraines ×

Submit answer

Reference ranges

Score: **24.8%**

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A 26-year-old female presents with a 4-month history of continuous left sided facial pain and frontotemporal headache. She reports it to be constantly present and throbbing in nature, with exacerbations of worsened severity every 3 days. She reports exacerbations to typically be associated with injected left eye and left nasal congestion, with occasional teariness in her left eye.

She has no past medical history or drug history except the oral contraceptive pill. Her routine blood tests are unremarkable. A MRI head organised by her GP also demonstrated no intracranial pathology. A trial indomethacin you have organised is positive.

What is the most likely diagnosis?

Short-lasting unilateral neuralgiform attacks with conjunctival injection and tearing (SUNCT)	22%
Hemicrania continua	41%
Cluster headaches	17%
Paroxysmal hemicrania	18%
Migraines	2%

The patient has a constant headache with exacerbations in the distribution of the left ophthalmic distribution of the fifth cranial nerve associated with autonomic symptoms: this represents one of the trigeminal autonomic cephalgias (TACs). TACs is a class of disorders differentiated by the frequency and duration of attacks, and their response to indomethacin.

SUNCT, paroxysmal hemicrania and cluster headaches present with attacks of approximate duration seconds, minutes to 30 minutes, up to 180 minutes respectively. Symptoms are not present in between attacks. Cluster headaches appear more commonly in men while young women are more predisposed to paroxysmal hemicrania. Hemicrania continua produces pain that is always present with exacerbations in severity. Crucially for diagnosis, only hemicrania continua and paroxysmal hemicrania are responsive to indomethacin, making it a useful diagnostic and therapeutic prophylactic drug.

Discuss (7) | Improve

Next question ›

Trigeminal autonomic cephalalgias ★

Trigeminal autonomic cephalalgias (TACs)

- cluster headache
- paroxysmal hemicrania
- hemicrania continua
- short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT syndrome)
- short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms (SUNA)

+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Media

NEUROLOGY
TRIGEMINAL AUTONOMIC CEPHALALGIAS

Severe headaches - Understanding Trigeminal Autonomic Cephalgias - types, pathophysiology, treatment

Armando Hasudungan - YouTube

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Report broken media

Score: **20.8%**

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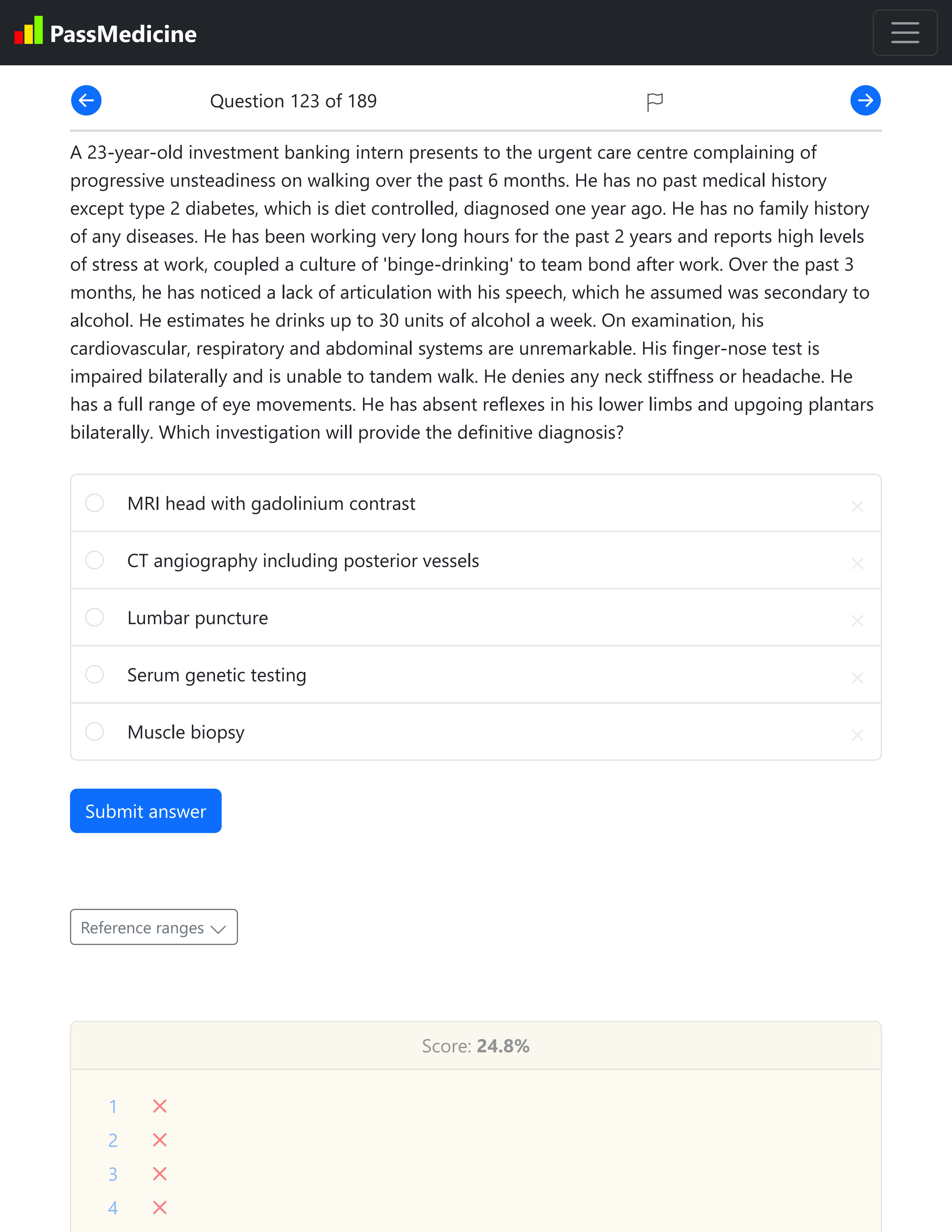

Question 123 of 189

A 23-year-old investment banking intern presents to the urgent care centre complaining of progressive unsteadiness on walking over the past 6 months. He has no past medical history except type 2 diabetes, which is diet controlled, diagnosed one year ago. He has no family history of any diseases. He has been working very long hours for the past 2 years and reports high levels of stress at work, coupled a culture of 'binge-drinking' to team bond after work. Over the past 3 months, he has noticed a lack of articulation with his speech, which he assumed was secondary to alcohol. He estimates he drinks up to 30 units of alcohol a week. On examination, his cardiovascular, respiratory and abdominal systems are unremarkable. His finger-nose test is impaired bilaterally and is unable to tandem walk. He denies any neck stiffness or headache. He has a full range of eye movements. He has absent reflexes in his lower limbs and upgoing plantars bilaterally. Which investigation will provide the definitive diagnosis?

- ○ MRI head with gadolinium contrast
- ○ CT angiography including posterior vessels
- ○ Lumbar puncture
- ○ Serum genetic testing
- ○ Muscle biopsy

Submit answer

Reference ranges

Score: **24.8%**

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MRI head with gadolinium contrast	42%
CT angiography including posterior vessels	7%
Lumbar puncture	3%
Serum genetic testing	37%
Muscle biopsy	10%

The patient describes cerebellar signs of dysarthria, poor coordination and ataxia, in addition to absent ankle jerks and upgoing plantars, presenting at an early age with progressive symptoms. Friedrich's ataxia and other spinocerebellar ataxias must be considered. A diagnosis of type 2 diabetes is odd in such a young patient. However, up to 1/3 of patients with Friedrich's ataxia experience impaired glucose tolerance or diabetes mellitus. These patients often present with cardiomyopathy as well, causing arrhythmias and heart failures.

This patient presents slightly late, most patients have symptoms by childhood. Up to 90% present by 25 years old[1]. It is often assumed that spinocerebellar ataxias, in this case an autosomal recessive one, must be accompanied by a family history. In 40% of Friedrich's ataxia, there is no positive family history at all[1].

Diagnosis is by serum genetic testing of the GAA trinucleotide repeat. Treatment is limited but involves a multidisciplinary team of neurologists, cardiologists and endocrinologists. There are theories that iron chelation is important in Friedrich's ataxia but these are yet to be proven in large-scale studies[2].

1. Durr A, Cossee M, Agid Y et al. Clinical and genetic abnormalities in patients with Friedreich's ataxia. N Engl J Med. 1996;335(16):1169
2. Schulz JB, Dehmer T, Schols L et al. Oxidative stress in patients with Friedreich ataxia. Neurology. 2000;55(11):1719

👍 👎 💬 Discuss (9) Improve

Next question ›

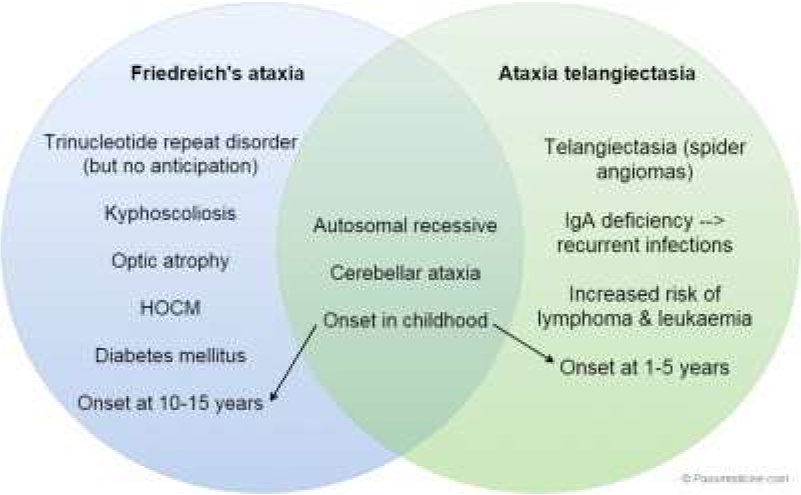
Friedreich's ataxia ★

Friedreich's ataxia is the most common of the early-onset hereditary ataxias. It is an autosomal recessive, trinucleotide repeat disorder characterised by a GAA repeat in the X25 gene on chromosome 9 (frataxin). Friedreich's ataxia is unusual amongst trinucleotide repeat disorders in not demonstrating the phenomenon of anticipation.

The typical age of onset is 10-15 years old. Gait ataxia and kyphoscoliosis are the most common presenting features.

Neurological features
- absent ankle jerks/extensor plantars
- cerebellar ataxia
- optic atrophy
- spinocerebellar tract degeneration

Other features
- hypertrophic obstructive cardiomyopathy (90%, most common cause of death)
- diabetes mellitus (10-20%)
- high-arched palate


Friedreich's ataxia
Ataxia telangiectasia
Trinucleotide repeat disorder (but no anticipation)
Kyphoscoliosis
Optic atrophy
HOCM
Diabetes mellitus
Onset at 10-15 years
Autosomal recessive
Cerebellar ataxia
Onset in childhood
Telangiectasia (spider angiomas)
IgA deficiency --> recurrent infections
Increased risk of lymphoma & leukaemia
Onset at 1-5 years
© Passmedicine.com

Comparison of Friedreich's ataxia and ataxic telangiectasia. Note in particular how ataxic telangiectasia tends to present much earlier, often at the age of 1-2 years

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Rare Disease Video Library - NORD 3 5

Friedreichâ€™s Ataxia

Suggest link Report broken link

Media

FRIEDREICH'S ATAXIA
Osmosis.org

Friedreichâ€™s ataxia

Osmosis - YouTube 👍 7 👎 0

Report broken media

Score: **20.8%**

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Back to top

A 58-year-old man presents with severe dyspnoea at rest. He has a past medical history of motor neurone disease which was diagnosed 9 months ago. On examination his chest sounds clear. His respiratory rate is 20 breaths per minute with a shallow depth.

You perform an arterial blood gas which is as follows:

PaO2	7.1 kPa
PCO2	6.3 kPa
pH	7.32
HCO3	38 mmol/l

How will you manage this patient?

- ○ BiPAP
- ○ CPAP
- ○ LTOT
- ○ Palliation of breathlessness with morphine
- ○ Intubation and ventilation

Submit answer

Reference ranges

Score: **24.8%**

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Back to top

A 58-year-old man presents with severe dyspnoea at rest. He has a past medical history of motor neurone disease which was diagnosed 9 months ago. On examination his chest sounds clear. His respiratory rate is 20 breaths per minute with a shallow depth.

You perform an arterial blood gas which is as follows:

PaO2	7.1 kPa
PCO2	6.3 kPa
pH	7.32
HCO3	38 mmol/l

How will you manage this patient?

BiPAP	63%
CPAP	15%
LTOT	4%
Palliation of breathlessness with morphine	4%
Intubation and ventilation	14%

Non-invasive ventilation (NIV) should be the treatment of choice for patients with chest wall or neuromuscular disease causing type 2 respiratory failure. Additional LTOT may be required in case of hypoxaemia not corrected with NIV

Important for me Less important

The patient has developed type 2 respiratory failure as demonstrated by a PaO2 < 8 kPa and a respiratory acidosis. This is likely due to diaphragmatic weakness secondary to motor neurone disease. Non-invasive ventilation (NIV), in particular BiPAP, should be the treatment of choice for patients with chest wall or neuromuscular disease causing type 2 respiratory failure. Additional LTOT may be required in case of hypoxaemia not corrected with NIV.

Discuss (2) Improve

Next question

Motor neuron disease: management

Motor neuron disease is a neurological condition of unknown cause which can present with both upper and lower motor neuron signs. It rarely presents before 40 years and various patterns of disease are recognised including amyotrophic lateral sclerosis, progressive muscular atrophy and bulbar palsy

Riluzole

- prevents stimulation of glutamate receptors
- used mainly in amyotrophic lateral sclerosis
- prolongs life by about 3 months

Respiratory care

- non-invasive ventilation (usually BIPAP) is used at night
- studies have shown a survival benefit of around 7 months

Nutrition

- percutaneous gastrostomy tube (PEG) is the preferred way to support nutrition and has been associated with prolonged survival

Prognosis

- poor: 50% of patients die within 3 years

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 6 👎 5

2016 Motor neurone disease: assessment and management

Suggest link

Report broken link

Media

MOTOR NEURONE DISEASE
SPEECH
TRUNK
MOTOR NEURON DEGENERATION
VENTILATION
LIMBS
TONGUE WASTING
BRISK REFLEXES

Motor neuron disease

Zero To Finals - YouTube 👍 0 👎 0

Report broken media

Score: **20.8%**

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A 22-year-old woman presents with a worsening headache. She states that it is worse in the morning and exacerbated by straining. On examination, you note bilateral papilloedema with enlarged blind spots. Visual acuity is 6/6 in both eyes.

Blood results are as follows:

Hb	122 g/L	Male: (135-180) Female: (115 - 160)
Platelets	162 * 10^9/L	(150 - 400)
WBC	9.6 * 10^9/L	(4.0 - 11.0)
Na^+	140 mmol/L	(135 - 145)
K^+	3.9 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	64 µmol/L	(55 - 120)
CRP	2 mg/L	(< 5)
Glucose	5.8 mmol/L	(4-7)

A CT head is organised:

CT head	Papilloedema with flattening of the posterior sclera; bilateral venous sinus stenosis predominantly of the lateral segments of the transverse sinuses

A diagnostic lumbar puncture is organised:

Opening pressure	52 cm H2O	(7-18)
WBC count	3 cells/µL	(0-5)
Glucose	5.4 mmol/L	(> 0.7 x serum glucose)
Protein	22 mg/dL	(<45)

What treatment is indicated?

Acetazolamide 53%

Optic nerve sheath decompression and fenestration	13%
Serial lumbar punctures	11%
Topiramate	1%
Ventriculoperitoneal shunt	22%

Acetazolamide is a carbonic anhydrase inhibitor that is used to treat idiopathic intracranial hypertension

Important for me Less important

The radiological and lumbar puncture results support a diagnosis of idiopathic intracranial hypertension (IIH).

The main principles of management of IIH are:
- to treat the underlying disease
- to protect the vision
- to minimise the headache morbidity

Acetazolamide is correct. The patient does not have an imminent threat to vision and should therefore be commenced on acetazolamide, and advised to lose weight if appropriate.

Optic nerve sheath decompression and fenestration is incorrect. There is evidence that many of the surgical procedures, such as CSF diversion and optic nerve sheath fenestration (ONSF), work well in the short term. However since the patient in this case does not have an imminent threat to vision, then medical management should be implemented in the first instance.

Serial lumbar punctures is incorrect. The relief from a lumbar puncture is typically short-lived as CSF is secreted from the choroid plexus at a rate of 25€‰mL/hour and consequently, the volume removed in a so-called therapeutic tap is rapidly replaced. Therefore, Serial lumbar punctures are not recommended for the management of IIH.

Topiramate is incorrect. Topiramate has carbonic anhydrase activity and can suppress appetite. There is growing evidence suggestive of the efficacy of this drug in treating IIH, however, it currently remains a second-line agent.

Ventriculoperitoneal shun is incorrect. Ventriculoperitoneal (VP) is the preferred CSF diversion procedure for visual deterioration in IIH, due to lower reported revisions per patient.

👍 👎 Discuss (7) Improve

Next question

Idiopathic intracranial hypertension ★

Idiopathic intracranial hypertension (also known as pseudotumour cerebri and formerly benign intracranial hypertension) is a condition classically seen in young, overweight females.

Risk factors

- obesity
- female sex
- pregnancy
- drugs
  - combined oral contraceptive pill
  - steroids
  - tetracyclines
  - retinoids (isotretinoin, tretinoin) / vitamin A
  - lithium

Features

- headache
- blurred vision
- papilloedema (usually present)
- enlarged blind spot
- sixth nerve palsy may be present

Management

- weight loss
  - whilst diet and exercise are important, medications such as semaglitide and topiramate may be considered by specialists. Topiramate is particularly beneficial as it also inhibits carbonic anhydrase
- carbonic anhydrase inhibitors e.g. acetazolamide
- topiramate is also used, and has the added benefit of causing weight loss in most patients
- repeated lumbar puncture may be used as a temporary measure but is not suitable for longer-term management
- surgery: optic nerve sheath decompression and fenestration may be needed to prevent damage to the optic nerve. A lumboperitoneal or ventriculoperitoneal shunt may also be performed to reduce intracranial pressure

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 12 👎 13

Idiopathic intracranial hypertension review

Suggest link

Report broken link

Score: **20.8%**

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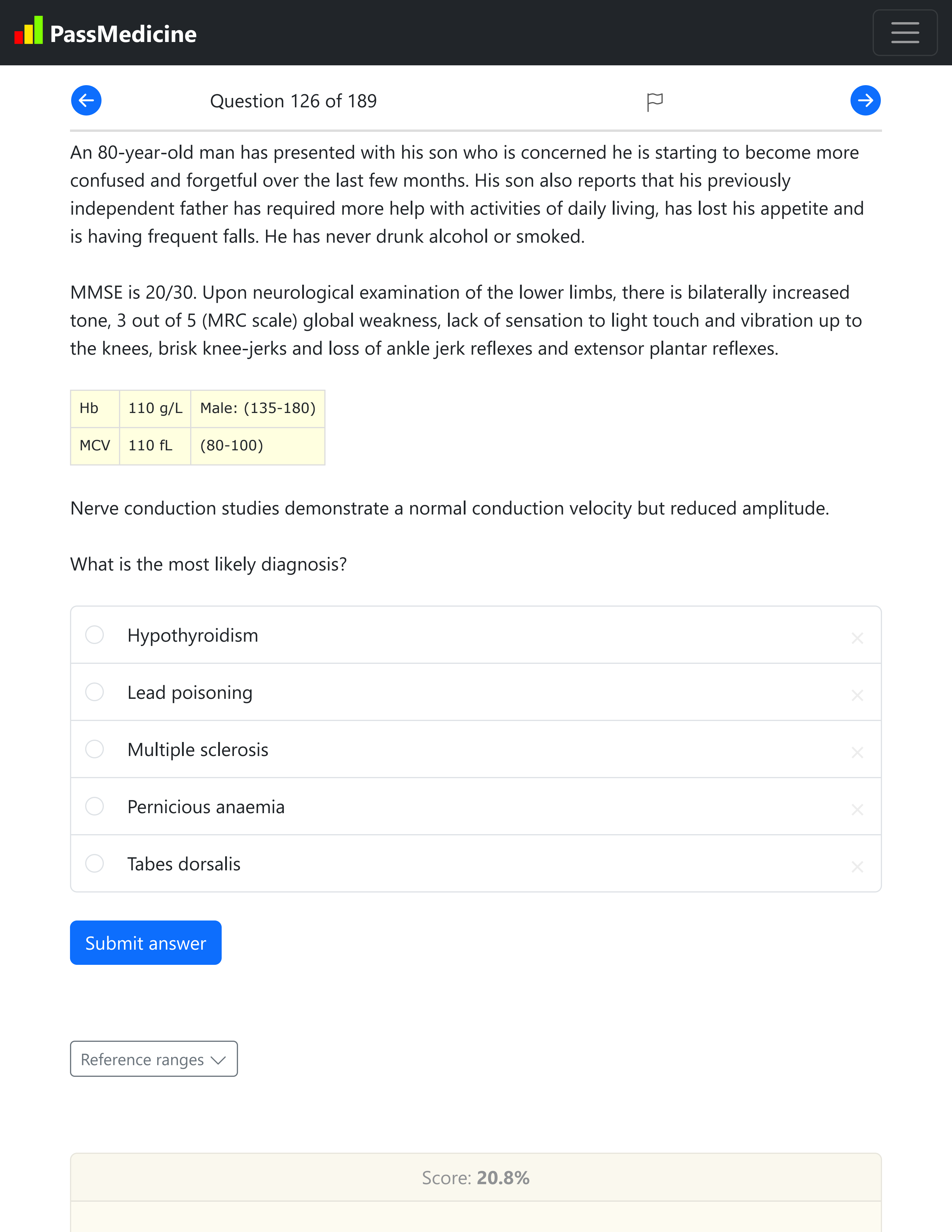

Question 126 of 189

An 80-year-old man has presented with his son who is concerned he is starting to become more confused and forgetful over the last few months. His son also reports that his previously independent father has required more help with activities of daily living, has lost his appetite and is having frequent falls. He has never drunk alcohol or smoked.

MMSE is 20/30. Upon neurological examination of the lower limbs, there is bilaterally increased tone, 3 out of 5 (MRC scale) global weakness, lack of sensation to light touch and vibration up to the knees, brisk knee-jerks and loss of ankle jerk reflexes and extensor plantar reflexes.

Hb	110 g/L	Male: (135-180)
MCV	110 fL	(80-100)

Nerve conduction studies demonstrate a normal conduction velocity but reduced amplitude.

What is the most likely diagnosis?

○ Hypothyroidism

○ Lead poisoning

○ Multiple sclerosis

○ Pernicious anaemia

○ Tabes dorsalis

Score: **20.8%**

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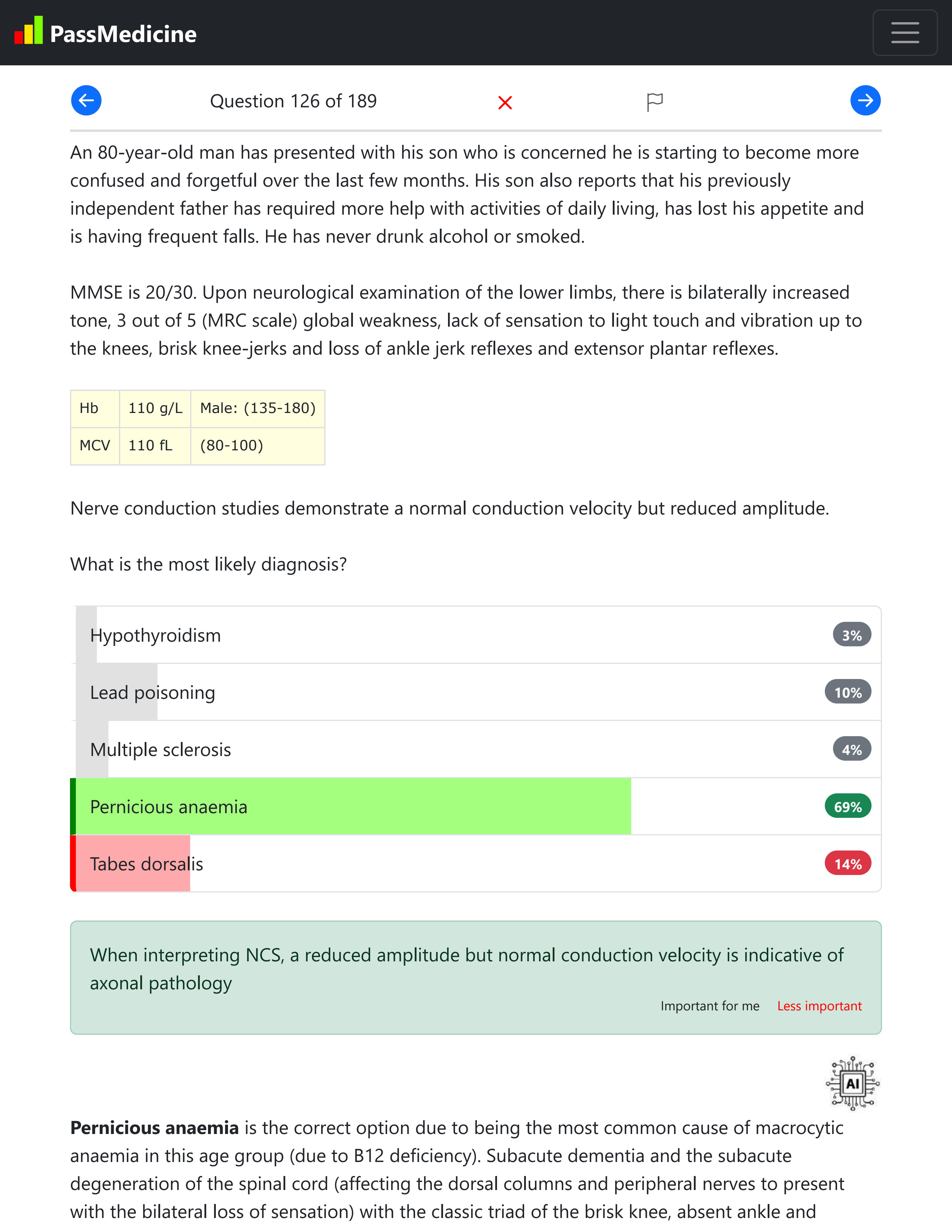

An 80-year-old man has presented with his son who is concerned he is starting to become more confused and forgetful over the last few months. His son also reports that his previously independent father has required more help with activities of daily living, has lost his appetite and is having frequent falls. He has never drunk alcohol or smoked.

MMSE is 20/30. Upon neurological examination of the lower limbs, there is bilaterally increased tone, 3 out of 5 (MRC scale) global weakness, lack of sensation to light touch and vibration up to the knees, brisk knee-jerks and loss of ankle jerk reflexes and extensor plantar reflexes.

Hb	110 g/L	Male: (135-180)
MCV	110 fL	(80-100)

Nerve conduction studies demonstrate a normal conduction velocity but reduced amplitude.

What is the most likely diagnosis?

Hypothyroidism	3%
Lead poisoning	10%
Multiple sclerosis	4%
Pernicious anaemia	69%
Tabes dorsalis	14%

When interpreting NCS, a reduced amplitude but normal conduction velocity is indicative of axonal pathology

Important for me Less important

Pernicious anaemia is the correct option due to being the most common cause of macrocytic anaemia in this age group (due to B12 deficiency). Subacute dementia and the subacute degeneration of the spinal cord (affecting the dorsal columns and peripheral nerves to present with the bilateral loss of sensation) with the classic triad of the brisk knee, absent ankle and extensor plantar reflexes can be explained by the B12 deficiency. This deficiency is a result of a chronic lack of intrinsic factors to metabolise dietary B12 to its active form. The lack of B12 has led to the axonal degeneration pattern seen with the reduced amplitude in the nerve conduction studies.

Hypothyroidism is incorrect. Untreated hypothyroidism would cause macrocytic anaemia and axonal neuropathy, however, would not explain the associated cord signs and symptoms described in this case.

Lead poisoning is incorrect, although it is another possible cause for an axonal pattern of nerve degeneration. However, in this case, the lead poisoning would not account for subacute dementia or cord signs.

Multiple sclerosis is incorrect as this does not usually result in cognitive impairment and would usually affect the corticospinal tracts also. Furthermore, it would not cause the axonal pattern in nerve conduction studies. The B12 deficiency is more likely in this case given the signs and symptoms.

Tabes dorsalis is incorrect. This is a result of tertiary syphilis and would explain the neurological signs seen here as it also affects the dorsal columns predominantly, however it would not explain the cause of the macrocytic anaemia.

👍 👎 💬 Discuss (1) Improve

Next question ›

Nerve conduction studies ★

Nerve conduction studies (NCS) are useful in determining between axonal and demyelinating pathology

Axonal
- normal conduction velocity
- reduced amplitude

Demyelinating
- reduced conduction velocity
- normal amplitude

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Score: **20%**

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Question 127 of 189

A 26-year-old woman presents with recurrent slurring of her speech that worsens as she continues to talk. She has double vision that worsens while watching TV and subsequently improves with rest, as well as intermittent weakness of the limbs.

On examination, she has visible ptosis - she explains this also improves with rest. Proximal muscle weakness is evident, with no wasting.

As well as the suspected neurological diagnosis, she demonstrates pyrexia and concurrent infection is suspected. The source is investigated.

Which antibiotic should be avoided, given the likely underlying neurological diagnosis?

- ○ Co-amoxiclav
- ○ Gentamicin
- ○ Meropenem
- ○ Nitrofurantoin
- ○ Vancomycin

Submit answer

Reference ranges

Score: **20.8%**

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A 26-year-old woman presents with recurrent slurring of her speech that worsens as she continues to talk. She has double vision that worsens while watching TV and subsequently improves with rest, as well as intermittent weakness of the limbs.

On examination, she has visible ptosis - she explains this also improves with rest. Proximal muscle weakness is evident, with no wasting.

As well as the suspected neurological diagnosis, she demonstrates pyrexia and concurrent infection is suspected. The source is investigated.

Which antibiotic should be avoided, given the likely underlying neurological diagnosis?

Co-amoxiclav	4%
Gentamicin	75%
Meropenem	2%
Nitrofurantoin	15%
Vancomycin	3%

> Aminoglycoside antibiotics are contraindicated in patients with myasthenia gravis due to the risk of fatal myasthenic crisis and respiratory failure
>
> Important for me Less important

The history described above is highly suggestive of myasthenia gravis, given the history and signs found on examination. In a patient with this condition, a number of drugs may cause significant exacerbations and increase the risk of myasthenic crises. A number of antibiotics are contraindicated, including aminoglycosides such as **gentamicin**. Other classes generally avoided include macrolides, quinolones and tetracyclines.

Co-amoxiclav is generally considered safe to use in myasthenia gravis, as are other penicillin-based antibiotics.

Meropenem is a beta-lactam antibiotic used in many situations where broad-spectrum treatment is needed. It is also considered safe to use in myasthenia gravis.

Nitrofurantoin is commonly used for the treatment of urinary tract infections. It is considered safe in patients with myasthenia gravis.

Vancomycin is a glycopeptide antibiotic, often used for complex or resistant infections. It is thought to be safe to use in myasthenia gravis.

Discuss (4) Improve

Next question

Myasthenia gravis: exacerbating factors

The most common exacerbating factor is exertion resulting in fatigability, which is the hallmark feature of myasthenia gravis.. Symptoms become more marked during the day

The following drugs may exacerbate myasthenia:
- penicillamine
- quinidine, procainamide
- beta-blockers
- lithium
- phenytoin
- antibiotics: gentamicin, macrolides, quinolones, tetracyclines

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians (Edin) 👍 4 👎 2

Myasthenia gravis review

Myasthenia Gravis Association 👍 9 👎 3

Drugs which may aggravate myasthenia gravis

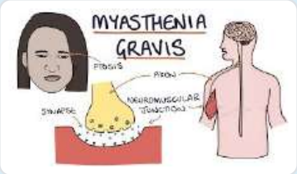
Suggest link Report broken link

Media

MYASTHENIA GRAVIS

Myasthenia gravis - causes, symptoms, treatment, pathology

Osmosis - YouTube 👍 1 👎 0


MYASTHENIA GRAVIS
PTOSIS
AXON
NEUROMUSCULAR JUNCTION
SYNAPSE

Myasthenia gravis

Zero to Finals - YouTube 👍 2 👎 1

Report broken media

Score: **20%**

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On examination he has a bilateral VII nerve weakness but no other cranial nerve lesions detected. The arms and legs show no focal neurology.

Skin is intact with no skin lesions seen. There is no evidence of any joint effusions and all joints appear to have a full range of motion

Investigations:

Haemoglobin	127 g/L (130-180)
White cell count	10.0 Ã— 109/L (4.0-11.0)

Chest X-ray	Normal

What is the most likely diagnosis?

- ○ Sarcoidosis
- ○ Lyme disease
- ○ Syphilis
- ○ Bells palsy
- ○ Subdural haemorrhage

Submit answer

Reference ranges

Score: **20.8%**

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Investigations:

Haemoglobin	127 g/L (130-180)
White cell count	10.0 Ã— 109/L (4.0-11.0)

Chest X-ray	Normal

What is the most likely diagnosis?

Sarcoidosis	5%
Lyme disease	72%
Syphilis	3%
Bells palsy	17%
Subdural haemorrhage	4%

This patient has bilateral seventh nerve palsy. The most likely diagnosis is Lyme disease. Lyme disease is caused by *Borrelia burgdorferi* bacteria that affects human from ticks that also feed on rodents.

Infected tick bites are common especially in the New Forest area of the UK. 70-80% of persons infected develop a rash (erythema migrans - target rash). Weeks after the initial bite the spirochetes spread via the bloodstream to joints, heart and nervous system.

Syphilis is on the rise but the more likely cause of the bilateral seventh nerve palsy is Lyme disease especially given the rash.

Subdural haemorrhage can occur after trauma - such as bike fall. Symptoms are likely to occur within days rather than having a first presentation one month after the accident.

Discuss (5) Improve

Next question >

Facial nerve palsy ★

The facial nerve is the main nerve supplying the structures of the second embryonic branchial arch. It is predominantly an efferent nerve to the muscles of facial expression, digastric muscle and also to many glandular structures. It contains a few afferent fibres which originate in the cells of its genicular ganglion and are concerned with taste.

Supply - 'face, ear, taste, tear'

- face: muscles of facial expression
- ear: nerve to stapedius
- taste: supplies anterior two-thirds of tongue
- tear: parasympathetic fibres to lacrimal glands, also salivary glands

Causes of bilateral facial nerve palsy

- sarcoidosis
- Guillain-Barre syndrome
- Lyme disease
- bilateral acoustic neuromas (as in neurofibromatosis type 2)
- as Bell's palsy is relatively common it accounts for up to 25% of cases f bilateral palsy, but this represents only 1% of total Bell's palsy cases

Causes of unilateral facial nerve palsy - as above plus

Lower motor neuron • **Bell's palsy** • **Ramsay-Hunt syndrome (due to herpes zoster)** • **acoustic neuroma** • **parotid tumours** • **HIV** • **multiple sclerosis*** • **diabetes mellitus**	**Upper motor neuron** • **stroke**

LMN vs. UMN
- upper motor neuron lesion 'spares' upper face i.e. forehead
- lower motor neuron lesion affects all facial muscles

*may also cause an UMN palsy

Path

Subarachnoid path
- Origin: motor- pons, sensory- nervus intermedius
- Pass through the petrous temporal bone into the internal auditory meatus with the vestibulocochlear nerve. Here they combine to become the facial nerve.

Facial canal path
- The canal passes superior to the vestibule of the inner ear
- At the medial aspect of the middle ear, it becomes wider and contains the geniculate ganglion.

- 3 branches:
- 1. greater petrosal nerve
- 2. nerve to stapedius
- 3. chorda tympani

Stylomastoid foramen
- Passes through the stylomastoid foramen (tympanic cavity anterior and mastoid antrum posteriorly)
- Posterior auricular nerve and branch to posterior belly of digastric and stylohyoid muscle

Next question >

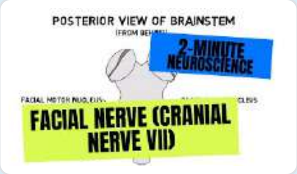
B *I* A

Textbooks

High-yield textbook

Extended textbook

Media


POSTERIOR VIEW OF BRAINSTEM
2-MINUTE NEUROSCIENCE
FACIAL NERVE (CRANIAL NERVE VII)

2-Minute Neuroscience: Facial Nerve (Cranial Nerve VII)

Neuroscientifically Challenged - YouTube 👍 1 👎 0

Report broken media

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A 25-year-old lady attends her first neurologist appointment after moving areas. She has been referred by her GP with possible migraines for which she was receiving acetazolamide.

She attends reporting that the headaches are severe. They are typically up to 10/10 and are worse at the start of the day and improves as her day progresses. She finds sitting in a chair helpful and can sometimes vomit with them. Coughing and chewing hard food makes the headaches worse.

On examination, she has normal eye movements. Her optic discs appear a little blurred and there are no visual field defects. She refuses to lie flat and is non tender over her temporal scalp. She has normal observations and is afebrile. Her most recent tests are transferred to you

Na^+	132 mmol/l
K^+	3.1 mmol/l
Urea	4.2 mmol/l
Creatinine	76 µmol/l
HCO3	18mmol/l
MRI head	No mass lesion seen, increased subarachnoid space around the optic nerves

What is the most appropriate management?

- ○ Endovascular clipping
- ○ Lumbar puncture
- ○ Nifedipine
- ○ Sumatriptan
- ○ Refer to neurosurgery

Submit answer

Reference ranges

Score: **20.8%**

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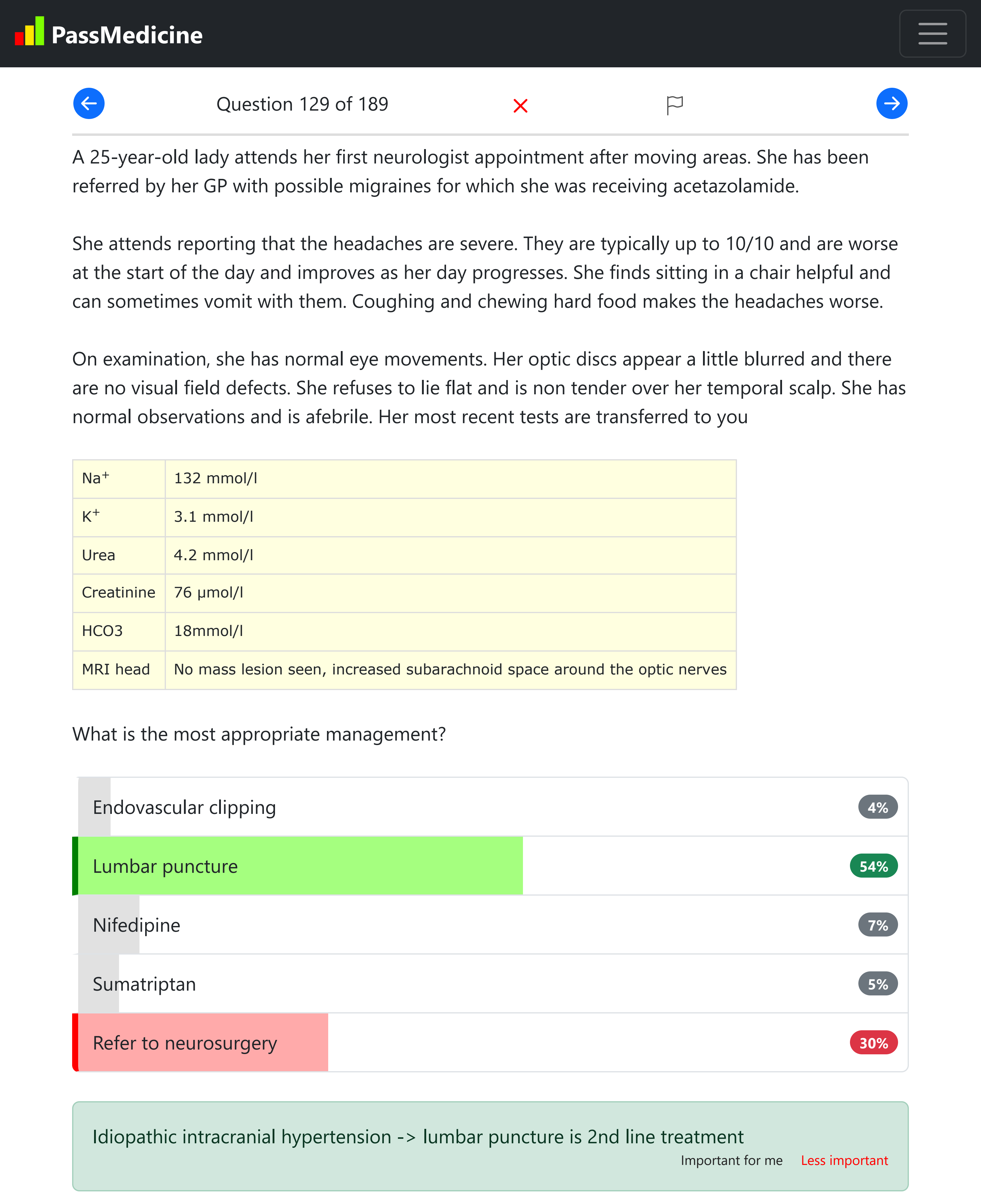

Question 129 of 189

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HCO3	18mmol/l
MRI head	No mass lesion seen, increased subarachnoid space around the optic nerves

What is the most appropriate management?

Endovascular clipping	4%
Lumbar puncture	54%
Nifedipine	7%
Sumatriptan	5%
Refer to neurosurgery	30%

Idiopathic intracranial hypertension -> lumbar puncture is 2nd line treatment

Important for me Less important

The key to headaches is the history. This is not sudden and so is not a subarachnoid haemorrhage and nifedipine and endovascular clipping would therefore be inappropriate. Likewise, it does not sound entirely migrainous and sumatriptan would probably not help. There are no features that warrant a referral to neurosurgery. The enlarged subarachnoid space and postural headache suggest this is idiopathic intracranial hypertension. The first line is medicines like carbonic anhydrase inhibitors and if these fail then therapeutic lumbar puncture should be provided.

👍 👎 Discuss (11) Improve

Next question >

Idiopathic intracranial hypertension

Idiopathic intracranial hypertension (also known as pseudotumour cerebri and formerly benign intracranial hypertension) is a condition classically seen in young, overweight females.

Risk factors
- obesity
- female sex
- pregnancy
- drugs
  - combined oral contraceptive pill
  - steroids
  - tetracyclines
  - retinoids (isotretinoin, tretinoin) / vitamin A
  - lithium

Features
- headache
- blurred vision
- papilloedema (usually present)
- enlarged blind spot
- sixth nerve palsy may be present

Management
- weight loss
  - whilst diet and exercise are important, medications such as semaglitide and topiramate may be considered by specialists. Topiramate is particularly beneficial as it also inhibits

carbonic anhydrase

- carbonic anhydrase inhibitors e.g. acetazolamide
- topiramate is also used, and has the added benefit of causing weight loss in most patients
- repeated lumbar puncture may be used as a temporary measure but is not suitable for longer-term management
- surgery: optic nerve sheath decompression and fenestration may be needed to prevent damage to the optic nerve. A lumboperitoneal or ventriculoperitoneal shunt may also be performed to reduce intracranial pressure

AI + Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 12 13

Idiopathic intracranial hypertension review

Suggest link Report broken link

Score: **20%**

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A 50-year-old man is seen in the Emergency Department with fevers. He has been feeling generally unwell for the last 3 days and has just returned from a 2 week stay with his sister who lives on the other side of the U.K. He has had a mild non-productive cough and shortness of breath. He has had no vomitting, diarrhoea, dysuria or abdominal pain.

He has a past medical history of epilepsy, schizophrenia, hypertension and diet-controlled type 2 diabetes.

On examination he has a temperature of 38.3 ºC and is saturating at 94% on room air. His heart rate is 111 beats per minute and blood pressure is 118/75 mmHg. He looks clammy and pale but systems examination is otherwise unremarkable.

His chest x-ray shows clear lung fields.

His blood tests are as follows:

Hb	140 g/l	Na^+	141 mmol/l
Platelets	443 * 10^9/l	K^+	3.7 mmol/l
WBC	1.5 * 10^9/l	Urea	7 mmol/l
Neuts	0.6 * 10^9/l	Creatinine	98 µmol/l
Lymphs	0.5 * 10^9/l	CRP	170 mg/l

He is started on broad spectrum antibiotics and fluids.

Which drug is most likely to have precipitated this condition?

- ○ Clobazam ×
- ○ Clozapine ×
- ○ Phenytoin ×
- ○ Sodium valproate ×
- ○ Venlafaxine ×

Submit answer

Reference ranges ⌵

Score: **20.8%**

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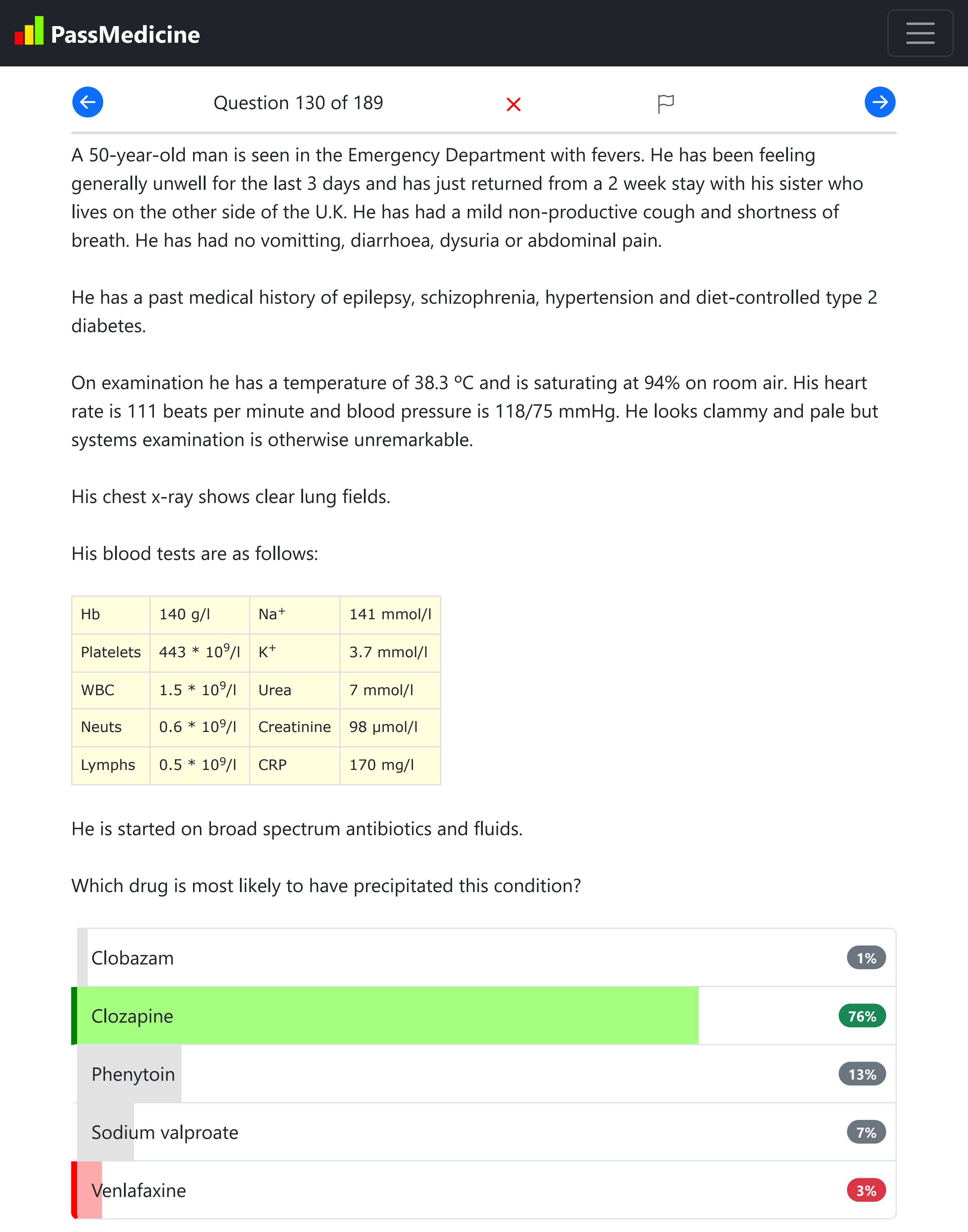


A 50-year-old man is seen in the Emergency Department with fevers. He has been feeling generally unwell for the last 3 days and has just returned from a 2 week stay with his sister who lives on the other side of the U.K. He has had a mild non-productive cough and shortness of breath. He has had no vomitting, diarrhoea, dysuria or abdominal pain.

He has a past medical history of epilepsy, schizophrenia, hypertension and diet-controlled type 2 diabetes.

On examination he has a temperature of 38.3 ºC and is saturating at 94% on room air. His heart rate is 111 beats per minute and blood pressure is 118/75 mmHg. He looks clammy and pale but systems examination is otherwise unremarkable.

His chest x-ray shows clear lung fields.

His blood tests are as follows:

Hb	140 g/l	Na^+	141 mmol/l
Platelets	$443 * 10^9$/l	K^+	3.7 mmol/l
WBC	$1.5 * 10^9$/l	Urea	7 mmol/l
Neuts	$0.6 * 10^9$/l	Creatinine	98 µmol/l
Lymphs	$0.5 * 10^9$/l	CRP	170 mg/l

He is started on broad spectrum antibiotics and fluids.

Which drug is most likely to have precipitated this condition?

Clobazam	1%
Clozapine	76%
Phenytoin	13%
Sodium valproate	7%
Venlafaxine	3%

Agranulocytosis/neutropenia is a life-threatening side effect of **clozapine** - monitor FBC

Important for me Less important

This gentleman has neutropenic sepsis. The antipsychotic clozapine is well documented to cause agranulocytosis, neutropenia and lymphopenia. Patients on clozapine must be registered with the monitoring service specific to their brand of the drug and have regular drug levels and full blood counts. This gentleman has been travelling and has likely missed his monitoring appointment.

Clobazam side effects include drowsiness and confusion.

Phenytoin can cause leucopenia but this is unlikely in the absence of its other extensive side effect profile which includes drowsiness, tremor, gum hypertrophy, acne and hirsutism.

Sodium valproate also has a broad side effect profile which includes nausea, diarrhoea, weight gain, extra-pyramidal symptoms and hair loss. Again leucopenia is a documented side effect but rare.

Venlafaxine is associated with changes to serum cholesterol, hypertension, micturition disturbance and vasodilatation.

Reference: British National Formulary.

👍 👎 Discuss (3) Improve

Next question >

Clozapine ★

Clozapine, one of the first atypical agents to be developed, carries a significant risk of agranulocytosis and full blood count monitoring is therefore essential during treatment. For this reason, clozapine should only be used in patients resistant to other antipsychotic medication. The BNF states:

> *Clozapine should be introduced if schizophrenia is not controlled despite the sequential use of*

> *two or more antipsychotic drugs (one of which should be a second-generation antipsychotic drug), each for at least 6-8 weeks.*

Adverse effects of clozapine

- agranulocytosis (1%), neutropaenia (3%)
- reduced seizure threshold - can induce seizures in up to 3% of patients
- constipation
- myocarditis: a baseline ECG should be taken before starting treatment
- hypersalivation

Dose adjustment of clozapine might be necessary if smoking is started or stopped during treatment.

AI + Q 123

Next question >

B *I* A T↕

Textbooks

High-yield textbook

Extended textbook

Links

BNF 👍 4 👎 2

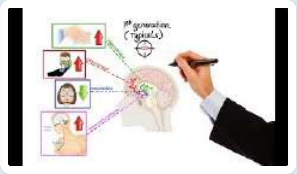
Clozapine

Clinical Knowledge Summaries 👍 3 👎 0

Schizophrenia > Prescribing Information > Adverse effects

Suggest link
Report broken link

Media



Antipsychotics

Speed Pharmacology - YouTube 👍 2 👎 0

Report broken media

Score: **20%**

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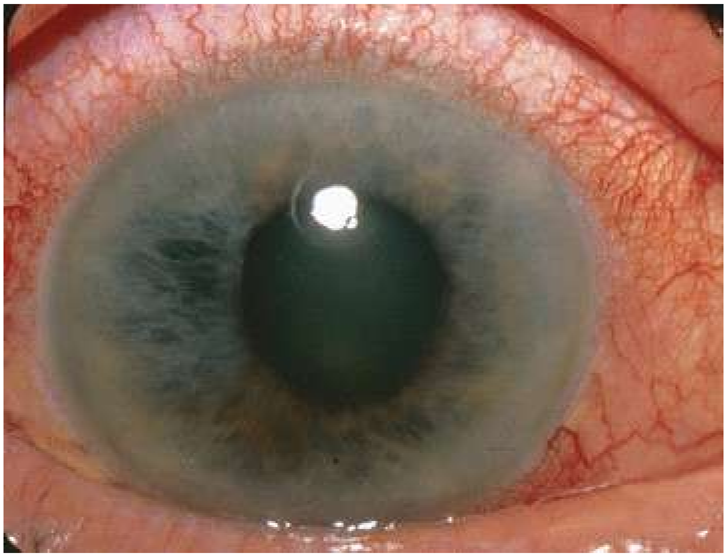
A 50-year-old man presents to the emergency department with a painful right eye. The pain started approximately 4 hours ago whilst watching a movie. He also reports that his vision is blurry in the same eye. His past medical history includes type 2 diabetes and rheumatoid arthritis.

His eye is examined, as shown below:



What is the most appropriate management for this patient?

- ○ Reassurance and discharge
- ○ Topical aciclovir
- ○ Topical chloramphenicol
- ○ Topical corticosteroids
- ○ Topical timolol

Submit answer

Reference ranges

Score: **20.8%**

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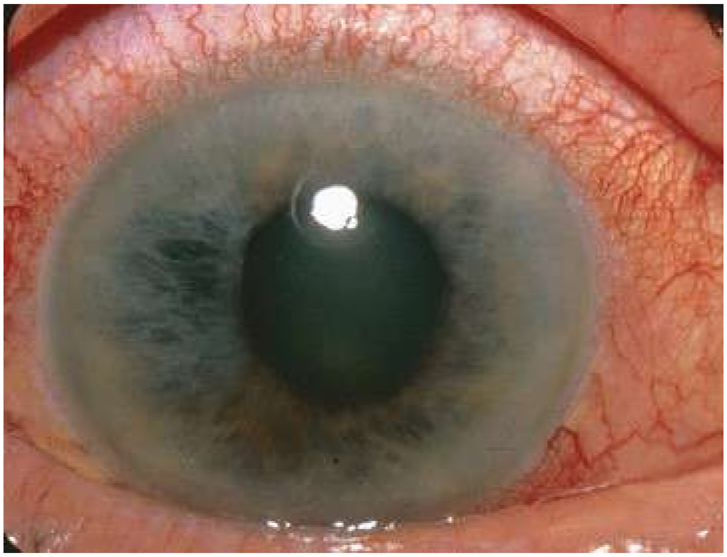
Question 131 of 189 ✓

A 50-year-old man presents to the emergency department with a painful right eye. The pain started approximately 4 hours ago whilst watching a movie. He also reports that his vision is blurry in the same eye. His past medical history includes type 2 diabetes and rheumatoid arthritis.

His eye is examined, as shown below:



What is the most appropriate management for this patient?

Reassurance and discharge	3%
Topical aciclovir	3%
Topical chloramphenicol	3%
Topical corticosteroids	26%
Topical timolol ✓	65%

Topical timolol is correct. This patient's right eye is erythematous with a semi-dilated pupil. Combined with the clinical history and examination findings of reduced visual acuity, the patient has a likely diagnosis of acute angle-closure glaucoma. Hypermetropia (long-sightedness) is also a risk factor for acute angle-closure glaucoma. The management consists of reducing the intraocular pressure with a combination of medications to reduce aqueous humour production and increase uveoscleral outflow. Timolol is a beta-blocker eye drop that decreases aqueous humour production. Of note, acute angle-closure glaucoma is an ophthalmological emergency and requires urgent referral to ophthalmology. A combination of drugs is required in addition to timolol including, a muscarinic agonist eye drop (e.g. pilocarpine) and intravenous acetazolamide.

Reassurance and discharge is incorrect. Acute angle-closure glaucoma is an ophthalmological emergency and discharging home without intervention can result in blindness in the affected eye.

Topical aciclovir is incorrect. This is used in the management of viral ocular infections (e.g. herpes simplex virus). This usually presents with eye pain, watering and irritation. The eye may be erythematous and there are occasionally ulcers or vesicles seen along the eyelid margins. Examination of the eye with blue light after fluorescein administration may reveal the presence of a dendritic ulcer.

Topical chloramphenicol is incorrect. This is used in the management of bacterial conjunctivitis and keratitis. Keratitis is a potential differential diagnosis for this patient given the history of contact lens use. Symptoms of keratitis include erythema, photophobia, pain and lacrimation that are similar to this patient. However, it does not cause pupil dilatation making acute angle-closure glaucoma more likely.

Topical corticosteroids is incorrect. This is used in the management of anterior uveitis that presents as ocular pain, erythema and reduced visual acuity. Anterior uveitis is often associated with inflammatory conditions (e.g. ankylosing spondylitis). However, a change in pupil size is not a feature of anterior uveitis making it less likely to be the underlying diagnosis.

Discuss (2) | Improve

Next question

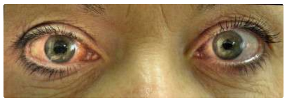
Acute angle-closure glaucoma

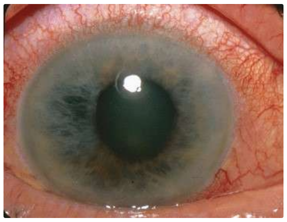
Glaucoma is a group of disorders characterised by optic neuropathy due, in the majority of patients, to raised intraocular pressure (IOP). It is now recognised that a minority of patients with raised IOP do not have glaucoma and vice versa.

In acute angle-closure glaucoma (AACG) there is a rise in IOP secondary to an impairment of aqueous outflow. Factors predisposing to AACG include:
- hypermetropia (long-sightedness)
- pupillary dilatation
- lens growth associated with age

Features

- severe pain: may be ocular or headache
- decreased visual acuity
- symptoms worse with mydriasis (e.g. watching TV in a dark room)
- hard, red-eye
- haloes around lights
- semi-dilated non-reacting pupil
- corneal oedema results in dull or hazy cornea
- systemic upset may be seen, such as nausea and vomiting and even abdominal pain





Investigations

- tonometry to assess for elevated IOP
- gonioscopy (literally looking, *oscopy*, at the angle, *gonio*): a special lens for the slit lamp that allows visualisation of the angle

Management

The management of AACG is an emergency and should prompt urgent referral to an ophthalmologist. Emergency medical treatment is required to lower the IOP with more definitive surgical treatment given once the acute attack has settled.

There are no guidelines for the initial medical treatment emergency treatment. An example regime would be:

- combination of eye drops, for example:
    - a direct parasympathomimetic (e.g. pilocarpine, causes contraction of the ciliary muscle → opening the trabecular meshwork → increased outflow of the aqueous humour)
    - a beta-blocker (e.g. timolol, decreases aqueous humour production)
    - an alpha-2 agonist (e.g. apraclonidine, dual mechanism, decreasing aqueous humour production and increasing uveoscleral outflow)
- intravenous acetazolamide
    - reduces aqueous secretions
- some guidelines also recommend the use of topical steroids to reduce inflammation

Definitive management

- laser peripheral iridotomy
    - creates a tiny hole in the peripheral iris → aqueous humour flowing to the angle

123

Next question >

Textbooks

High-yield textbook

Extended textbook

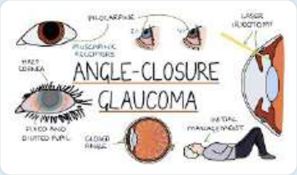
Links

Patient.info 👍 27 👎 9

Acute angle closure glaucoma review

Suggest link Report broken link

Media


ANGLE-CLOSURE
GLAUCOMA
PILOCARPINE
LASER IRIDOTOMY
MUSCARINIC RECEPTORS
HAZY CORNEA
FIXED AND DILATED PUPIL
CLOSED ANGLE
INITIAL MANAGEMENT

Acute angle closure glaucoma

Zero To Finals - YouTube 👍 4 👎 0

GLAUCOMA
Osmosis.org

Glaucoma (open-angle, closed-angle, and normal-tension)

Osmosis - YouTube 👍 6 👎 3

Report broken media

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A 80 year old woman is referred to neurology clinic after experiencing increasing diplopia, typically worsening during the course of the day. The patient reported no speech or swallowing problems and no limb weakness.

Past medical history included chronic obstructive pulmonary disease and ischaemic heart disease. The patient was able to mobilise around her flat with a frame but was required to use a wheelchair outside the home due to exertional breathlessness. Regular medications were inhaled salbutamol and tiotropium, aspirin, simvastatin, bisoprolol and ramipril. The patient lived with her husband and had once daily carers to assist with activities of daily living.

Examination in clinic was significant for the development of ptosis on prolonged upwards gaze. There was no significant weakness of facial muscles, palate or tongue. There was no evidence of fatiguable weakness in the arms or legs.

A summary of the patients investigations is given below.

Serum acetylcholine receptor antibodies	negative
Serum muscle specific tyrosine kinase	positive

Neurophysiology: no evidence of repetitive nerve stimulation

CT thorax: retro-sternal soft-tissue density mass equal in attenuation to muscle; mass demonstrates heterogeneous enhancement following contrast injection

What is the appropriate management of the retro-sternal mass?

- ○ Proceed to thymectomy
- ○ Proceed to thymectomy if patient does not respond to first line treatment
- ○ Surgical biopsy
- ○ No action required
- ○ Trans-bronchial biopsy

Submit answer

Reference ranges

Score: **20.8%**

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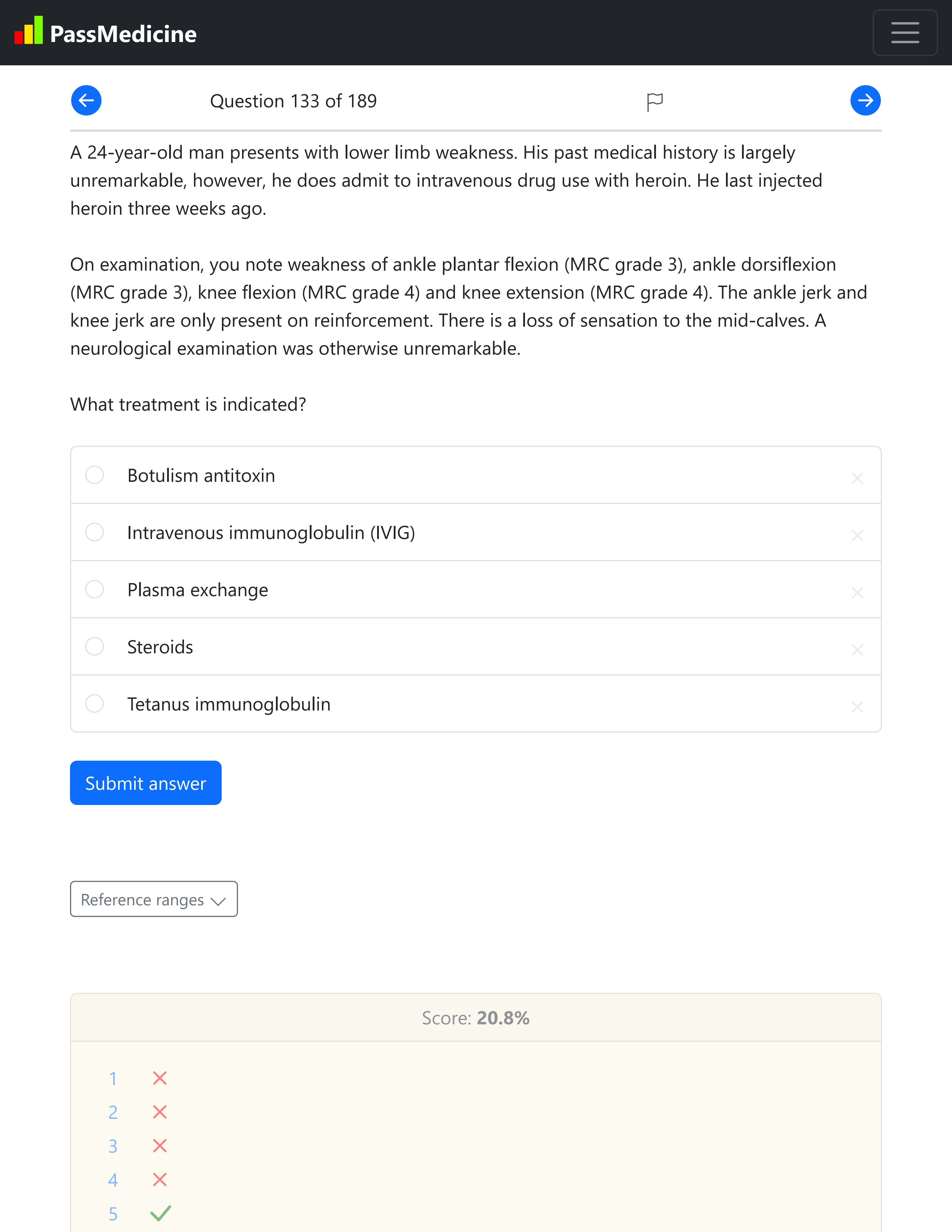

Question 133 of 189

A 24-year-old man presents with lower limb weakness. His past medical history is largely unremarkable, however, he does admit to intravenous drug use with heroin. He last injected heroin three weeks ago.

On examination, you note weakness of ankle plantar flexion (MRC grade 3), ankle dorsiflexion (MRC grade 3), knee flexion (MRC grade 4) and knee extension (MRC grade 4). The ankle jerk and knee jerk are only present on reinforcement. There is a loss of sensation to the mid-calves. A neurological examination was otherwise unremarkable.

What treatment is indicated?

- ○ Botulism antitoxin
- ○ Intravenous immunoglobulin (IVIG)
- ○ Plasma exchange
- ○ Steroids
- ○ Tetanus immunoglobulin

Score: **20.8%**

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A 80 year old woman is referred to neurology clinic after experiencing increasing diplopia, typically worsening during the course of the day. The patient reported no speech or swallowing problems and no limb weakness.

Past medical history included chronic obstructive pulmonary disease and ischaemic heart disease. The patient was able to mobilise around her flat with a frame but was required to use a wheelchair outside the home due to exertional breathlessness. Regular medications were inhaled salbutamol and tiotropium, aspirin, simvastatin, bisoprolol and ramipril. The patient lived with her husband and had once daily carers to assist with activities of daily living.

Examination in clinic was significant for the development of ptosis on prolonged upwards gaze. There was no significant weakness of facial muscles, palate or tongue. There was no evidence of fatiguable weakness in the arms or legs.

A summary of the patients investigations is given below.

Serum acetylcholine receptor antibodies	negative
Serum muscle specific tyrosine kinase	positive

Neurophysiology: no evidence of repetitive nerve stimulation

CT thorax: retro-sternal soft-tissue density mass equal in attenuation to muscle; mass demonstrates heterogeneous enhancement following contrast injection

What is the appropriate management of the retro-sternal mass?

Proceed to thymectomy	48%
Proceed to thymectomy if patient does not respond to first line treatment	31%
Surgical biopsy	3%
✓ No action required	12%
✕ Trans-bronchial biopsy	5%

Thymectomy is a well recognised treatment for myasthenia gravis and should also be considered in non-thymomatous generalised myasthenia in patients with antibodies to acetylcholine receptor who are aged under 50. Biopsy is not generally required prior to surgery.

However, thymectomy is not generally carried out in myasthenia gravis when patients have antibodies to MUSK, late onset disease or purely ocular disease as in this case. The patient's poor performance status would also argue against surgical intervention.

Repetitive nerve stimulation is a specific test for myasthenia gravis but has a relatively low sensitivity, especially in ocular only disease.

Spillane J, Higham E, Kullman D. Myasthenia gravis. BMJ 2012;345:e8497.

👍 👎 Discuss (16) Improve

Next question >

Myasthenia gravis ★

Myasthenia gravis is an autoimmune disorder resulting in insufficient functioning acetylcholine receptors. Antibodies to acetylcholine receptors are seen in 85-90% of cases*. Myasthenia is more common in women (2:1)

The key feature is muscle fatigability - muscles become progressively weaker during periods of activity and slowly improve after periods of rest:

- extraocular muscle weakness: diplopia
- proximal muscle weakness: face, neck, limb girdle
- ptosis
- dysphagia

Associations

- thymomas in 15%
- autoimmune disorders: pernicious anaemia, autoimmune thyroid disorders, rheumatoid, SLE
- thymic hyperplasia in 50-70%

Investigations

- single fibre electromyography: high sensitivity (92-100%)
- CT thorax to exclude thymoma
- CK normal
- antibodies to acetylcholine receptors
    - positive in around 85-90% of patients

  - n the remaining patients, about about 40% are positive for anti-muscle-specific tyrosine kinase antibodies
- Tensilon test: IV edrophonium reduces muscle weakness temporarily - not commonly used any more due to the risk of cardiac arrhythmia

Management

- long-acting acetylcholinesterase inhibitors
  - pyridostigmine is first-line
- immunosuppression is usually not started at diagnosis, but the majority of patients eventually require it in addition to long-acting acetylcholinesterase inhibitors:
  - prednisolone initially
  - azathioprine, cyclosporine, mycophenolate mofetil may also be used
- thymectomy

Management of myasthenic crisis

- plasmapheresis
- intravenous immunoglobulins

*antibodies are less commonly seen in disease limited to the ocular muscles

AI + Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

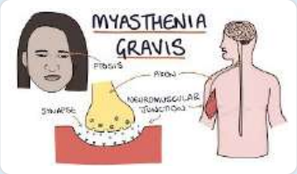
Links

Postgraduate Medical Journal 👍 13 👎 3

Review of myasthenia gravis

Suggest link Report broken link

Media


MYASTHENIA GRAVIS
PTOSIS
AXON
NEUROMUSCULAR JUNCTION
SYNAPSE

Myasthenia gravis

Zero to Finals - YouTube 👍 12 👎 1

NEUROLOGY
MYASTHENIA GRAVIS

Myasthenia gravis

Armando Hasudungan - YouTube 👍 1 👎 1

MYASTHENIA GRAVIS

Myasthenia gravis - causes, symptoms, treatment, pathology

Osmosis - YouTube 👍 2 👎 2

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A 23-year-old university student presented with a year long history of occipital headache. This was worse on coughing, sneezing and straining and partially relieved by lying flat. On one occasion the headache had been associated with vomiting. She had previously consulted her family doctor who was treating her for migraine. Over the past two months she had noticed pain in her both arms and felt unsteady on her feet.

On examination touching her arms caused pain and there was reduced appreciation of pinprick and temperature sensation throughout both arms. Tone, power and reflexes in the upper limbs were normal. On testing upper limb co-ordination there was some past-pointing and a very mild intention tremor. On inspection of the feet there was pes cavus and Rombergs test was positive. Again tone, power and reflexes were normal in the lower limbs and plantars were downgoing. Proprioception was impaired with absent joint position sense until the level of the knee. Vibration sensation was impaired in both feet.

Given the clinical findings outlined above which of the following is the most likely diagnosis?

- ○ Syringomyelia
- ○ Friedrichs ataxia
- ○ Brown-Sequard syndrome
- ○ Charcot-Marie-Tooth disease
- ○ Multiple sclerosis

Submit answer

Reference ranges

Score: **20.8%**

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Question 133 of 189

A 24-year-old man presents with lower limb weakness. His past medical history is largely unremarkable, however, he does admit to intravenous drug use with heroin. He last injected heroin three weeks ago.

On examination, you note weakness of ankle plantar flexion (MRC grade 3), ankle dorsiflexion (MRC grade 3), knee flexion (MRC grade 4) and knee extension (MRC grade 4). The ankle jerk and knee jerk are only present on reinforcement. There is a loss of sensation to the mid-calves. A neurological examination was otherwise unremarkable.

What treatment is indicated?

Botulism antitoxin	35%
Intravenous immunoglobulin (IVIG)	46%
Plasma exchange	5%
Steroids	7%
Tetanus immunoglobulin	7%

IVIG is generally used first-line for Guillain-Barre syndrome

Important for me Less important

Intravenous immunoglobulin (IVIG) is correct. The most likely diagnosis is Guillain-Barre syndrome. IVIG is generally used as first-line management for Guillain-Barre syndrome.

Botulism antitoxin is incorrect. Botulism is characterised by a descending, flaccid paralysis, in contrast to Guillain-Barre syndrome which presents with an ascending paralysis. It is important to note that the patient is an intravenous drug user which is a risk factor for botulism.

Plasma exchange is incorrect. Although plasma exchange is just as effective as IVIG, it has more risks associated with it and is much more labour-intensive. For these reasons, IVIG is usually used first-line.

Steroids is incorrect. Several trials have shown no benefit of steroids or immunosuppressants in Guillain-Barre syndrome.

Tetanus immunoglobulin is incorrect. Tetanus is a life-threatening disease characterised by a descending spastic paralysis. Although the causative bacteria *Clostridium tetani* has been found in contaminated heroin, the clinical features of an ascending paralysis are more suggestive of Guillain-Barre syndrome.

Discuss (7) Improve

Next question >

Guillain-Barre syndrome: management

Guillain-Barre syndrome describes an immune-mediated demyelination of the peripheral nervous system often triggered by an infection (classically *Campylobacter jejuni*).

Management
- IV immunoglobulins (IVIG) or plasma exchange can be used
  - IVIG is as effective as plasma exchange better tolerated and easier to administer
  - therefore IVIG is generally used first-line for Guillain-Barre syndrome where patients require immunomodulatory therapy
  - no benefit in combining both treatments
- steroids and immunosuppressants have not been shown to be beneficial
- FVC regularly to monitor respiratory function

Prognosis
- severe motor problems persist in about 15%
- around 5% die

+ Q 123

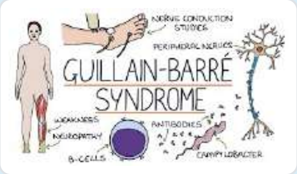
Next question >

Textbooks

High-yield textbook

Extended textbook

Media


GUILLAIN-BARRÉ SYNDROME
NERVE CONDUCTION STUDIES
PERIPHERAL NERVES
WEAKNESS
NEUROPATHY
B-CELLS
ANTIBODIES
CAMPYLOBACTER

Understanding Guillain-Barre Syndrome

Zero To Finals - YouTube 👍 3 👎 1

Guillain-Barre syndrome

Osmosis - YouTube 👍 1 👎 1

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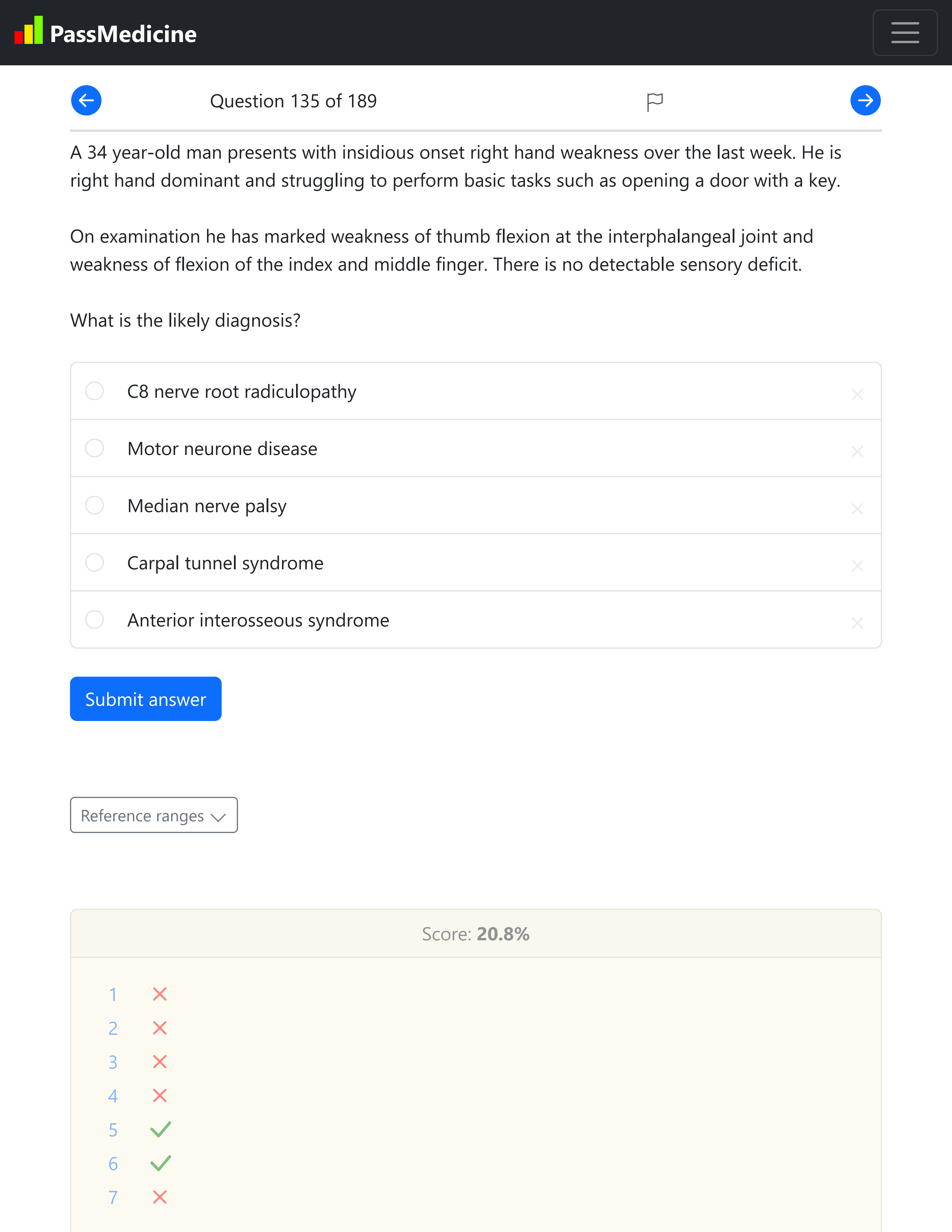

Question 135 of 189

A 34 year-old man presents with insidious onset right hand weakness over the last week. He is right hand dominant and struggling to perform basic tasks such as opening a door with a key.

On examination he has marked weakness of thumb flexion at the interphalangeal joint and weakness of flexion of the index and middle finger. There is no detectable sensory deficit.

What is the likely diagnosis?

- ○ C8 nerve root radiculopathy
- ○ Motor neurone disease
- ○ Median nerve palsy
- ○ Carpal tunnel syndrome
- ○ Anterior interosseous syndrome

Score: **20.8%**

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A 23-year-old university student presented with a year long history of occipital headache. This was worse on coughing, sneezing and straining and partially relieved by lying flat. On one occasion the headache had been associated with vomiting. She had previously consulted her family doctor who was treating her for migraine. Over the past two months she had noticed pain in her both arms and felt unsteady on her feet.

On examination touching her arms caused pain and there was reduced appreciation of pinprick and temperature sensation throughout both arms. Tone, power and reflexes in the upper limbs were normal. On testing upper limb co-ordination there was some past-pointing and a very mild intention tremor. On inspection of the feet there was pes cavus and Rombergs test was positive. Again tone, power and reflexes were normal in the lower limbs and plantars were downgoing. Proprioception was impaired with absent joint position sense until the level of the knee. Vibration sensation was impaired in both feet.

Given the clinical findings outlined above which of the following is the most likely diagnosis?

Syringomyelia	37%
Friedrichs ataxia	25%
Brown-Sequard syndrome	3%
Charcot-Marie-Tooth disease	32%
Multiple sclerosis	3%

Syringomyelia results from the formation of a fluid filled cavitation (called a syrinx) within the central canal of the spinal cord. The most common cause of a syrinx is obstruction to the flow of cerebrospinal fluid (CSF) by an Arnold Chiari malformation. An Arnold Chiari malformation is a developmental abnormality in which the cerebellar tonsils project through the foramen magnum and sit in a position which is normally occupied by the cervical spinal cord.

In this case the patient has features suggestive of an underlying Arnold Chiari malformation: occipital headache exacerbated by valsalva manoeuvre (coughing, sneezing and straining), pes cavus and cerebellar signs (past pointing and intention tremor in the upper limbs).

She also has features of an associated syringomyelia. The spinothalamic tracts are involved early in the process due to their decussation within the cord. This means that there is loss of pain and temperature sensation corresponding to the level of the syrinx. This often occurs in a shawl-like distribution. Hyperalgesia and allodynia are also seen in the upper limbs in some cases.

As the syrinx enlarges the dorsal columns are affected resulting in loss of proprioception and vibration sensation in the lower limbs. This explains the positive Rombergs test which indicates that there is a proprioceptive problem.

If the syrinx expands further the anterior horn cells can be affected resulting in wasting and weakness in the hands.

Friedrichs ataxia is an autosomal recessive inherited condition in which there is progressive ataxia, dysarthria, loss of proprioceptive and vibration sensation and weakness.

Brown-Sequard syndrome results from a lateral hemisection of the spinal cord. It presents with a loss of pain and temperature sensation on the side of the body contralateral to the lesion and a hemiplegia and loss of vibration sensation and proprioception on the ipsilateral side to the lesion.

Charcot-Marie-Tooth (also known as Hereditary Sensory and Motor neuropathy) encompasses a group of inherited conditions which present in mid-adult life with peripheral neuropathy. Patients commonly have pes cavus and often present with foot drop.

Multiple sclerosis (MS) can present in a variety of ways with cerebellar features, optic neuritis, paraesthesiae and tranverse myelitis all being reported. Although in theory many of the clinical features in this question could be explained by an underlying diagnosis of MS, the clinical presentation here is typical of syringomyelia secondary to an underlying Arnold Chiari malformation.

👍 👎 Discuss (14) Improve

Next question >

Syringomyelia ★

Syringomyelia ('syrinx' for short) describes a collection of cerebrospinal fluid within the spinal cord.

Syringobulbia is a similar phenomenon in which there is a fluid-filled cavity within the medulla of the brainstem. This is often an extension of the syringomyelia but in rare cases can be an isolated finding.

Causes include:

- a Chiari malformation: strong association
- trauma
- tumours
- idiopathic

Features

- a 'cape-like' (neck, shoulders and arms)
    - loss of sensation to temperature but the preservation of light touch, proprioception and vibration
    - classic examples are of patients who accidentally burn their hands without realising
    - this is due to the crossing spinothalamic tracts in the anterior commissure of the spinal cord being the first tracts to be affected
- spastic weakness (predominantly of the lower limbs)
- neuropathic pain
- upgoing plantars
- autonomic features:
- Horner's syndrome due to compression of the sympathetic chain, but this is rare
    - bowel and bladder dysfunction
- scoliosis will occur over a matter of years if the syrinx is not treated

Investigations

- full spine MRI with contrast to exclude a tumour or tethered cord
- a brain MRI is also needed to exclude a Chiari malformation

Treatment will be directed at treating the cause of the syrinx. In patients with a persistent or symptomatic syrinx, a shunt into the syrinx can be placed.

+ Q 123

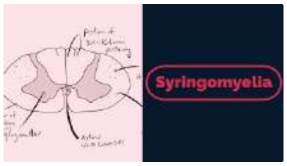
Next question >

Textbooks

High-yield textbook

Extended textbook

Media



Syringomyelia

Soton Brain Hub - YouTube 👍 11 👎 0

Report broken media

Score: **20%**

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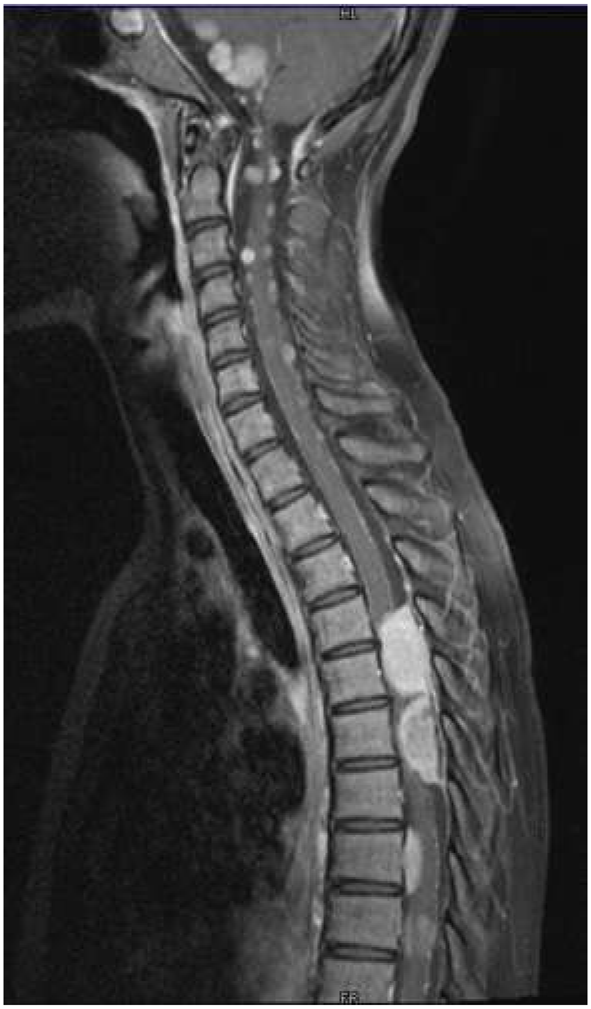
Question 136 of 189

A 24-year-old woman is referred urgently to neurology for lower limb weakness. This has been getting gradually worse over the past 4 weeks and she is now experiencing problems walking.

She also complains of bilateral tinnitus which has been present for the past 3 months. This is associated with episodes of vertigo.

On examination, both lower limbs have reduced power (3/5), with increased tone and hyperreflexia. The patient has one 1cm tan lesion on her torso but no other skin lesions are noted.

An MRI of her spine is requested:



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What is the most likely diagnosis?

○ Multiple sclerosis

○ Neurofibromatosis ×

○ Von Hippel-Lindau disease ×

○ Tuberous sclerosis ×

○ Ataxia telangiectasia ×

Submit answer

Reference ranges ∨

Score: **20.8%**

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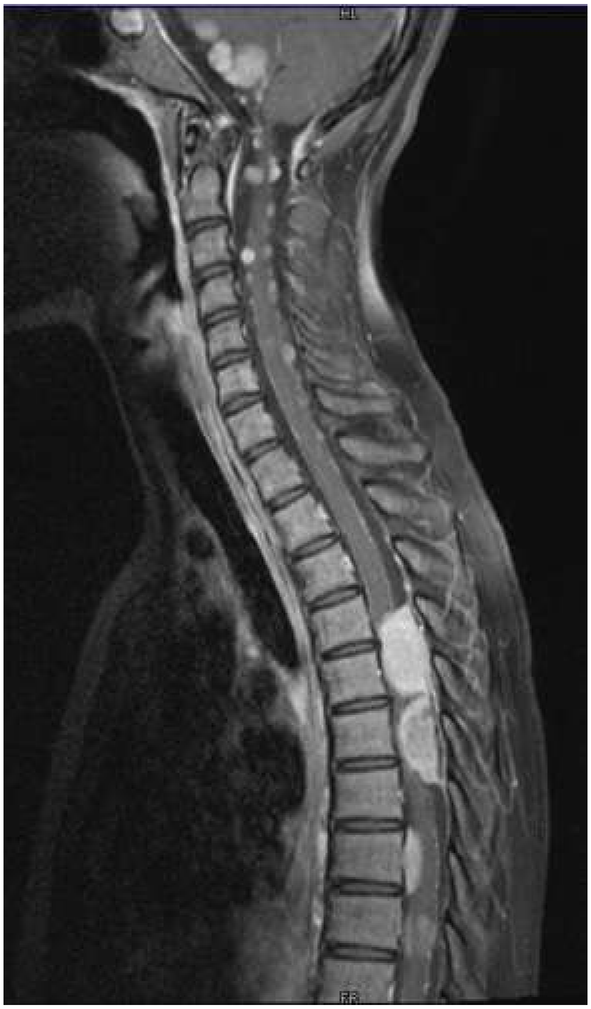

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What is the most likely diagnosis?

Multiple sclerosis 18%

Neurofibromatosis	44%
Von Hippel-Lindau disease	13%
Tuberous sclerosis	23%
Ataxia telangiectasia	2%

This patient had neurofibromatosis type 2 (NF2). The MRI shows multiple tumours of the spinal cord in the context of the syndrome of multiple intracranial schwannomas, mengiomas and ependymomas.

Patients with NF2, in contrast to type 1, often have few skin changes. This patient had tinnitus due to the presence of bilateral acoustic neuromas.

The **diagnostic criteria** for NF2 are:

Bilateral vestibular schwannomas

A first degree relative with NF2 AND
- Unilateral vestibular schwannoma OR
- Any two of: meningioma, schwannoma, glioma, neurofibroma, posterior subcapsular lenticular opacities

Unilateral vestibular schwannoma AND
- Any two of: meningioma, schwannoma, glioma, neurofibroma, posterior subcapsular lenticular opacities

Multiple meningiomas AND
- Unilateral vestibular schwannoma OR
- Any two of: schwannoma, glioma, neurofibroma, cataract

👍 👎 Discuss (2) Improve

Next question >

Neurofibromatosis ★

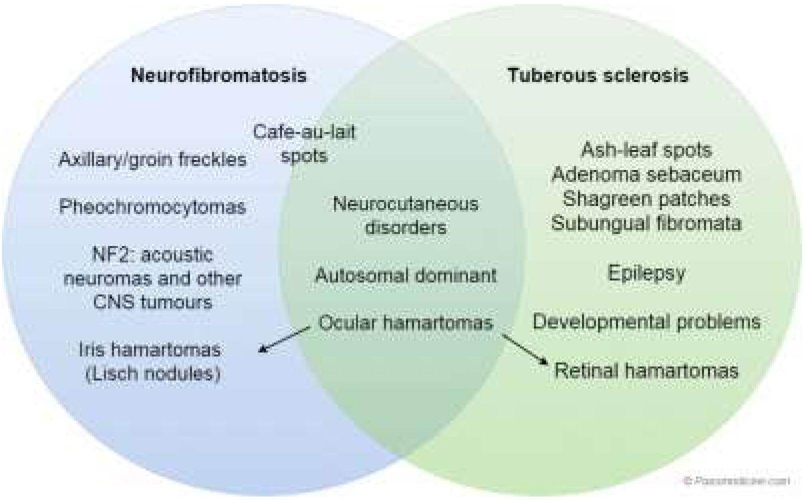
There are two types of neurofibromatosis, NF1 and NF2. Both are inherited in an autosomal dominant fashion

NF1 is also known as von Recklinghausen's syndrome. It is caused by a gene mutation on chromosome 17 which encodes neurofibromin and affects around 1 in 4,000

NF2 is caused by gene mutation on chromosome 22 and affects around 1 in 100,000

Features

NF1	**NF2**
Cafe-au-lait spots (>= 6, 15 mm in diameter) Axillary/groin freckles Peripheral neurofibromas Iris hamatomas (Lisch nodules) in > 90% Scoliosis Pheochromocytomas	Bilateral vestibular schwannomas Multiple intracranial schwannomas, mengiomas and ependymomas


Neurofibromatosis
Tuberous sclerosis
Cafe-au-lait spots
Axillary/groin freckles
Pheochromocytomas
NF2: acoustic neuromas and other CNS tumours
Iris hamartomas (Lisch nodules)
Neurocutaneous disorders
Autosomal dominant
Ocular hamartomas
Ash-leaf spots
Adenoma sebaceum
Shagreen patches
Subungual fibromata
Epilepsy
Developmental problems
Retinal hamartomas
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Comparison of neurofibromatosis and tuberous sclerosis. Note that whilst they are both autosomal dominant neurocutaneous disorders there is little overlap otherwise

Next question

Textbooks

High-yield textbook

Extended textbook

Links

National Center for Biotechnology Information 👍 6 👎 5

Guidelines for the diagnosis and management of individuals with neurofibromatosis 1

Suggest link

Report broken link

Score: **20%**

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Back to top

A 65-year-old male presents to the neurology outpatient department with a history of recurrent bouts of unsteadiness and vomiting over the last 10 years, with partial resolution. He has also had episodes of visual problems, which he describes as the sudden loss of vision in the right eye, with an almost complete recovery of vision over the course of the next few weeks. He has a history of type 2 diabetes and is hypertensive and is generally non-compliant with his treatment. He is also a smoker with a 50 pack year history.

His medication includes glimepiride 2mg daily and metformin 500mg TDS. He also takes telmisartan 40mg daily.

On examination, he has nystagmus in the right eye with the fast component towards the right. His gait is ataxic and he has evidence of spasticity in both lower limbs with exaggerated reflexes and bilateral ankle clonus. Fundoscopic examination revealed a pale optic disc.

MRI brain shows diffuse lesions in multiple sites. The report queried demyelinating plaques vs multiple infarcts.

Which of the following would be the most appropriate next investigation?

○ MRI spinal cord ×

○ CSF examination for oligoclonal bands ×

○ MR angiography of the verterbrobasilar system ×

○ Titres of anti-Hu antibodies ×

○ Visual evoked potentials ×

Submit answer

Reference ranges

Score: **20.8%**

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MRI brain shows diffuse lesions in multiple sites. The report queried demyelinating plaques vs multiple infarcts.

Which of the following would be the most appropriate next investigation?

MRI spinal cord	38%
CSF examination for oligoclonal bands	36%
MR angiography of the verterbrobasilar system	11%
Titres of anti-Hu antibodies	7%
Visual evoked potentials	9%

The diagnosis, in this case, is that of multiple sclerosis and the question aims to assess the candidate's ability to plan further investigations if the MRI of the brain is inconclusive.

An MRI of the brain is a sensitive imaging modality for the detection of plaques in patients with MS, but it lacks specificity. Over the age of 50, small ischaemic lesions may be difficult to distinguish from demyelination. The presence of spinal cord lesions, however, is quite specific for inflammatory disorders such as MS rather than ischaemic lesions. Thus cord imaging is useful when there is diagnostic difficulty.

Oligoclonal bands are found in the CSF in >90% of cases of MS but are not specific for MS. They are also found in

- Lyme disease
- Systemic lupus erythematosus
- Neurosarcoidosis
- Subacute sclerosing panencephalitis
- Subarachnoid hemorrhage
- Syphilis
- Primary central nervous system lymphoma
- Sjogren's syndrome
- Guillain-Barre syndrome
- Meningeal carcinomatosis
- Neuromyelitis optica

👍 👎 Discuss (10) Improve

Next question >

Multiple sclerosis: investigation ★

Diagnosis requires demonstration of lesions disseminated in time and space

MRI

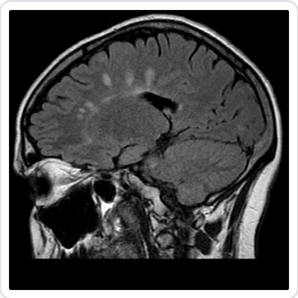
- high signal T2 lesions
- periventricular plaques
- Dawson fingers: often seen on FLAIR images - hyperintense lesions penpendicular to the corpus callosum

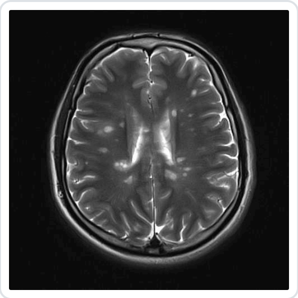
CSF

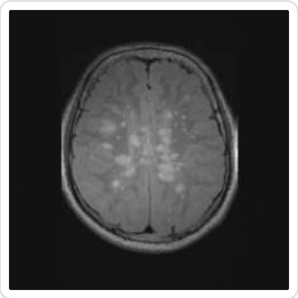
- oligoclonal bands (and not in serum)
- increased intrathecal synthesis of IgG

Visual evoked potentials

- delayed, but well preserved waveform







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Next question >

Textbooks

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Extended textbook

Media

DEMYELINATION
T-CELLS
PLAQUES
MULTIPLE SCLEROSIS
OLIGO CLONAL BANDS
RELAPSING-REMITTING

Multiple sclerosis

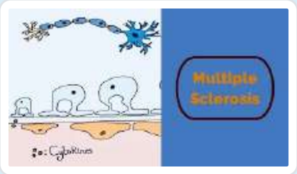
Rhesus Medicine - YouTube 👍 1 👎 0

MULTIPLE SCLEROSIS

Multiple sclerosis - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube

👍 6 👎 1


Multiple Sclerosis

Multiple sclerosis

Soton Brain Hub - YouTube 👍 2 👎 1

Multiple sclerosis

Podmedics - YouTube 👍 1 👎 3

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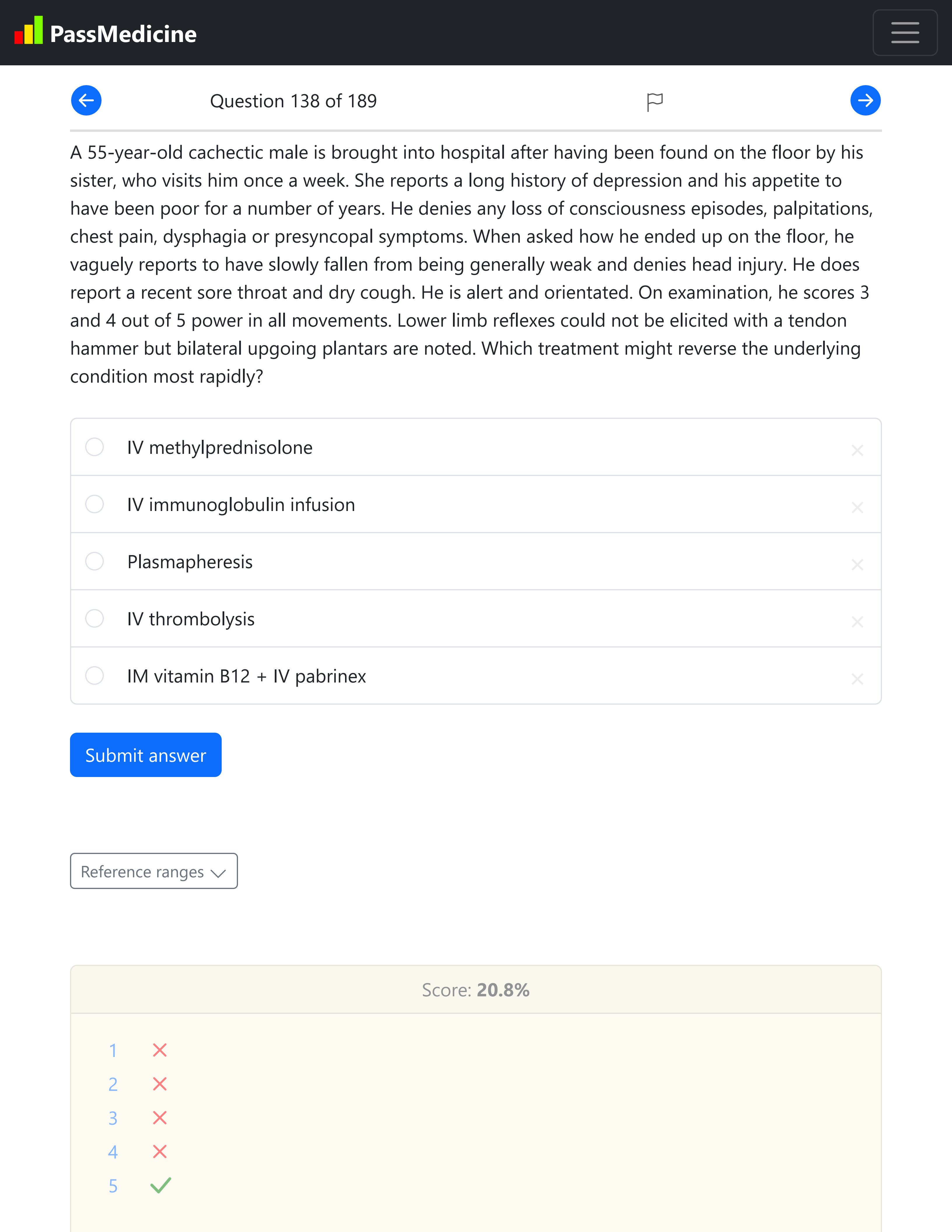

Question 138 of 189

A 55-year-old cachectic male is brought into hospital after having been found on the floor by his sister, who visits him once a week. She reports a long history of depression and his appetite to have been poor for a number of years. He denies any loss of consciousness episodes, palpitations, chest pain, dysphagia or presyncopal symptoms. When asked how he ended up on the floor, he vaguely reports to have slowly fallen from being generally weak and denies head injury. He does report a recent sore throat and dry cough. He is alert and orientated. On examination, he scores 3 and 4 out of 5 power in all movements. Lower limb reflexes could not be elicited with a tendon hammer but bilateral upgoing plantars are noted. Which treatment might reverse the underlying condition most rapidly?

○ IV methylprednisolone ×

○ IV immunoglobulin infusion ×

○ Plasmapheresis ×

○ IV thrombolysis ×

○ IM vitamin B12 + IV pabrinex ×

Score: **20.8%**

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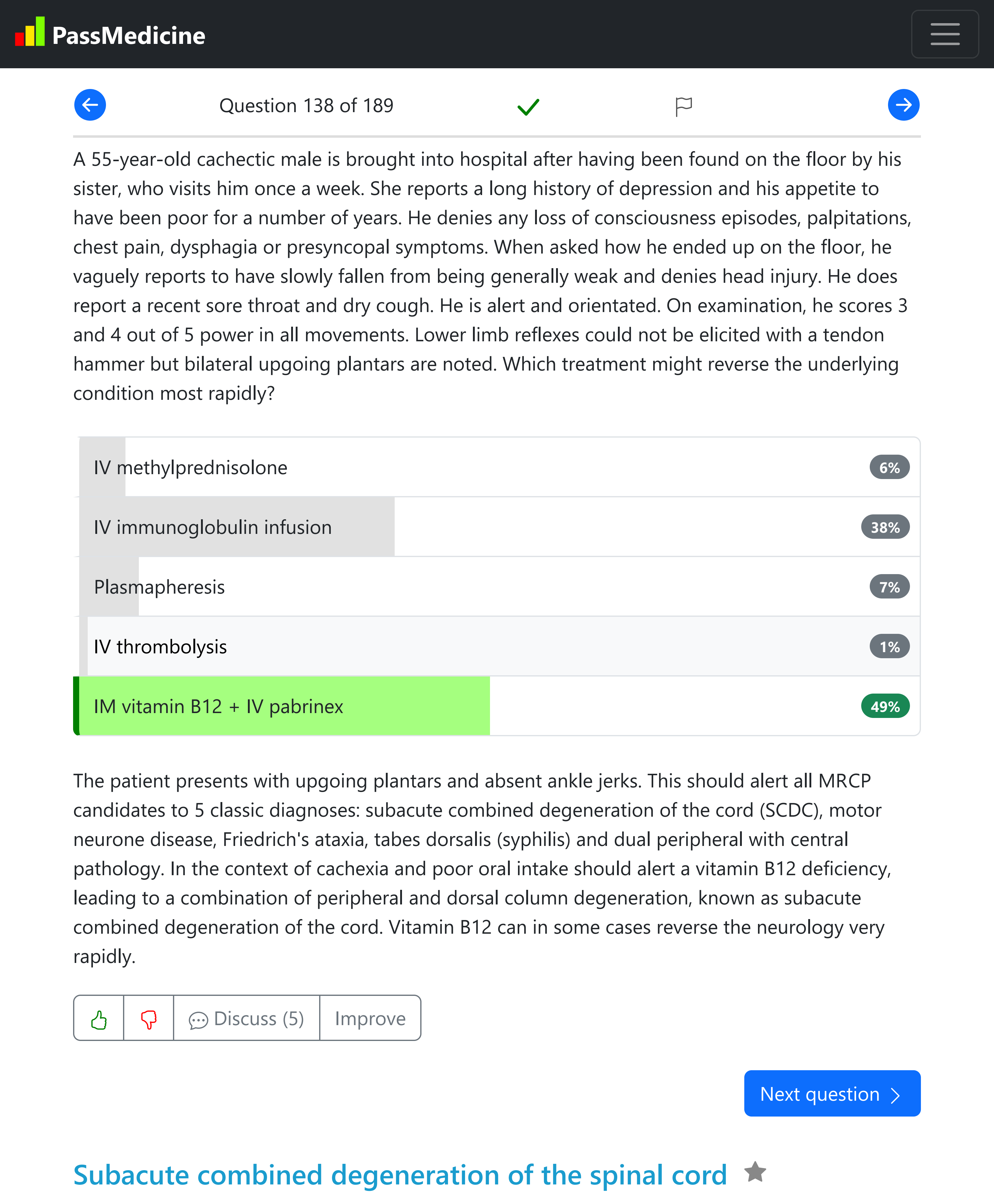

A 55-year-old cachectic male is brought into hospital after having been found on the floor by his sister, who visits him once a week. She reports a long history of depression and his appetite to have been poor for a number of years. He denies any loss of consciousness episodes, palpitations, chest pain, dysphagia or presyncopal symptoms. When asked how he ended up on the floor, he vaguely reports to have slowly fallen from being generally weak and denies head injury. He does report a recent sore throat and dry cough. He is alert and orientated. On examination, he scores 3 and 4 out of 5 power in all movements. Lower limb reflexes could not be elicited with a tendon hammer but bilateral upgoing plantars are noted. Which treatment might reverse the underlying condition most rapidly?

IV methylprednisolone	6%
IV immunoglobulin infusion	38%
Plasmapheresis	7%
IV thrombolysis	1%
IM vitamin B12 + IV pabrinex ✓	49%

The patient presents with upgoing plantars and absent ankle jerks. This should alert all MRCP candidates to 5 classic diagnoses: subacute combined degeneration of the cord (SCDC), motor neurone disease, Friedrich's ataxia, tabes dorsalis (syphilis) and dual peripheral with central pathology. In the context of cachexia and poor oral intake should alert a vitamin B12 deficiency, leading to a combination of peripheral and dorsal column degeneration, known as subacute combined degeneration of the cord. Vitamin B12 can in some cases reverse the neurology very rapidly.

Subacute combined degeneration of the spinal cord

Subacute combined degeneration of the spinal cord is due to vitamin B12 deficiency resulting in impairment of the dorsal columns, lateral corticospinal tracts and spinocerebellar tracts..

Recreational nitrous oxide inhalation may also result in vitamin B12 deficiency → subacute combined degeneration of the spinal cord.

Features

- dorsal column involvement
  - distal tingling/burning/sensory loss is symmetrical and tends to affect the legs more than the arms
  - impaired proprioception and vibration sense
- lateral corticospinal tract involvement
  - muscle weakness, hyperreflexia, and spasticity
  - upper motor neuron signs typically develop in the legs first
  - brisk knee reflexes
  - absent ankle jerks
  - extensor plantars
- spinocerebellar tract involvement
  - sensory ataxia → gait abnormalities
  - positive Romberg's sign

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20%**

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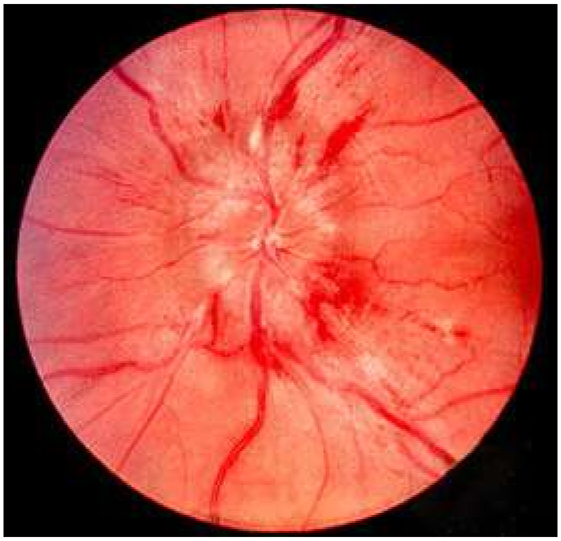
A 44-year-old woman presents to the emergency department with a 2-week history of worsening headaches. She also complains of occasional bouts of dizziness and double vision. Her past medical history includes borderline type 2 diabetes and her regular medications include metformin and the combined contraceptive pill.

On examination, her observations are within normal limits. Her BMI is 32 kg/m². There is no focal neurology to find on examination. Fundoscopy is carried out (as shown below):



Given the finding from this patient's fundoscopy, what is the next step in the management of this patient?

- ○ CT head
- ○ Reassurance and routine ophthalmology follow up in 2 weeks
- ○ Topical corticosteroids
- ○ Urgent lumbar puncture
- ○ Urgent referral to ophthalmology

Submit answer

Reference ranges ⌵

Score: **20.8%**

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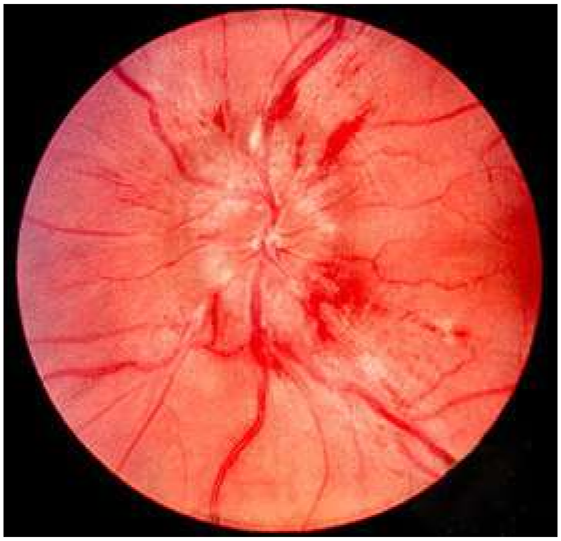


Given the finding from this patient's fundoscopy, what is the next step in the management of this patient?

CT head	46%
Reassurance and routine ophthalmology follow up in 2 weeks	2%
Topical corticosteroids	1%
Urgent lumbar puncture	23%
Urgent referral to ophthalmology	28%

This patient has papilloedema on fundoscopic examination, as seen with the enlarged optic disc with blurred margins and venous congestion. Papilloedema arises secondary to raised intracranial pressure (e.g. inflammation, infection, malignancy or idiopathic intracranial hypertension). Given this finding on examination, in addition to the symptoms of headache, dizziness and blurred vision, it is most important to complete a **CT head** to rule out any space-occupying lesion that may be causing raised intracranial pressure.

Reassurance and routine ophthalmology follow-up in 2 weeks is incorrect. It is not appropriate to discharge with follow-up at this stage as a combination of papilloedema and headaches can be suggestive of serious underlying pathology such as an intracranial tumour. Therefore, a CT head must be requested.

Topical corticosteroids are incorrect. Topical corticosteroids are used for the treatment of anterior uveitis, a condition that causes a painful red eye with photophobia and blurred vision. Patients also require an urgent review by ophthalmology. However, topical corticosteroids have no role in the treatment of papilloedema.

Urgent lumbar puncture is incorrect. A lumbar puncture is a reasonable investigation to request given the possibility of this patient having idiopathic intracranial hypertension (given the presence of an obese woman taking oral contraceptives) or an infectious cause of her headache. However, a lumbar puncture must not be completed until after a CT head is performed to rule out a space-occupying lesion that may be responsible for raised intracranial pressure.

Urgent referral to ophthalmology is incorrect. Although papilloedema is a worrying feature to find on examination, it does not require an urgent referral to ophthalmology. Rather, it requires neuroimaging to rule out intracranial pathology.

👍 👎 Discuss (2) Improve

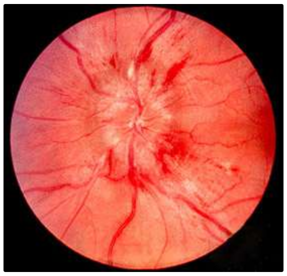
Next question >

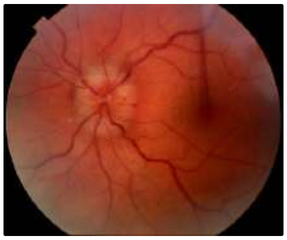
Papilloedema

Papilloedema describes optic disc swelling that is caused by increased intracranial pressure. It is almost always bilateral.

The following features may be observed during fundoscopy:

- venous engorgement: usually the first sign
- loss of venous pulsation: although many normal patients do not have normal pulsation
- blurring of the optic disc margin
- elevation of optic disc
- loss of the optic cup
- Paton's lines: concentric/radial retinal lines cascading from the optic disc





Causes of papilloedema

- space-occupying lesion: neoplastic, vascular
- malignant hypertension
- idiopathic intracranial hypertension
- hydrocephalus
- hypercapnia

Rare causes include

- hypoparathyroidism and hypocalcaemia
- vitamin A toxicity

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **20%**

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Question 140 of 189

A 24-year-old woman is admitted with a fever and headache. She reports this began acutely and worsened over the last 24 hours, now being associated with difficulty looking at lights.

She is febrile and photophobic with neck stiffness present. Her other observations are normal. GCS is 15 and no seizure activity is reported. There are no focal neurological deficits and no rash is observed.

She is commenced on IV ceftriaxone and dexamethasone for presumed bacterial meningitis. A lumbar puncture is performed which yields the following results.

Opening pressure	14cm H_2O (6-15)
WBC	127 cells/mm^3 (<5)
WBC Differential	85% lymphocytes, 15 % polymorphs
CSF Protein	0.7g/L (0.2-0.4)
CSF Glucose	4.9mmol/L (>2/3 serum)
Serum glucose	5.6mmol/L (3.3 - 5.8)
Culture	No organisms isolated
Bacterial PCR	Negative for *Neisseria meningitidis*, *Streptococcus pneumoniae* and *Haemophilus influenzae*
Viral PCR	Positive for Herpes simplex virus type 2

What is the most appropriate course of action?

- ○ Add IV aciclovir
- ○ Continue dexamethasone for 4 days and ceftriaxone for 10 days
- ○ Stop ceftriaxone and dexamethasone, start IV aciclovir for 14 days then repeat LP
- ○ Stop ceftriaxone and dexamethasone, start oral valaciclovir
- ○ Stop ceftriaxone and dexamethasone, supportive treatment only

Submit answer

Reference ranges ⌵

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A 24-year-old woman is admitted with a fever and headache. She reports this began acutely and worsened over the last 24 hours, now being associated with difficulty looking at lights.

She is febrile and photophobic with neck stiffness present. Her other observations are normal. GCS is 15 and no seizure activity is reported. There are no focal neurological deficits and no rash is observed.

She is commenced on IV ceftriaxone and dexamethasone for presumed bacterial meningitis. A lumbar puncture is performed which yields the following results.

Opening pressure	14cm H2O (6-15)
WBC	127 cells/mm³ (<5)
WBC Differential	85% lymphocytes, 15 % polymorphs
CSF Protein	0.7g/L (0.2-0.4)
CSF Glucose	4.9mmol/L (>2/3 serum)
Serum glucose	5.6mmol/L (3.3 - 5.8)
Culture	No organisms isolated
Bacterial PCR	Negative for *Neisseria meningitidis*, *Streptococcus pneumoniae* and *Haemophilus influenzae*
Viral PCR	Positive for Herpes simplex virus type 2

What is the most appropriate course of action?

Add IV aciclovir	27%
Continue dexamethasone for 4 days and ceftriaxone for 10 days	1%
Stop ceftriaxone and dexamethasone, start IV aciclovir for 14 days then repeat LP	46%
Stop ceftriaxone and dexamethasone, start oral valaciclovir	6%
✓ Stop ceftriaxone and dexamethasone, supportive treatment only	21%

Herpes simplex meningitis does not require treatment. This is in contrast to herpes simplex encephalitis which requires 14-21 days of intravenous aciclovir

Important for me Less important

The correct answer is **stop ceftriaxone and dexamethasone, supportive treatment only**. This patient's clinical presentation is consistent with a meningitic process which presents with fever, headache, neck stiffness, and photophobia. Viral meningitis, in stark contrast to viral encephalitis or bacterial meningitis, generally does not require treatment. Antibiotics should be discontinued and with supportive treatment, the condition generally resolves in 2-5 days. Enteroviruses are the most common cause, but HSV type 2 is recognised and associated with a condition now called recurrent benign lymphocytic meningitis.

Add IV aciclovir is incorrect. IV aciclovir is the treatment of choice for viral encephalitis caused by herpes simplex virus type 1. This condition involves inflammation of the brain parenchyma rather than the meninges. It results in an altered conscious level, agitation, seizures, and/or focal neurological deficits. None of these are described in this case. There would also be no rationale for continuing ceftriaxone and dexamethasone with negative cultures and a CSF examination consistent with a viral infection.

Continue dexamethasone for 4 days and ceftriaxone for 10 days is incorrect. This is the treatment recommendation for pneumococcal meningitis. This more commonly affects older adults, with bacterial meningitis in younger adults more commonly meningococcal. CSF in pneumococcal meningitis generally shows a predominance of polymorphs with raised protein and low glucose. Cultures may be negative where antibiotics have been given before lumbar puncture, but the negative bacterial PCR is reassuring.

Stop ceftriaxone and dexamethasone, start IV aciclovir for 14 days then repeat LP is incorrect. This would be the treatment of choice for HSV encephalitis, usually caused by HSV type 1. As discussed above, this presents with an altered conscious level, agitation, seizures, and/or focal neurological deficit. If the repeat LP at 14 days remains PCR positive, treatment is extended for a further 7 days.

Stop ceftriaxone and dexamethasone, start oral valaciclovir is incorrect. Valaciclovir is an alternative agent to oral aciclovir which can help with compliance, as it is administered twice daily rather than 5 times daily. However, its use is in oral or genital HSV infection rather than CNS infection. Also, as discussed above, there is no need for treatment in the case described.

👍 👎 Discuss (7) Improve

Next question ›

Cerebrospinal fluid: raised lymphocytes ★

Normal values of cerebrospinal fluid (CSF) are as follows:

- pressure = 60-150 mm (patient recumbent)
- protein = 0.2-0.4 g/l
- glucose = > 2/3 blood glucose
- cells: red cells = 0, white cells < 5/mm^3

The following conditions are associated with raised lymphocytes

- viral meningitis/encephalitis
- TB meningitis
- partially treated bacterial meningitis
- Lyme disease
- Behcet's, SLE
- lymphoma, leukaemia

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20%**

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Back to top

A 65-year-old woman presents to her general practitioner with a two-month history of a severe, 'all-over' throbbing headache, which varies in intensity over the course of the day. She has a past medical history of obesity and a traumatic fracture of the knee three months previously. She takes codeine regularly. She smokes 10 cigarettes per day.

The examination is unremarkable and the fundoscopy is normal. Her body mass index is 42 kg/m². A CT of her brain shows no abnormality.

Blood tests:

CRP	3mg/L	(<5)

Given the likely diagnosis, what is the appropriate management?

○ Acetazolamide

○ Prednisolone

○ Therapeutic lumbar puncture

○ Withdraw codeine gradually

○ Withdraw codeine now

Submit answer

Reference ranges

Score: **20.8%**

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A 65-year-old woman presents to her general practitioner with a two-month history of a severe, 'all-over' throbbing headache, which varies in intensity over the course of the day. She has a past medical history of obesity and a traumatic fracture of the knee three months previously. She takes codeine regularly. She smokes 10 cigarettes per day.

The examination is unremarkable and the fundoscopy is normal. Her body mass index is 42 kg/m^2. A CT of her brain shows no abnormality.

Blood tests:

CRP	3mg/L	(<5)

Given the likely diagnosis, what is the appropriate management?

Acetazolamide	17%
Prednisolone	0%
Therapeutic lumbar puncture	6%
Withdraw codeine gradually	67%
Withdraw codeine now	11%

Medication overuse headache

- simple analgesia + triptans: stop abruptly
- opioid analgesia: withdraw gradually

Important for me Less important

Withdraw codeine gradually is the correct answer. The patient presents with a new daily headache. As she has had a recent fracture, it is likely that codeine treatment was started at the same time. This new headache occurring after commencing opioid analgesia is likely reflective of a medication overuse headache. It varies over the course of the day as the dose of codeine wears off before the next dose is taken. Management of a codeine-associated medication overuse headache is to withdraw the medication gradually.

Acetazolamide is incorrect. She is overweight and this would put her at risk of developing idiopathic intracranial hypertension (IIH). However, fundoscopy has excluded papilloedema and so this makes this diagnosis unlikely. Furthermore, headaches associated with raised intracranial pressure are typically specifically worse in the morning rather than varying over the course of the day.

Therapeutic lumbar puncture is incorrect. This is an option for the treatment of IIH if confirmed. However, that diagnosis is unlikely in the setting of a normal fundoscopic examination.

Prednisolone is incorrect. Any new headache in someone > 50 should prompt consideration of giant cell arteritis (GCA). However, the headache of GCA is typically unilateral and associated with scalp tenderness. Additionally, it is extremely unlikely to have this condition and a normal CRP (now felt to be a more accurate marker of systemic inflammation than ESR in this condition).

Withdraw codeine now is incorrect. Withdrawal of opioids in medication overuse headache should be gradual rather than abrupt to avoid withdrawal and a worsening of the clinical situation.

👍 👎 Discuss (2) Improve

Next question

Medication overuse headache

Medication overuse headache is one of the most common causes of chronic daily headache. It may affect up to 1 in 50 people

Features
- present for 15 days or more per month
- developed or worsened whilst taking regular symptomatic medication
- patients using opioids and triptans are at most risk
- may be psychiatric co-morbidity

Management (from 2008 SIGN guidelines)
- simple analgesics and triptans should be withdrawn abruptly (may initially worsen headaches)
- opioid analgesics should be gradually withdrawn

Withdrawal symptoms such as vomiting, hypotension, tachycardia, restlessness, sleep disturbances and anxiety may occur when medication is stopped

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20%**

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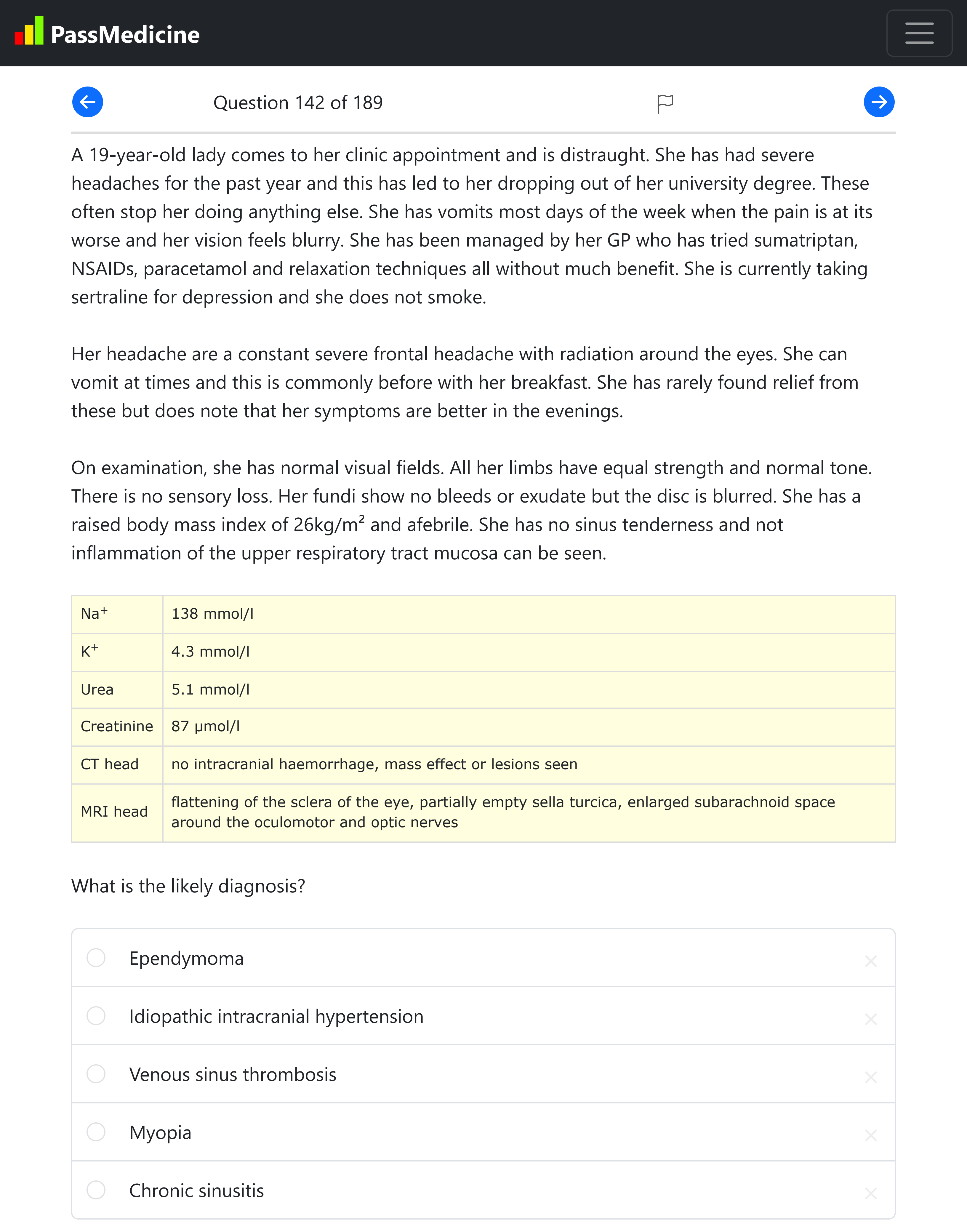

A 19-year-old lady comes to her clinic appointment and is distraught. She has had severe headaches for the past year and this has led to her dropping out of her university degree. These often stop her doing anything else. She has vomits most days of the week when the pain is at its worse and her vision feels blurry. She has been managed by her GP who has tried sumatriptan, NSAIDs, paracetamol and relaxation techniques all without much benefit. She is currently taking sertraline for depression and she does not smoke.

Her headache are a constant severe frontal headache with radiation around the eyes. She can vomit at times and this is commonly before with her breakfast. She has rarely found relief from these but does note that her symptoms are better in the evenings.

On examination, she has normal visual fields. All her limbs have equal strength and normal tone. There is no sensory loss. Her fundi show no bleeds or exudate but the disc is blurred. She has a raised body mass index of 26kg/m^2 and afebrile. She has no sinus tenderness and not inflammation of the upper respiratory tract mucosa can be seen.

Na^+	138 mmol/l
K^+	4.3 mmol/l
Urea	5.1 mmol/l
Creatinine	87 µmol/l
CT head	no intracranial haemorrhage, mass effect or lesions seen
MRI head	flattening of the sclera of the eye, partially empty sella turcica, enlarged subarachnoid space around the oculomotor and optic nerves

What is the likely diagnosis?

○ Ependymoma

○ Idiopathic intracranial hypertension

○ Venous sinus thrombosis

○ Myopia

○ Chronic sinusitis

Submit answer

Reference ranges

Score: **20.8%**

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A 19-year-old lady comes to her clinic appointment and is distraught. She has had severe headaches for the past year and this has led to her dropping out of her university degree. These often stop her doing anything else. She has vomits most days of the week when the pain is at its worse and her vision feels blurry. She has been managed by her GP who has tried sumatriptan, NSAIDs, paracetamol and relaxation techniques all without much benefit. She is currently taking sertraline for depression and she does not smoke.

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What is the likely diagnosis?

Ependymoma	9%
Idiopathic intracranial hypertension	75%
Venous sinus thrombosis	14%
Myopia	1%
Chronic sinusitis	1%

> Postural headache but normal imaging -> idiopathic intracranial hypertension
>
> Important for me Less important

This is a chronic postural headache suggested by its improvement as the day progresses. Myopia and chronic sinusitis would not cause a postural headache. Ependymoma and a venous sinus thrombosis would both be seen on imaging. Therefore, by exclusion, this headache is idiopathic intracranial hypertension.

Discuss (5) Improve

Next question ›

Idiopathic intracranial hypertension

Idiopathic intracranial hypertension (also known as pseudotumour cerebri and formerly benign intracranial hypertension) is a condition classically seen in young, overweight females.

Risk factors
- obesity
- female sex
- pregnancy
- drugs
    - combined oral contraceptive pill
    - steroids
    - tetracyclines
    - retinoids (isotretinoin, tretinoin) / vitamin A
    - lithium

Features
- headache
- blurred vision
- papilloedema (usually present)
- enlarged blind spot
- sixth nerve palsy may be present

Management
- weight loss
    - whilst diet and exercise are important, medications such as semaglitide and topiramate may be considered by specialists. Topiramate is particularly beneficial as it also inhibits

carbonic anhydrase

- carbonic anhydrase inhibitors e.g. acetazolamide
- topiramate is also used, and has the added benefit of causing weight loss in most patients
- repeated lumbar puncture may be used as a temporary measure but is not suitable for longer-term management
- surgery: optic nerve sheath decompression and fenestration may be needed to prevent damage to the optic nerve. A lumboperitoneal or ventriculoperitoneal shunt may also be performed to reduce intracranial pressure

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 12 👎 13

Idiopathic intracranial hypertension review

Suggest link Report broken link

Score: **20%**

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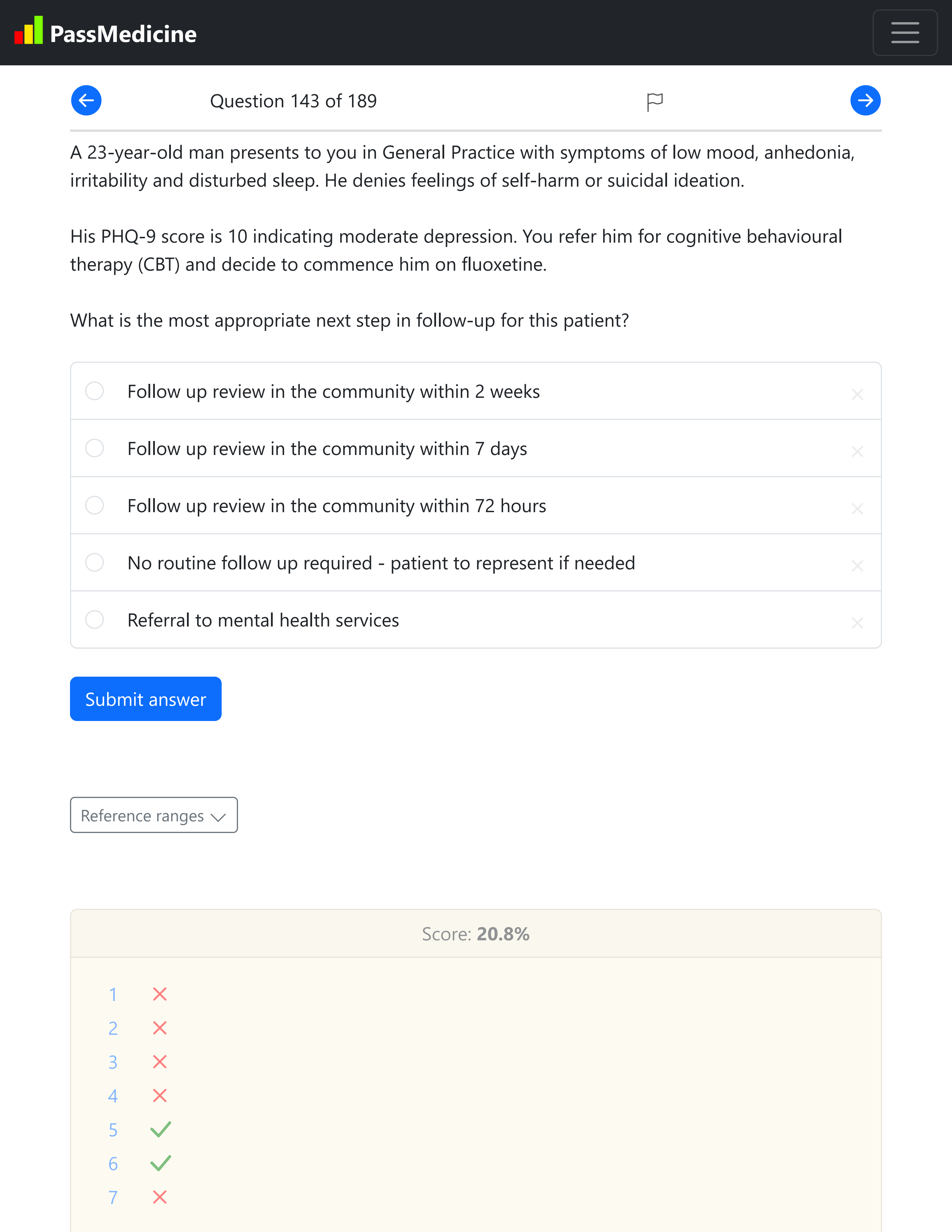

Question 143 of 189

A 23-year-old man presents to you in General Practice with symptoms of low mood, anhedonia, irritability and disturbed sleep. He denies feelings of self-harm or suicidal ideation.

His PHQ-9 score is 10 indicating moderate depression. You refer him for cognitive behavioural therapy (CBT) and decide to commence him on fluoxetine.

What is the most appropriate next step in follow-up for this patient?

○ Follow up review in the community within 2 weeks

○ Follow up review in the community within 7 days

○ Follow up review in the community within 72 hours

○ No routine follow up required - patient to represent if needed

○ Referral to mental health services

Score: **20.8%**

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A 23-year-old man presents to you in General Practice with symptoms of low mood, anhedonia, irritability and disturbed sleep. He denies feelings of self-harm or suicidal ideation.

His PHQ-9 score is 10 indicating moderate depression. You refer him for cognitive behavioural therapy (CBT) and decide to commence him on fluoxetine.

What is the most appropriate next step in follow-up for this patient?

Follow up review in the community within 2 weeks	37%
Follow up review in the community within 7 days	55%
Follow up review in the community within 72 hours	4%
No routine follow up required - patient to represent if needed	1%
Referral to mental health services	3%

Patients ≤ 25 years who have been started on an SSRI should be reviewed after 1 week

Important for me Less important

This patient has moderate depression as indicated by their PHQ-9 score. Patients under the age of 25 who are started on an SSRI should be followed up within 1 week due to the increased risk of suicide within this age group and therefore **option 2 - follow-up review in the community within 7 days** is the correct answer. Patients over the age of 25 who are started on antidepressant therapy should be followed up in the community within 2 weeks.

Follow-up in the community within 2 weeks and **follow up in the community within 72 hours** are incorrect as they are not the recommended time scales for follow-up.

No routine follow-up required - patient to represent if needed is incorrect as all patients who are initiated on antidepressant therapy should have planned follow-up due to the risk of worsening symptoms when initiating treatment.

Referral to mental health services is incorrect as there is no indication for referral to mental health services at this stage.

👍 👎 Discuss Improve

Next question ›

Selective serotonin reuptake inhibitors ★

Selective serotonin reuptake inhibitors (SSRIs) are considered first-line treatment for the majority of patients with depression.

- citalopram (although see below re: QT interval) and fluoxetine are currently the preferred SSRIs
- sertraline is useful post myocardial infarction as there is more evidence for its safe use in this situation than other antidepressants
- SSRIs should be used with caution in children and adolescents. Fluoxetine is the drug of choice when an antidepressant is indicated

Adverse effects

- gastrointestinal symptoms are the most common side-effect
- there is an increased risk of gastrointestinal bleeding in patients taking SSRIs. A proton pump inhibitor should be prescribed if a patient is also taking a NSAID
- patients should be counselled to be vigilant for increased anxiety and agitation after starting a SSRI
- fluoxetine and paroxetine have a higher propensity for drug interactions

Citalopram and the QT interval

- the Medicines and Healthcare products Regulatory Agency (MHRA) released a warning on the use of citalopram in 2011
- it advised that citalopram and escitalopram are associated with dose-dependent QT interval prolongation and should not be used in those with: congenital long QT syndrome; known pre-existing QT interval prolongation; or in combination with other medicines that prolong the QT interval
- the maximum daily dose is now 40 mg for adults; 20 mg for patients older than 65 years; and 20 mg for those with hepatic impairment

Interactions

- NSAIDs: NICE guidelines advise 'do not normally offer SSRIs', but if given co-prescribe a proton pump inhibitor

- warfarin / heparin: NICE guidelines recommend avoiding SSRIs and considering mirtazapine
- aspirin
- triptans - increased risk of serotonin syndrome
- monoamine oxidase inhibitors (MAOIs) - increased risk of serotonin syndrome

Following the initiation of antidepressant therapy patients should normally be reviewed by a doctor after 2 weeks. For patients under the age of 25 years or at increased risk of suicide should be reviewed after 1 week. If a patient makes a good response to antidepressant therapy they should continue on treatment for at least 6 months after remission as this reduces the risk of relapse.

When stopping a SSRI the dose should be gradually reduced over a 4 week period (this is not necessary with fluoxetine). Paroxetine has a higher incidence of discontinuation symptoms.

Discontinuation symptoms

- increased mood change
- restlessness
- difficulty sleeping
- unsteadiness
- sweating
- gastrointestinal symptoms: pain, cramping, diarrhoea, vomiting
- paraesthesia

SSRIs and pregnancy
- BNF says to weigh up benefits and risk when deciding whether to use in pregnancy.
- Use during the first trimester gives a small increased risk of congenital heart defects
- Use during the third trimester can result in persistent pulmonary hypertension of the newborn
- Paroxetine has an increased risk of congenital malformations, particularly in the first trimester

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 29 👎 43

2022 Depression in adults: treatment and management

Suggest link Report broken link

Media

Antidepressants

Speed Pharmacology - YouTube 👍 6 👎 0

ANTIDEPRESSANTS
PHARMACOLOGY

Antidepressants Pharmacology

Soton Brain Hub - YouTube 👍 0 👎 0

Report broken media

Score: **20%**

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Question 144 of 189

A 60 yr old woman went swimming with her family, and after finishing her swim and having a shower, her daughter noted that she became confused suddenly, repetitively asking if they had gone for a swim today. She could not remember events that occurred in the past 24 hours, and when told the answers to her questions, would ask the same question 5 minutes later. There was no change in her personality, no change in her speech, nor any muscle weakness. She is able to recall her address, the names of her daughters and husband, and her date of birth.
Her daughter said her mother did not suffer any trauma during the swim, and did not lose consciousness anytime throughout the day. The patient's past medical history includes hypertension and depression, and she takes ramipril 2.5mg once daily, and citalopram 20mg once daily.

On examination the patient was alert, but constantly asked where she was and why was she in hospital. She was afebrile, heart rate 80 bpm, blood pressure 138/68 mmHg, respiratory rate of 18 breaths per minute, and oxygen saturation of 99% on air. Neurological examination was unremarkable, but her abbreviated mental test score was 6/10.

Her investigation results were as follow:

C Reactive protein	4 mg/l
Haemoglobin	14.8 g/dl
White cell count	5.6 x 10^9/L
Na+	142 mmol/l
K+	4.3 mmol/l
Urea	4.2 mmol/l
Creatinine	68 µmol/l
Corrected calcium	2.32 mmol/l
Plasma glucose	5.8 mmol/l

Computer Tomography (CT) head scan No acute intracranial pathology.

Over the next 12 hours, her memory improves and she is discharged from the observation ward.

What is the best advice for the patient with regards to driving in the future?

○ She can drive 4 weeks after the event if the cause has been identified and treated

○ DVLA need not be notified, no driving restrictions ×

○ She must be symptom free for 1 year before being eligible to drive ×

○ DVLA must be informed, and her licence will be revoked for 6 months ×

○ She must cease driving until there is satisfactory control of her symptoms ×

Submit answer

Reference ranges ∨

Score: **20.8%**

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Computer Tomography (CT) head scan No acute intracranial pathology.

Over the next 12 hours, her memory improves and she is discharged from the observation ward.

What is the best advice for the patient with regards to driving in the future?

She can drive 4 weeks after the event if the cause has been identified and treated 23%

DVLA need not be notified, no driving restrictions 51%

She must be symptom free for 1 year before being eligible to drive 5%

DVLA must be informed, and her licence will be revoked for 6 months 7%

She must cease driving until there is satisfactory control of her symptoms 14%

This lady has suffered an episode of transient global amnesia, a neurological condition characterised by acute short term memory loss, lasting no more than 24 hours. Patients often developed perseveration, with repetitive questioning as they are unable to keep short term memory, but are able to recall long term memories. The aetiology of this condition is unknown.

DVLA guidelines for transient global amnesia are that as long as epilepsy, any sequelae from head injury and other causes of altered awareness have been excluded, there are no restrictions on driving and the DVLA need not be informed.

👍 👎 Discuss (3) Improve

Next question >

DVLA: neurological disorders ★

The guidelines below relate to car/motorcycle use unless specifically stated. For obvious reasons, the rules relating to drivers of heavy goods vehicles tend to be much stricter

Epilepsy/seizures - all patient must not drive and must inform the DVLA
- first unprovoked/isolated seizure: 6 months off if there are no relevant structural abnormalities on brain imaging and no definite epileptiform activity on EEG. If these conditions are not met then this is increased to 12 months
- for patients with established epilepsy or those with multiple unprovoked seizures:
  - may qualify for a driving licence if they have been free from any seizure for 12 months
  - if there have been no seizures for 5 years (with medication if necessary) a 'til 70 licence is usually restored
- withdrawawl of epilepsy medication: should not drive whilst anti-epilepsy medication is being withdrawn and for 6 months after the last dose

Syncope
- simple faint: no restriction

- single episode, explained and treated: 4 weeks off
- single episode, unexplained: 6 months off
- two or more episodes: 12 months off

Other conditions

- stroke or TIA: 1 month off driving, may not need to inform DVLA if no residual neurological deficit
- multiple TIAs over short period of times: 3 months off driving and inform DVLA
- craniotomy e.g. For meningioma: 1 year off driving*
- pituitary tumour: craniotomy: 6 months; trans-sphenoidal surgery 'can drive when there is no debarring residual impairment likely to affect safe driving'
- narcolepsy/cataplexy: cease driving on diagnosis, can restart once 'satisfactory control of symptoms'
- chronic neurological disorders e.g. multiple sclerosis, motor neuron disease: DVLA should be informed, complete PK1 form (application for driving licence holders state of health)

*if the tumour is a benign meningioma and there is no seizure history, licence can be reconsidered 6 months after surgery if remains seizure free

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

DVLA

👍 31 👎 68

Fitness to drive guidelines

Suggest link

Report broken link

Score: **20%**

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A 43-year-old right-handed female legal secretary has presented into your general medical clinic in an extremely distressed state. Over the past 2 weeks, she has been 'unable to write'. When questioned further, she reports that she 'wants to write but my hand just stops as soon as I pick up the pen.' She has no past medical history, lives with her husband and is a non-smoker and non-drinker. On examination, her neurological examination is unremarkable. You ask her to write. Her hand and fingers suddenly flex, resulting in illegible handwriting. What is the most likely diagnosis?

- ○ Psychogenic functional neurology ×
- ○ Focal dystonia ×
- ○ Dominant parietal lobe space occupying lesion ×
- ○ Right median nerve palsy ×
- ○ Brachial plexopathy ×

Submit answer

Reference ranges

Score: **20.8%**

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A 43-year-old right-handed female legal secretary has presented into your general medical clinic in an extremely distressed state. Over the past 2 weeks, she has been 'unable to write'. When questioned further, she reports that she 'wants to write but my hand just stops as soon as I pick up the pen.' She has no past medical history, lives with her husband and is a non-smoker and non-drinker. On examination, her neurological examination is unremarkable. You ask her to write. Her hand and fingers suddenly flex, resulting in illegible handwriting. What is the most likely diagnosis?

Psychogenic functional neurology	50%
Focal dystonia ✓	33%
Dominant parietal lobe space occupying lesion	10%
Right median nerve palsy	3%
Brachial plexopathy ✗	3%

The patient describes a history suggestive of writers cramp, a focal dystonia characterised by flexion, extension or rotation of the muscles of the hand. The underlying pathophysiology is unclear but is thought to relate to a change in the plasticity of cortical networks.

👍 👎 Discuss (5) Improve

Next question >

Focal dystonia ★

Focal dystonias are distinguished from segmental or generalised dystonias, which involve a greater number of muscle groups. Focal dystonias are often relieved by a geste antagoniste, in which palpation of another unaffected part of the body leads to relief of symptoms, thought to be a result of alternative sensory input to cortical networks with altered plasticity.

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

The Dystonia Society 👍 1 👎 0

Dystonia: A Guide to Best Practice

Suggest link Report broken link

Score: **20%**

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Question 146 of 189

A 68-year-old female presents via blue light ambulance to the emergency department with a first seizure, witnessed by her husband. He describes a sudden onset limb jerking lasting for around 6 minutes, associated with urinary incontinence and tongue biting, with a period of confusion and drowsiness immediately after. He is not aware of her having had any previous seizures, she has no other past medical history and does not take drugs on a medical history. He, however, does describe her to be 'not quite herself' over the past four weeks when she appears to have been extremely agitated and occasionally extremely paranoid. He had thought it to be linked to her not feeling very well, during a period when she had complained of a 'flu-like' headache, generalised muscle ache and a non-productive cough.

You examine her, finding significant gait and limb ataxia with no truncal ataxia. Her blood tests are unremarkable except for positive anti-NMDA antibodies. Her MRI scan demonstrates swelling in bilateral limbic cortices and other no other intracranial abnormalities. She has declined a lumbar puncture and is thought to retain capacity.

Which investigation would most likely provide the underlying diagnosis?

- ○ CT abdomen/pelvis with contrast ×
- ○ Lumbar puncture ×
- ○ Nerve conduction studies and EMG ×
- ○ MRI spine ×
- ○ Muscle biopsy ×

Submit answer

Reference ranges

Score: **20.8%**

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146 -

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A 68-year-old female presents via blue light ambulance to the emergency department with a first seizure, witnessed by her husband. He describes a sudden onset limb jerking lasting for around 6 minutes, associated with urinary incontinence and tongue biting, with a period of confusion and drowsiness immediately after. He is not aware of her having had any previous seizures, she has no other past medical history and does not take drugs on a medical history. He, however, does describe her to be 'not quite herself' over the past four weeks when she appears to have been extremely agitated and occasionally extremely paranoid. He had thought it to be linked to her not feeling very well, during a period when she had complained of a 'flu-like' headache, generalised muscle ache and a non-productive cough.

You examine her, finding significant gait and limb ataxia with no truncal ataxia. Her blood tests are unremarkable except for positive anti-NMDA antibodies. Her MRI scan demonstrates swelling in bilateral limbic cortices and other no other intracranial abnormalities. She has declined a lumbar puncture and is thought to retain capacity.

Which investigation would most likely provide the underlying diagnosis?

CT abdomen/pelvis with contrast	57%
Lumbar puncture	15%
Nerve conduction studies and EMG	13%
MRI spine	10%
Muscle biopsy	6%

This middle aged woman has presented with seizures on a background of personality change, agitation, paranoia and cerebellar ataxia. The diagnostic test is her positive anti-NMDA antibody, with MRI confirm limbic encephalitis as a result of a paraneoplastic syndrome. The underlying diagnosis is classically an ovarian teratoma, presenting antigens to the body's immune system similar to the brain limbic areas, driving an immune-related encephalitis. Treatment requires immunosuppression with intravenous steroids, immunoglobulin's or plasma exchange, in addition to removal of the underlying tumour by surgery.

Discuss (4) | Improve

Next question >

Anti-NMDA receptor encephalitis ★

Anti-NMDA receptor encephalitis is a paraneoplastic syndrome, presenting as prominent psychiatric features including agitation, hallucinations, delusions and disordered thinking; seizures, insomnia, dyskinesias and autonomic instability. Ovarian teratomas are detected in up to half of all female adult patients, particularly prevalent in Afro-Caribbean patients. MRI head can be normal but abnormalities can be visualised on FLAIR sequences in the deep subcortical limbic structures. CSF may demonstrate pleocytosis but can be normal initially. Anti-MuSK is an autoantibody specific to muscle kinase in myasthenia gravis with no evidence of a thymoma and without antibodies to acetylcholine receptors. Anti-GM1 is an autoantibody specific to acute inflammatory demyelinating polyneuropathy (AIDP) variant of Guillain-Barre syndrome.

Treatment of anti-NMDA encephalitis is based of immunosuppression with intravenous steroids, immunoglobulins, rituximab, cyclophosphamide or plasma exchange, alone or in combination. Resection of teratoma is also therapeutic.

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20%**

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Question 147 of 189

A 44-year-old male presents to your headache clinic with a 6-month history of left-sided daily headaches located in the frontal and retroorbital area. He denies any pain-free periods. The headaches are of moderate 5/10 severity with unpredictable exacerbations of severe pain going up to 9/10 severity. He describes to you what sounds to you like left-sided conjunctival injection and lacrimation often occurring alongside the headaches. Which of the following is most likely to aid you in making a diagnosis?

- ○ a trial of high flow 100% oxygen
- ○ Lumbar puncture
- ○ a trial of indomethacin
- ○ CT brain
- ○ MRI brain

Submit answer

Reference ranges

Score: **20.8%**

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A 44-year-old male presents to your headache clinic with a 6-month history of left-sided daily headaches located in the frontal and retroorbital area. He denies any pain-free periods. The headaches are of moderate 5/10 severity with unpredictable exacerbations of severe pain going up to 9/10 severity. He describes to you what sounds to you like left-sided conjunctival injection and lacrimation often occurring alongside the headaches. Which of the following is most likely to aid you in making a diagnosis?

a trial of high flow 100% oxygen	34%
Lumbar puncture	0%
a trial of indomethacin	58%
CT brain	2%
MRI brain	6%

A headache with absolutely no pain-free periods otherwise described like this (i.e. like a trigeminal autonomic cephalalgia (TAC)) is hemicrania continua.

The trigeminal autonomic cephalalgias should be side-locked (i.e. one side only) and typically but not exclusively in the retroorbital/temporal region. They should include some autonomic parasympathetic feature such as eye-watering, red eye, running nose, a sensation of fullness in the ear, or miosis. These should be ipsilateral to the pain. They are a spectrum and most conveniently differentiated by how long the headache lasts for and how frequent the attacks happen per day. Some features are quite noticeable in TAC, e.g in a cluster headache (aka 'suicide headaches') people are usually very animated and pace about/hold their head in pain/want to smash their head against the wall (contrast this to migraine: which is not a TAC and usually involves sitting still in a dark room and not making any noise).

It is worth looking up the International Classification of Headache Disorders diagnostic criteria for TACs. Here are some important features:

- Cluster headaches: attacks have a frequency between one every other day and 8 per day and last 15-180 minutes.
- Paroxysmal hemicrania: attacks have a frequency above five per day and last 2-30 min. The condition responds absolutely to indomethacin

- Hemicrania continua: a constant form of paroxysmal hemicrania (no headache-free periods). Also responds absolutely to indomethacin.

- Short-lasting unilateral neuralgiform headache attacks: could get 20 attacks in a day, each last around 1600 seconds.

Notice the differences between how long each lasts and how many attacks there can be per day. The rest of the features are quite similar to each other between the headaches, so learn the general features of a TAC then memorise how long each should last and its frequency to help you differentiate them.

Returning to the question: hemicrania continua and paroxysmal hemicrania clearly are the two that completely resolve with indomethacin. The history, therefore, helps differentiate which is which. Practically speaking, you bring these patients in, on two separate days, for a trial injection of indomethacin or placebo (water) where patient (and nurse if possible) is blinded to which is which and we see which one of the two days was associated with resolution of headaches at a later date in clinic (give the patient a headache diary to take home with). Note the 100% oxygen trial would be used for cluster headaches, which tends to respond to this.

Ref: https://www.ichd-3.org/3-trigeminal-autonomic-cephalalgias/

👍 👎 Discuss (11) Improve

Next question ›

Trigeminal autonomic cephalalgias ★

Trigeminal autonomic cephalalgias (TACs)
- cluster headache
- paroxysmal hemicrania
- hemicrania continua
- short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT syndrome)
- short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms (SUNA)

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Media

NEUROLOGY
TRIGEMINAL AUTONOMIC
CEPHALALGIAS

Severe headaches - Understanding Trigeminal Autonomic Cephalgias - types, pathophysiology, treatment

Armando Hasudungan - YouTube 👍 10 👎 0

Report broken media

Score: **20%**

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151	-

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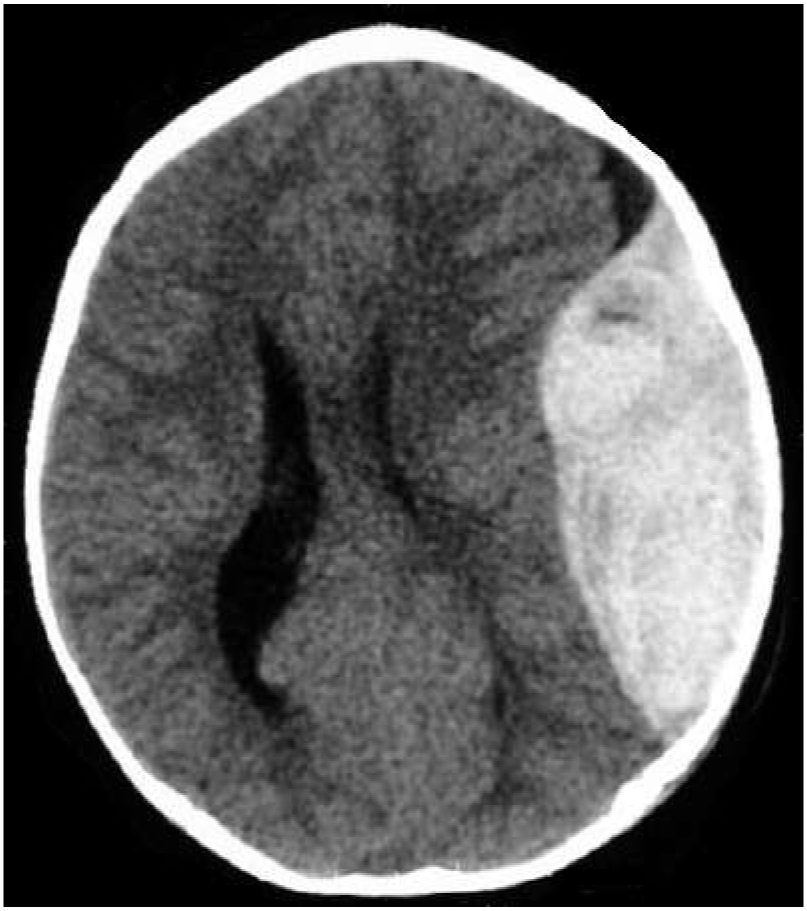
You are asked to review a 32-year-old man who was brought in by ambulance after an assault. A witness states that he lost consciousness before waking up fully. He has subsequently become more drowsy. His current GCS is 9/15 (E2 V3 M4).

Neurological examination is difficult due to GCS. Of note, pupils are symmetrical and there is an extensor right plantar.

A cross-section of his CT brain is shown below.



© Image used on license from Radiopaedia

Which of the following parts of the history is associated with a poorer prognosis?

○ Extensor plantar response

○ Low GCS at presentation

○ Lucid period

○ Symmetrical pupils ×

○ Young age ×

Submit answer

Reference ranges ⌵

Score: **20.8%**

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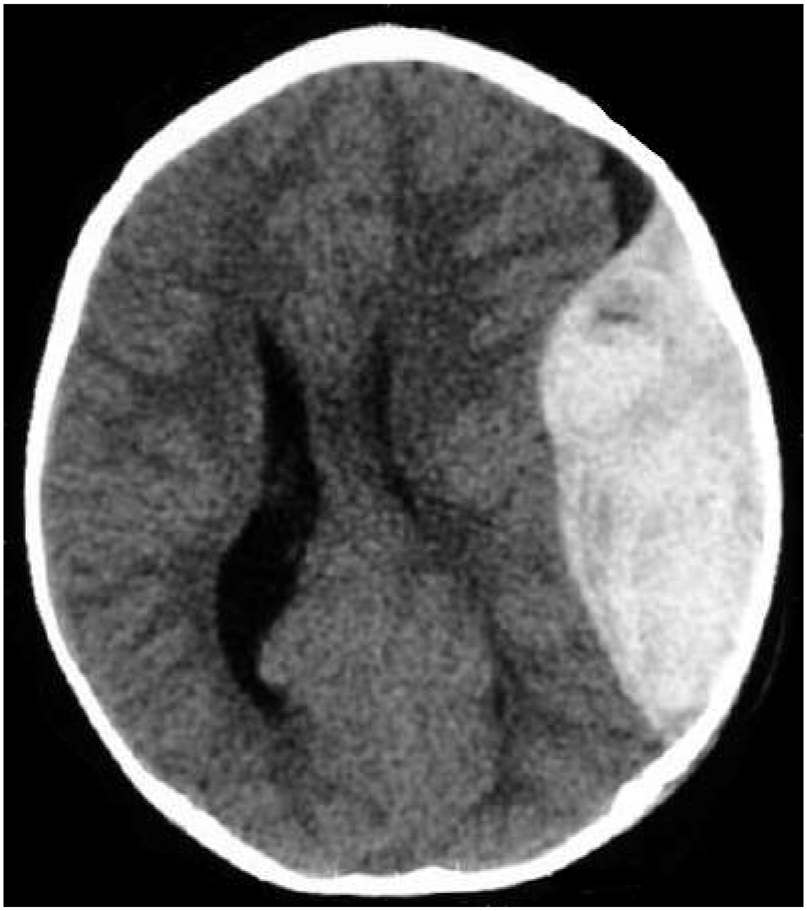


© Image used on license from Radiopaedia

Which of the following parts of the history is associated with a poorer prognosis?

Extensor plantar response	17%
Low GCS at presentation	55%
Lucid period	22%

Symmetrical pupils	2%
Young age	2%

The above history describes a reduced level of consciousness following an assault. The CT brain shows a biconvex hyperdensity in the left temporal region which is in keeping with an extradural haematoma. There is evidence of mass effect with midline shift and effacement of the left lateral ventricle which, combined with a reducing conscious level, is concerning for raised intracranial pressure (ICP).

A low GCS at presentation is correct. Low GCS is likely associated with raised ICP and/or traumatic brain injury and therefore carries a poorer prognosis.

Extensor plantar response eludes to an upper motor neuron lesion, in this case, an epidural haematoma, and would be an expected finding but does not denote a poorer prognosis.

Poorer prognosis is associated with patients that do not experience a lucid period.

Asymmetrical pupils are associated with a poor prognosis and are a sign of raised ICP.

Young age is a protective factor compared to old age which carries a worse prognosis.

👍 👎 Discuss (1) Improve

Next question >

Head injury: types of traumatic brain injury ★

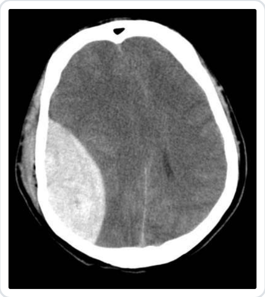
Basics

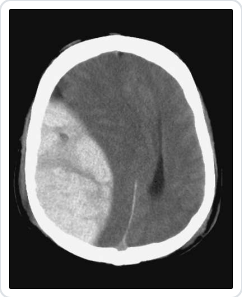
- primary brain injury may be focal (contusion/haematoma) or diffuse (diffuse axonal injury)
- diffuse axonal injury occurs as a result of mechanical shearing following deceleration, causing disruption and tearing of axons
- intra-cranial haematomas can be extradural, subdural or intracerebral, while contusions may occur adjacent to (coup) or contralateral (contre-coup) to the side of impact
- secondary brain injury occurs when cerebral oedema, ischaemia, infection, tonsillar or tentorial herniation exacerbates the original injury. The normal cerebral auto regulatory processes are disrupted following trauma rendering the brain more susceptible to blood flow changes and hypoxia
- the Cushings reflex (hypertension and bradycardia) often occurs late and is usually a pre terminal event

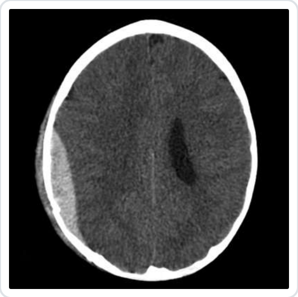
Type of injury	Notes
Extradural (epidural) haematoma	Bleeding into the space between the dura mater and the skull. Often results from acceleration-deceleration trauma or a blow to the side of the head. The majority of epidural haematomas occur in the temporal region where skull fractures cause a rupture of the middle meningeal artery. Features • features of raised intracranial pressure • some patients may exhibit a lucid interval
Subdural haematoma	Bleeding into the outermost meningeal layer. Most commonly occur around the frontal and parietal lobes. Risk factors include old age, alcoholism and anticoagulation. Slower onset of symptoms than a epidural haematoma. There may be fluctuating confusion/consciousness
Subarachnoid haemorrhage	Classically causes a sudden occipital headache. Usually occurs spontaneously in the context of a ruptured cerebral aneurysm but may be seen in association with other injuries when a patient has sustained a traumatic brain injury
Intracerebral haematoma	An intracerebral (or intraparenchymal) haemorrhage is a collection of blood within the substance of the brain. Causes / risk factors include: hypertension, vascular lesion (e.g. aneurysm or arteriovenous malformation), cerebral amyloid angiopathy, trauma, brain tumour or infarct (particularly in stroke patients undergoing thrombolysis). Patients will present similarly to an ischaemic stroke (which is why it is crucial to obtain a CT in head in all stroke patients prior to thrombolysis) or with a decrease in consciousness. CT imaging will show a hyperdensity (bright lesion) within the substance of the brain. Treatment is often conservative under the care of stroke physicians, but large clots in patients with impaired consciousness may warrant surgical evacuation.

Image gallery

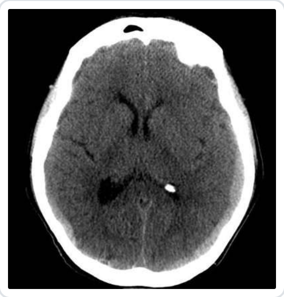
Extradural (epidural) haematoma:

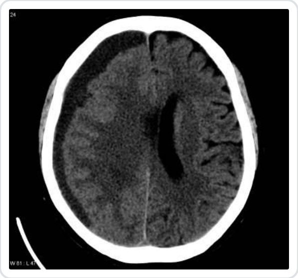


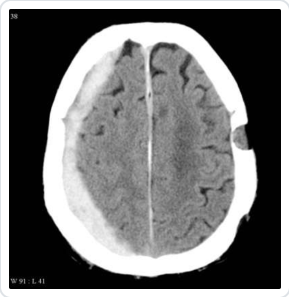




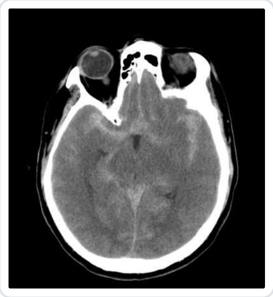
Subdural haematoma:

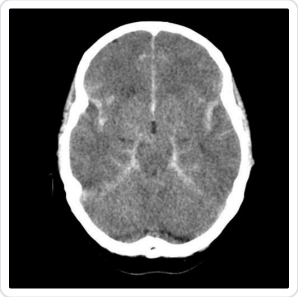






Subarachnoid haemorrhage:





Next question >

Textbooks

High-yield textbook

Extended textbook

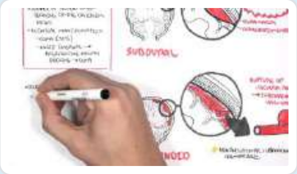
Links

NICE 👍 11 👎 2

2014 Head injury guidelines

Suggest link Report broken link

Media



Intracranial Haemorrhage Types

Armando Hasudungan - YouTube 👍 6 👎 0

Intracranial
Bleeds
subdural
&
extradural

Intracranial bleeds - Subdural and Extradural haematoma

Soton Brain Hub - YouTube 👍 4 👎 0

TRAUMATIC
BRAIN INJURY
~ PATHOLOGY REVIEW ~
Osmosis.org

Traumatic brain injury: pathology review

Osmosis - YouTube 👍 4 👎 1

Report broken media

Score: **20%**

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Question 149 of 189

A 52-year-old woman presents to your clinic with a history of vertigo.

She describes short-lived episodes of true vertigo which often occur whilst lying down in bed, turning over or sitting up.

The episodes last between ten to twenty seconds and then quickly dissipate.

You perform the Dix-Hallpike test in the clinic which gives a positive result and you make a diagnosis of BPPV.

Whilst consenting the patient to perform an Epley manoeuvre to treat her BPPV the patient asks how successful this is at treating her condition.

In what percentage of cases is this successful?

- ○ 25%
- ○ 50%
- ○ 60%
- ○ 80%
- ○ 99%

Submit answer

Reference ranges

Score: **20.8%**

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149 -

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Question 149 of 189

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Whilst consenting the patient to perform an Epley manoeuvre to treat her BPPV the patient asks how successful this is at treating her condition.

In what percentage of cases is this successful?

25%	2%
50%	5%
60%	13%
80%	75%
99%	6%

The Epley manoeuvre is successful in treating around 80% of cases of BPPV

Important for me   Less important

The Epley manoeuvre is successful in treating around **80% of cases** of BPPV.

Repeated treatments with the Epley manoeuvre can be helpful if it fails at the first attempt.

Patients can also be shown how to do 'home Epley' manoeuvres to complete by themselves.

Discuss (2)   Improve

Next question ›

Benign paroxysmal positional vertigo

Benign paroxysmal positional vertigo (BPPV) is one of the most common causes of vertigo encountered. It is characterised by the sudden onset of dizziness and vertigo triggered by changes in head position. The average age of onset is 55 years and it is less common in younger patients.

Features
- vertigo triggered by change in head position (e.g. rolling over in bed or gazing upwards)
- may be associated with nausea
- each episode typically lasts 10-20 seconds
- positive Dix-Hallpike manoeuvre
    - rapidly lower the patient to the supine position with an extended neck
    - a positive test recreates the symptoms of benign paroxysmal positional vertigo
    - rotatory nystagmus

BPPV has a good prognosis and usually resolves spontaneously after a few weeks to months. Symptomatic relief may be gained by:
- **Epley manoeuvre** (successful in around 80% of cases)
- teaching the patient exercises they can do themselves at home, termed vestibular rehabilitation, for example Brandt-Daroff exercises

Medication is often prescribed (e.g. Betahistine) but it tends to be of limited value.

Around half of people with BPPV will have a recurrence of symptoms 3-5 years after their diagnosis

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 33 👎 11

Benign paroxysmal positional vertigo guidelines

Suggest link

Report broken link

Media

Patient sitting upright with head turned
to side 45 degrees

Hallpike Test and Epley Maneuver

Individual - YouTube 👍 14 👎 0

Report broken media

Score: **20%**

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A 76-year-old man was brought into the emergency department by his wife. She was concerned because she had noticed that he had been slurring his words at breakfast that morning at 07:00. She had also noticed he was suffering from left-sided weakness. These symptoms persisted and he was struggling to feed himself when they ate lunch at 13:30.

He had a past medical history of hypertension for which he takes amlodipine. He did not smoke and was independent in his activities of daily living.

On examination, he was dysarthric and had hemisensory loss of his left side. Power was 2/5 in his left leg and left arm. He had normal sensation and power on the right side of his body. His pulse was irregular.

Observations were as follows:

- oxygen saturations 98% on room air
- respiratory rate 14/min
- heart rate 67bpm
- blood pressure 135/78mmHg
- temperature 37.1°C.

A CT angiogram was performed with perfusion imaging which revealed the presence of a thrombus in the right middle cerebral artery. There was a small area of infarct present and a large area of surrounding ischaemic penumbra.

What is the most appropriate management of this patient?

○ Load with aspirin and admit to the stroke ward

○ Thrombectomy

○ IV thrombolysis with alteplase

○ IV heparin infusion

○ Decompressive hemicraniectomy

Submit answer

Reference ranges ⌄

Score: **20.8%**

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A 76-year-old man was brought into the emergency department by his wife. She was concerned because she had noticed that he had been slurring his words at breakfast that morning at 07:00. She had also noticed he was suffering from left-sided weakness. These symptoms persisted and he was struggling to feed himself when they ate lunch at 13:30.

He had a past medical history of hypertension for which he takes amlodipine. He did not smoke and was independent in his activities of daily living.

On examination, he was dysarthric and had hemisensory loss of his left side. Power was 2/5 in his left leg and left arm. He had normal sensation and power on the right side of his body. His pulse was irregular.

Observations were as follows:

- oxygen saturations 98% on room air
- respiratory rate 14/min
- heart rate 67bpm
- blood pressure 135/78mmHg
- temperature 37.1°C.

A CT angiogram was performed with perfusion imaging which revealed the presence of a thrombus in the right middle cerebral artery. There was a small area of infarct present and a large area of surrounding ischaemic penumbra.

What is the most appropriate management of this patient?

Load with aspirin and admit to the stroke ward	16%
Thrombectomy	73%
IV thrombolysis with alteplase	6%
IV heparin infusion	1%
Decompressive hemicraniectomy	3%

For thrombectomy in acute ischaemic stroke, an extended target time of 6-24 hours may be considered if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Important for me Less important

This patient has had an ischaemic stroke, likely as a result of inadequate anticoagulation in the context of atrial fibrillation. As the symptoms were first noticed during breakfast, this patient is outside the 4.5 hour time window in which thrombolysis can be attempted.

Despite this, his CT perfusion scan shows only a small area of infarct, surrounded by a large area of potentially salvageable ischaemic tissue (the penumbra). NICE guidelines suggest that thrombectomy should be offered as soon as possible to patients who have suffered an ischaemic stroke (as demonstrated by CTA) within 6 to 24 hours (including wake-up strokes) *if there is the potential to salvage brain tissue* (as shown on CT perfusion imaging or diffusion-weighted MRI).

Patients who have a pre-existing disability (e.g. frailty, poor functional baseline) often have poor outcomes even if thrombectomy is successful, so NICE recommends that thrombectomy is reserved for those with a pre-stroke Modified Rankin score of <3. This patient meets these inclusion criteria given his good baseline function.

Patients who are outside the time window or otherwise do not meet the criteria for thrombolysis/thrombectomy should be managed on a stroke ward with antiplatelet agents and control of risk factors (e.g. blood pressure control, anticoagulation for atrial fibrillation commenced 2 weeks after the stroke).

IV heparin infusions are not used to treat ischaemic strokes. Unfractionated heparin is used for rapid anticoagulation (in the treatment of thromboembolic disease for example) in patients who are at high risk of bleeding (the anticoagulant effect can be terminated rapidly by stopping the infusion).

A decompressive hemicraniectomy is reserved for malignant middle cerebral artery syndrome.

👍 | 👎 | Discuss (4) | Improve

Next question >

Stroke: management of acute ischaemic stroke ★

The National Clinical Guideline for Stroke was published in 2023 and is a partial update of the 2016 Royal College of Physicians (RCP) guidelines.

Selected points relating to the management of acute stroke include:

- blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits
- blood pressure
    - blood pressure should not be lowered in the acute phase of ischaemic stroke unless there are complications e.g. Hypertensive encephalopathy or they are being considered for thrombolysis (see below)
    - blood pressure control should be considered for patients who present with an acute ischaemic stroke, if they present within 6 hours and have a systolic blood pressure > 150 mmHg
- aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded
- with regards to atrial fibrillation, the RCP state: 'anticoagulants should not be started until brain imaging has excluded haemorrhage, and usually not until 14 days have passed from the onset of an ischaemic stroke'
- if the cholesterol is > 3.5 mmol/l patients should be commenced on a statin. Many physicians will delay treatment until after at least 48 hours due to the risk of haemorrhagic transformation

Thrombolysis for acute ischaemic stroke

Thrombolysis is now given to around 10% of patients who present with an acute ischaemic stroke in the UK. It requires a dedicated team to determine eligibility, ensure timely delivery and provide hyperacute stroke services to patients afterwards.

The standard criteria for thrombolysis with alteplase or tenecteplase are as follows:

- it is administered within 4.5 hours of onset of stroke symptoms
- haemorrhage has been definitively excluded (i.e. Imaging has been performed)

The 2023 National Clinical Guideline for Stroke broadened the potential inclusion criteria.

- it recommends that patients with an acute ischaemic stroke, regardless of age or stroke severity, who were last known to be well more than 4.5 hours earlier, should be considered for thrombolysis with alteplase if:
    - treatment can be started between 4.5 and 9 hours of known onset, or within 9 hours of the midpoint of sleep when they have woken with symptoms, AND
    - they have evidence from CT/MR perfusion (core-perfusion mismatch) or MRI (DWI-FLAIR mismatch) of the potential to salvage brain tissue
- this should be irrespective of whether they have a large artery occlusion and require mechanical thrombectomy.

- there are specific criteria in the guidelines that determine the imagine criteria that determine whether thrombolysis should be performed

Blood pressure should be lowered to 185/110 mmHg before thrombolysis.

Contraindications to thrombolysis

Absolute	Relative
- Previous intracranial haemorrhage - Seizure at onset of stroke - Intracranial neoplasm - Suspected subarachnoid haemorrhage - Stroke or traumatic brain injury in preceding 3 months - Lumbar puncture in preceding 7 days - Gastrointestinal haemorrhage in preceding 3 weeks - Active bleeding - Oesophageal varices - Uncontrolled hypertension >200/120mmHg	- Pregnancy - Concurrent anticoagulation (INR >1.7) - Haemorrhagic diathesis - Active diabetic haemorrhagic retinopathy - Suspected intracardiac thrombus - Major surgery / trauma in the preceding 2 weeks

Thrombectomy for acute ischaemic stroke

Mechanical thrombectomy is an exciting new treatment option for patients with an acute ischaemic stroke. NICE incorporated recommendations into their 2019 guidelines. It is important to remember the significant resources and senior personnel to provide such a service 24 hours a day. NICE recommend that all decisions about thrombectomy take into account a patient's overall clinical status:

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS)

Offer thrombectomy as soon as possible and within 6 hours of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have:
acute ischaemic stroke and

- confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA)

Offer thrombectomy as soon as possible to people who were last known to be well between 6

hours and 24 hours previously (including wake-up strokes):

- confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to 24 hours previously (including wake-up strokes):

- who have acute ischaemic stroke and confirmed occlusion of the **proximal posterior circulation (that is, basilar or posterior cerebral artery)** demonstrated by CTA or MRA and
- if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

Secondary prevention

Recommendations from NICE include:

- clopidogrel is now recommended by NICE ahead of combination use of aspirin plus modified-release (MR) dipyridamole in people who have had an ischaemic stroke
- aspirin is recommended after an ischaemic stroke only if clopidogrel is contraindicated or not tolerated

Carotid endarterectomy is recommend if the patient has suffered a stroke or TIA in the carotid territory and is not severely disabled

- should only be considered if the stenosis > 50% according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

- it should be noted that previous guidelines have used other reporting methods, that have resulted in different cut-offs, e.g. European Carotid Surgery Trial (ECST) with a cut-off of 70%

- it should be performed as soon as possible within 7 days

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 11 👎 15

Thrombolysis and thrombectomy for acute ischaemic stroke

Clinical Knowledge Summaries 👍 16 👎 15

Stroke and TIA guidelines

Age and Ageing 👍 20 👎 16

Interesting article on managing blood pressure during acute stroke

National Clinical Guideline for Stroke 👍 5 👎 3

2023 National Clinical Guideline for Stroke

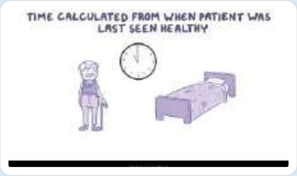
NICE 👍 36 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

Suggest link

Report broken link

Media


TIME CALCULATED FROM WHEN PATIENT WAS LAST SEEN HEALTHY

Stroke

Osmosis - YouTube 👍 12 👎 0

Radiopaedia
Acute Ischaemic Stroke

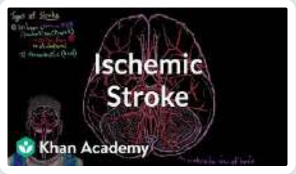
GB Search Avatar image Stroke: Acute infarction - radiology video tutorial (CT, MRI, angiography)

Radiology Channel - YouTube 👍 9 👎 0

Management of ischaemic stroke

Stroke

Podmedics - YouTube 👍 2 👎 1


Ischemic Stroke
Khan Academy

Iscchaemic stroke

Khan Academy - YouTube 👍 3 👎 2

Hemorrhagic Strokes
Khan Academy

Haemorrhagic stroke

Khan Academy - YouTube 👍 1 👎 1

Report broken media

Score: **20%**

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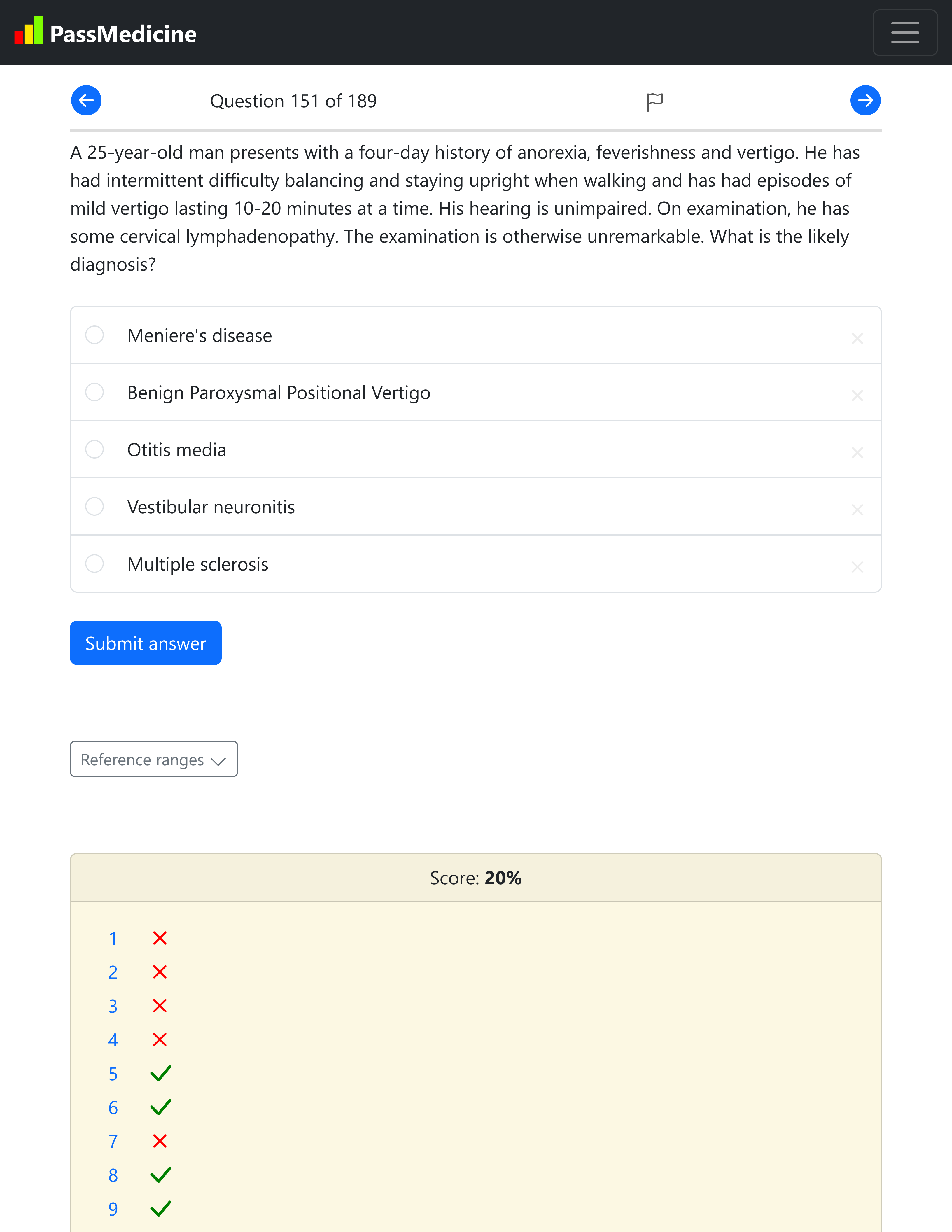

Question 151 of 189

A 25-year-old man presents with a four-day history of anorexia, feverishness and vertigo. He has had intermittent difficulty balancing and staying upright when walking and has had episodes of mild vertigo lasting 10-20 minutes at a time. His hearing is unimpaired. On examination, he has some cervical lymphadenopathy. The examination is otherwise unremarkable. What is the likely diagnosis?

- ○ Meniere's disease
- ○ Benign Paroxysmal Positional Vertigo
- ○ Otitis media
- ○ Vestibular neuronitis
- ○ Multiple sclerosis

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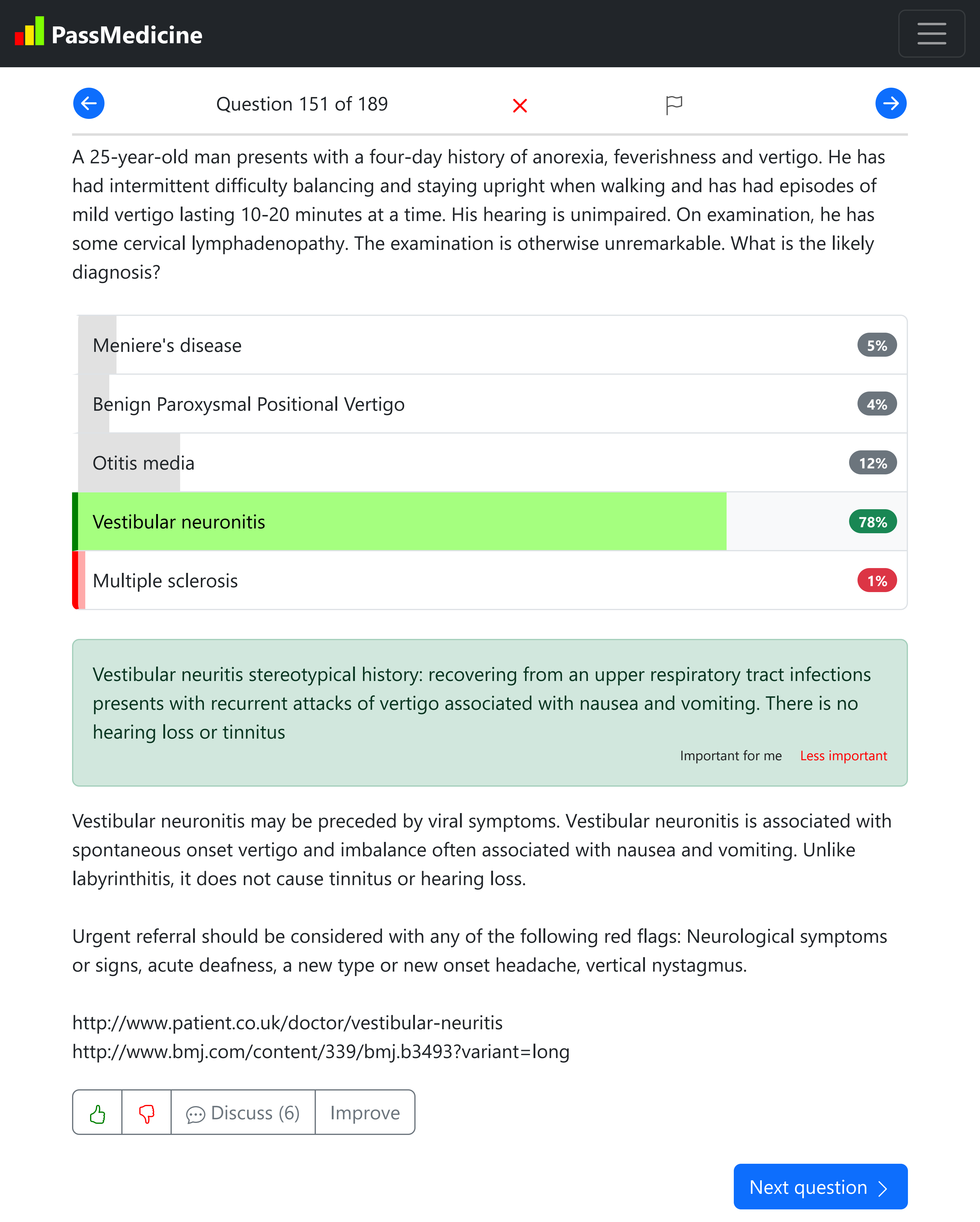


A 25-year-old man presents with a four-day history of anorexia, feverishness and vertigo. He has had intermittent difficulty balancing and staying upright when walking and has had episodes of mild vertigo lasting 10-20 minutes at a time. His hearing is unimpaired. On examination, he has some cervical lymphadenopathy. The examination is otherwise unremarkable. What is the likely diagnosis?

Meniere's disease	5%
Benign Paroxysmal Positional Vertigo	4%
Otitis media	12%
Vestibular neuronitis	78%
Multiple sclerosis	1%

Vestibular neuritis stereotypical history: recovering from an upper respiratory tract infections presents with recurrent attacks of vertigo associated with nausea and vomiting. There is no hearing loss or tinnitus

Vestibular neuronitis may be preceded by viral symptoms. Vestibular neuronitis is associated with spontaneous onset vertigo and imbalance often associated with nausea and vomiting. Unlike labyrinthitis, it does not cause tinnitus or hearing loss.

Urgent referral should be considered with any of the following red flags: Neurological symptoms or signs, acute deafness, a new type or new onset headache, vertical nystagmus.

http://www.patient.co.uk/doctor/vestibular-neuritis
http://www.bmj.com/content/339/bmj.b3493?variant=long

Vertigo

Vertigo may be defined as the false sensation that the body or environment is moving.

The table below lists the main characteristics of the most important causes of vertigo

Disorder	Notes
Viral labyrinthitis	Recent viral infection Sudden onset Nausea and vomiting Hearing may be affected
Vestibular neuronitis	Recent viral infection Recurrent vertigo attacks lasting hours or days No hearing loss
Benign paroxysmal positional vertigo	Gradual onset Triggered by change in head position Each episode lasts 10-20 seconds
Meniere's disease	Associated with hearing loss, tinnitus and sensation of fullness or pressure in one or both ears
Vertebrobasilar ischaemia	Elderly patient Dizziness on extension of neck
Acoustic neuroma	Hearing loss, vertigo, tinnitus Absent corneal reflex is important sign Associated with neurofibromatosis type 2

Other causes of vertigo include
- posterior circulation stroke
- trauma
- multiple sclerosis
- ototoxicity e.g. gentamicin

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 11 👎 6

Vestibular neuritis and labyrinthitis

Suggest link

Report broken link

Media

Normal HIT (worrisome)

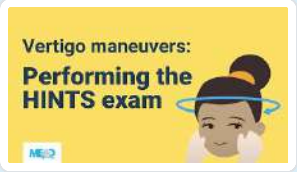
The HINTS exam in vertigo

Individual - YouTube 👍 9 👎 0

VERTIGO
CAUSES OF VERTIGO
INNER EAR
SEMICIRCULAR CANALS
VESTIBULAR NUCLEUS
VESTIBULAR NEURITIS

Understanding the Causes of Vertigo

Zero to Finals - YouTube 👍 2 👎 0


Vertigo maneuvers:
Performing the HINTS exam

Vertigo maneuvers: Performing the HINTS exam

Medmastery - YouTube 👍 1 👎 1

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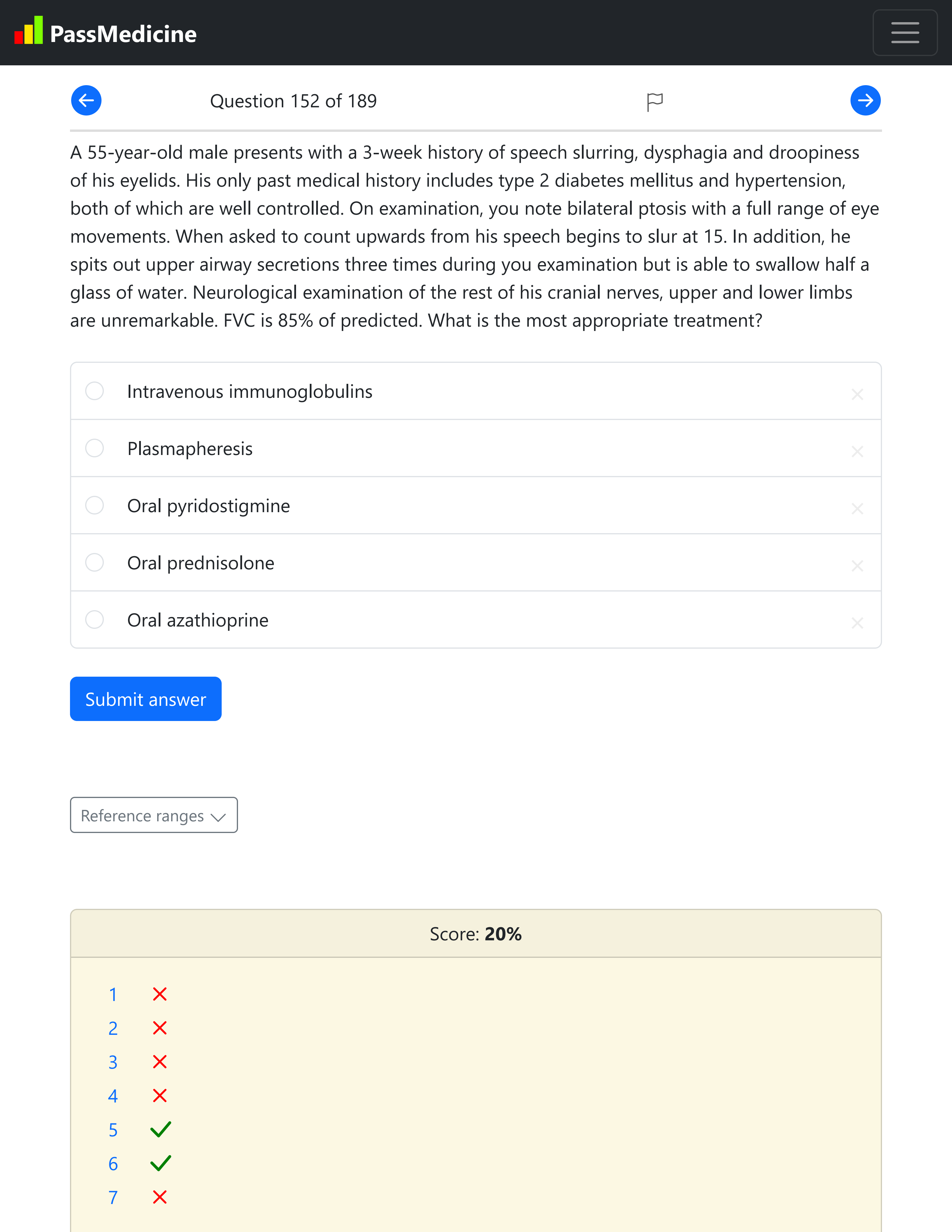

Question 152 of 189

A 55-year-old male presents with a 3-week history of speech slurring, dysphagia and droopiness of his eyelids. His only past medical history includes type 2 diabetes mellitus and hypertension, both of which are well controlled. On examination, you note bilateral ptosis with a full range of eye movements. When asked to count upwards from his speech begins to slur at 15. In addition, he spits out upper airway secretions three times during you examination but is able to swallow half a glass of water. Neurological examination of the rest of his cranial nerves, upper and lower limbs are unremarkable. FVC is 85% of predicted. What is the most appropriate treatment?

○ Intravenous immunoglobulins

○ Plasmapheresis

○ Oral pyridostigmine

○ Oral prednisolone

○ Oral azathioprine

Score: **20%**

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152 -
153 -
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155 -

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A 55-year-old male presents with a 3-week history of speech slurring, dysphagia and droopiness of his eyelids. His only past medical history includes type 2 diabetes mellitus and hypertension, both of which are well controlled. On examination, you note bilateral ptosis with a full range of eye movements. When asked to count upwards from his speech begins to slur at 15. In addition, he spits out upper airway secretions three times during you examination but is able to swallow half a glass of water. Neurological examination of the rest of his cranial nerves, upper and lower limbs are unremarkable. FVC is 85% of predicted. What is the most appropriate treatment?

Intravenous immunoglobulins	24%
Plasmapheresis	7%
Oral pyridostigmine	66%
Oral prednisolone	3%
Oral azathioprine	0%

Pyridostigmine is a long-acting acetylcholinesterase inhibitor that reduces the breakdown of acetylcholine in the neuromuscular junction, temporarily improving symptoms of myasthenia gravis

Important for me   Less important

The patient describes a clear history of bulbar and ocular myasthenia gravis. The key distinction is that this patient is not in crisis when respiratory function is compromised or complete dysphagia is noted. During a crisis, IVIg and plasmapheresis are indicated but not in a new diagnosis setting. The appropriate treatment is pyridostigmine, an acetylcholinesterase inhibitor. An immunomodulating agent is added only if the patient remains symptomatic with pyridostigmine. Steroids such as prednisolone or azathioprine, ciclosporin or mycophenolate mofetil are reasonable choices. Neither would immediately be required in a new diagnosis.

Discuss (3)   Improve

Next question

Myasthenia gravis

Myasthenia gravis is an autoimmune disorder resulting in insufficient functioning acetylcholine receptors. Antibodies to acetylcholine receptors are seen in 85-90% of cases*. Myasthenia is more common in women (2:1)

The key feature is muscle fatigability - muscles become progressively weaker during periods of activity and slowly improve after periods of rest:

- extraocular muscle weakness: diplopia
- proximal muscle weakness: face, neck, limb girdle
- ptosis
- dysphagia

Associations

- thymomas in 15%
- autoimmune disorders: pernicious anaemia, autoimmune thyroid disorders, rheumatoid, SLE
- thymic hyperplasia in 50-70%

Investigations

- single fibre electromyography: high sensitivity (92-100%)
- CT thorax to exclude thymoma
- CK normal
- antibodies to acetylcholine receptors
    - positive in around 85-90% of patients
    - n the remaining patients, about about 40% are positive for anti-muscle-specific tyrosine kinase antibodies
- Tensilon test: IV edrophonium reduces muscle weakness temporarily - not commonly used any more due to the risk of cardiac arrhythmia

Management

- long-acting acetylcholinesterase inhibitors
    - pyridostigmine is first-line
- immunosuppression is usually not started at diagnosis, but the majority of patients eventually require it in addition to long-acting acetylcholinesterase inhibitors:
    - prednisolone initially
    - azathioprine, cyclosporine, mycophenolate mofetil may also be used
- thymectomy

Management of myasthenic crisis

- plasmapheresis
- intravenous immunoglobulins

*antibodies are less commonly seen in disease limited to the ocular muscles

123

Next question

Textbooks

High-yield textbook

Extended textbook

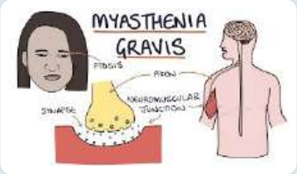
Links

Postgraduate Medical Journal 13 3

Review of myasthenia gravis

Suggest link Report broken link

Media


MYASTHENIA GRAVIS
PTOSIS
AXON
NEUROMUSCULAR JUNCTION
SYNAPSE

Myasthenia gravis

Zero to Finals - YouTube 12 1

NEUROLOGY
MYASTHENIA GRAVIS

Myasthenia gravis

Armando Hasudungan - YouTube 👍 1 👎 1

MYASTHENIA GRAVIS

Myasthenia gravis - causes, symptoms, treatment, pathology

Osmosis - YouTube 👍 2 👎 2

Report broken media

Score: **20.6%**

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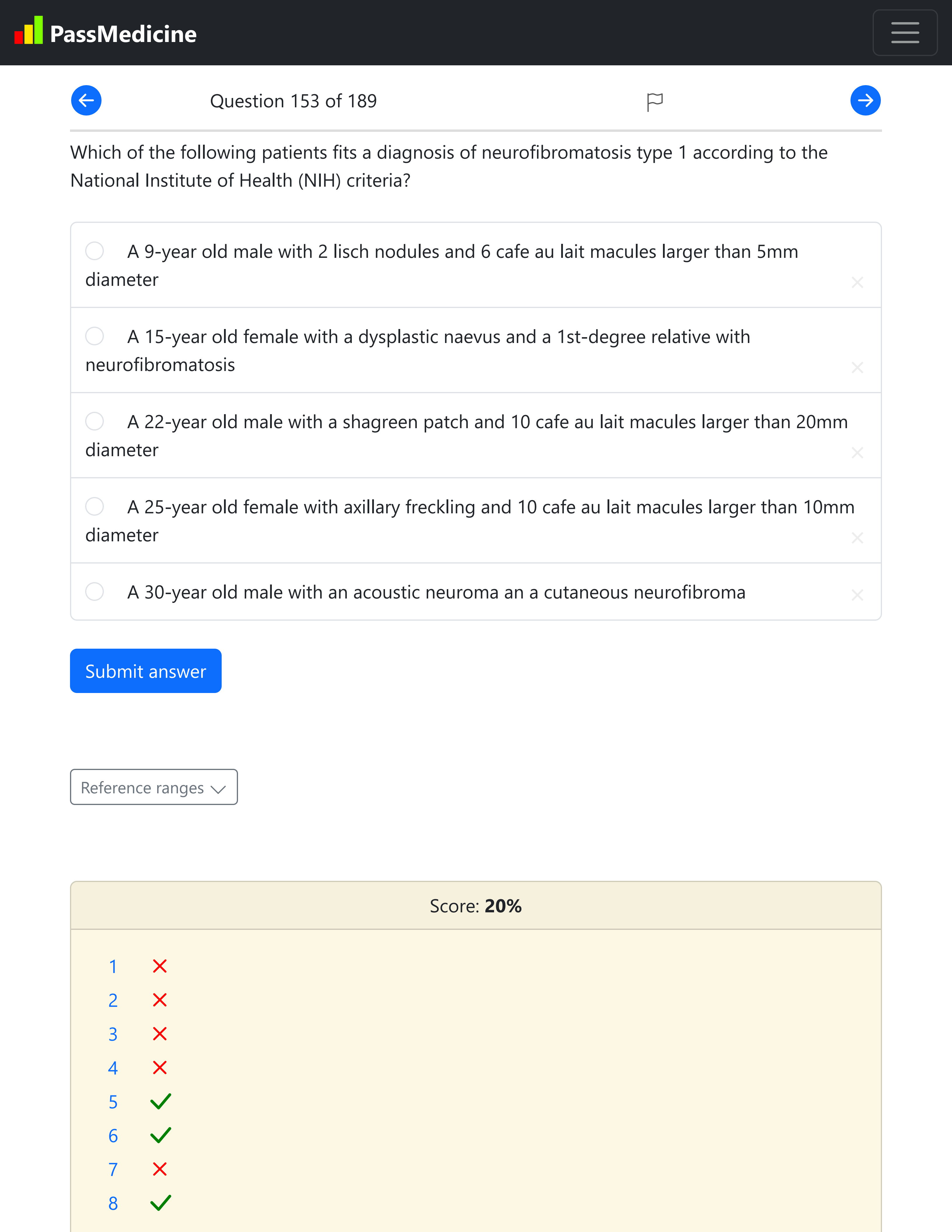

Question 153 of 189

Which of the following patients fits a diagnosis of neurofibromatosis type 1 according to the National Institute of Health (NIH) criteria?

○ A 9-year old male with 2 lisch nodules and 6 cafe au lait macules larger than 5mm diameter

○ A 15-year old female with a dysplastic naevus and a 1st-degree relative with neurofibromatosis

○ A 22-year old male with a shagreen patch and 10 cafe au lait macules larger than 20mm diameter

○ A 25-year old female with axillary freckling and 10 cafe au lait macules larger than 10mm diameter

○ A 30-year old male with an acoustic neuroma an a cutaneous neurofibroma

Score: **20%**

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Which of the following patients fits a diagnosis of neurofibromatosis type 1 according to the National Institute of Health (NIH) criteria?

A 9-year old male with 2 lisch nodules and 6 cafe au lait macules larger than 5mm diameter	39%
A 15-year old female with a dysplastic naevus and a 1st-degree relative with neurofibromatosis	3%
A 22-year old male with a shagreen patch and 10 cafe au lait macules larger than 20mm diameter	13%
A 25-year old female with axillary freckling and 10 cafe au lait macules larger than 10mm diameter	37%
A 30-year old male with an acoustic neuroma an a cutaneous neurofibroma	7%

The NIH consensus development group published some criteria to aid diagnosis of neurofibromatosis type 1 in 1988, as neurocutaneous disorders can be difficult to distinguish. It is important to note that these criteria focus on cafe au lait macule size in adults and children. A child with 6 or more cafe au lait macules larger than 0.5 cm would fit one of the criteria but they would need to be 1.5 cm or larger in an adult. Other criteria include axillary freckling, 2 or more cutaneous/subcutaneous neurofibromas or one plexiform neurofibroma, optic pathway glioma, bony dysplasia, 2 or more Lisch nodules or a 1st-degree relative with neurofibromatosis type 1. At least two of these criteria are required to reach clinical diagnosis.

Acoustic neuromas are associated with neurofibromatosis type 2.

Discuss (10) | Improve

Next question

Neurofibromatosis

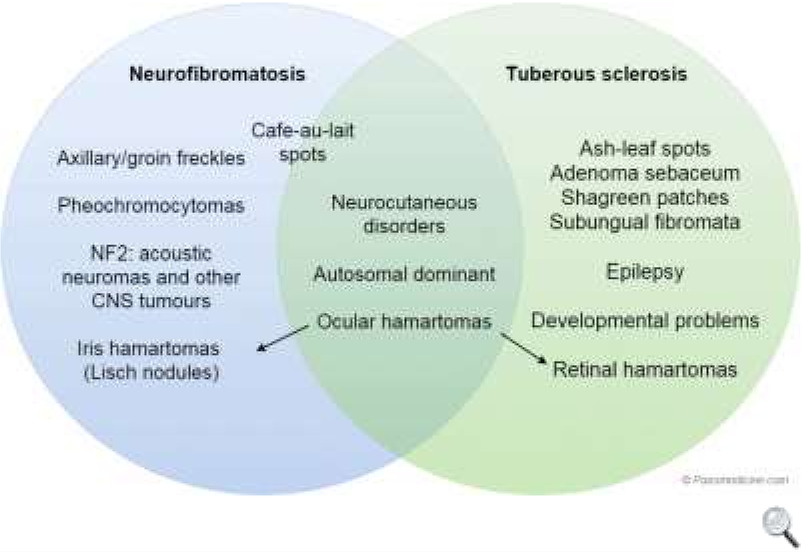
There are two types of neurofibromatosis, NF1 and NF2. Both are inherited in an autosomal dominant fashion

NF1 is also known as von Recklinghausen's syndrome. It is caused by a gene mutation on chromosome 17 which encodes neurofibromin and affects around 1 in 4,000

NF2 is caused by gene mutation on chromosome 22 and affects around 1 in 100,000

Features

NF1	**NF2**
Cafe-au-lait spots (>= 6, 15 mm in diameter) Axillary/groin freckles Peripheral neurofibromas Iris hamatomas (Lisch nodules) in > 90% Scoliosis Pheochromocytomas	Bilateral vestibular schwannomas Multiple intracranial schwannomas, mengiomas and ependymomas


Neurofibromatosis
Tuberous sclerosis
Axillary/groin freckles
Pheochromocytomas
NF2: acoustic neuromas and other CNS tumours
Iris hamartomas (Lisch nodules)
Cafe-au-lait spots
Neurocutaneous disorders
Autosomal dominant
Ocular hamartomas
Ash-leaf spots
Adenoma sebaceum
Shagreen patches
Subungual fibromata
Epilepsy
Developmental problems
Retinal hamartomas
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Comparison of neurofibromatosis and tuberous sclerosis. Note that whilst they are both autosomal dominant neurocutaneous disorders there is little overlap otherwise

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

National Center for Biotechnology Information 👍 6 👎 5

Guidelines for the diagnosis and management of individuals with neurofibromatosis 1

Suggest link

Report broken link

Score: **20.6%**

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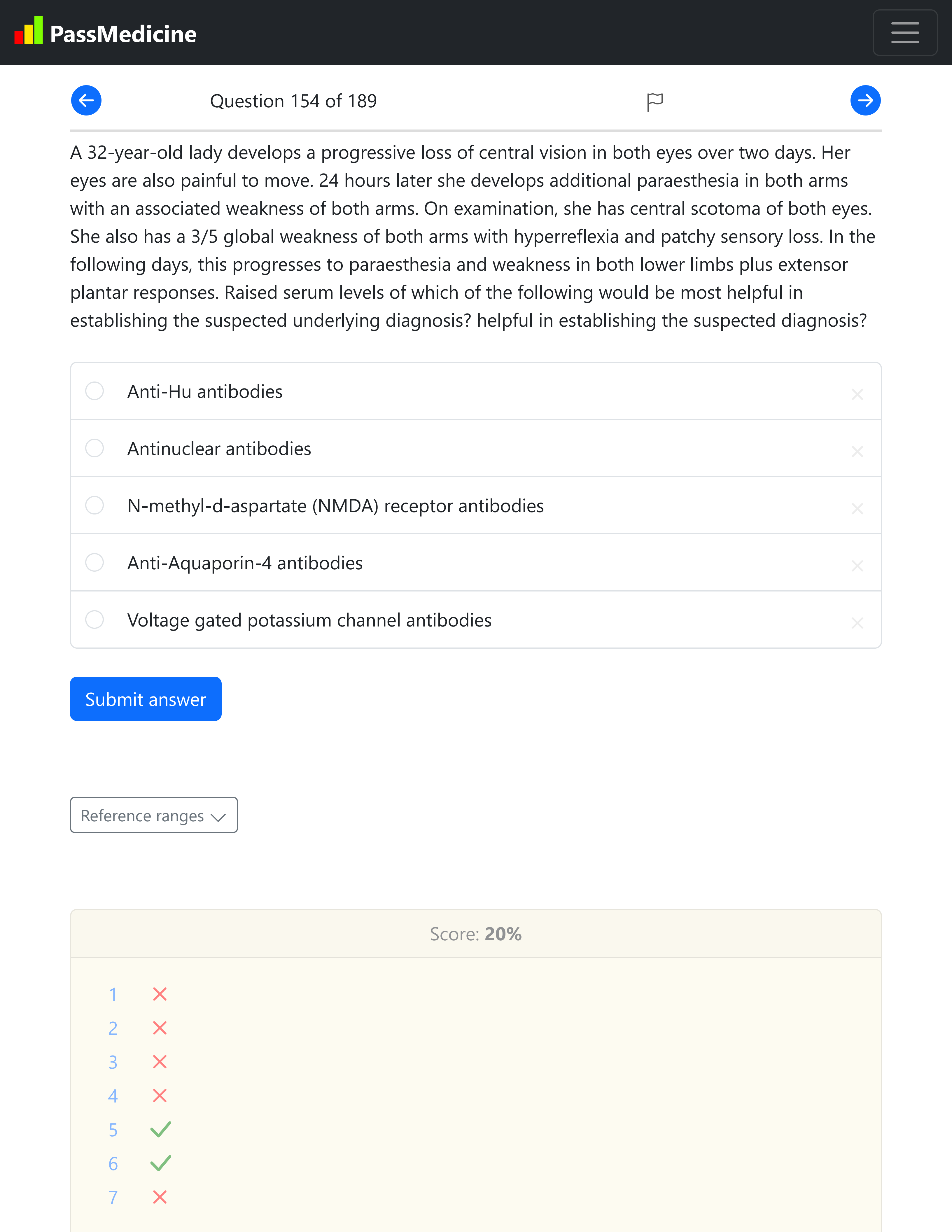

A 32-year-old lady develops a progressive loss of central vision in both eyes over two days. Her eyes are also painful to move. 24 hours later she develops additional paraesthesia in both arms with an associated weakness of both arms. On examination, she has central scotoma of both eyes. She also has a 3/5 global weakness of both arms with hyperreflexia and patchy sensory loss. In the following days, this progresses to paraesthesia and weakness in both lower limbs plus extensor plantar responses. Raised serum levels of which of the following would be most helpful in establishing the suspected underlying diagnosis? helpful in establishing the suspected diagnosis?

○ Anti-Hu antibodies ×

○ Antinuclear antibodies ×

○ N-methyl-d-aspartate (NMDA) receptor antibodies ×

○ Anti-Aquaporin-4 antibodies ×

○ Voltage gated potassium channel antibodies ×

Submit answer

Reference ranges

Score: **20%**

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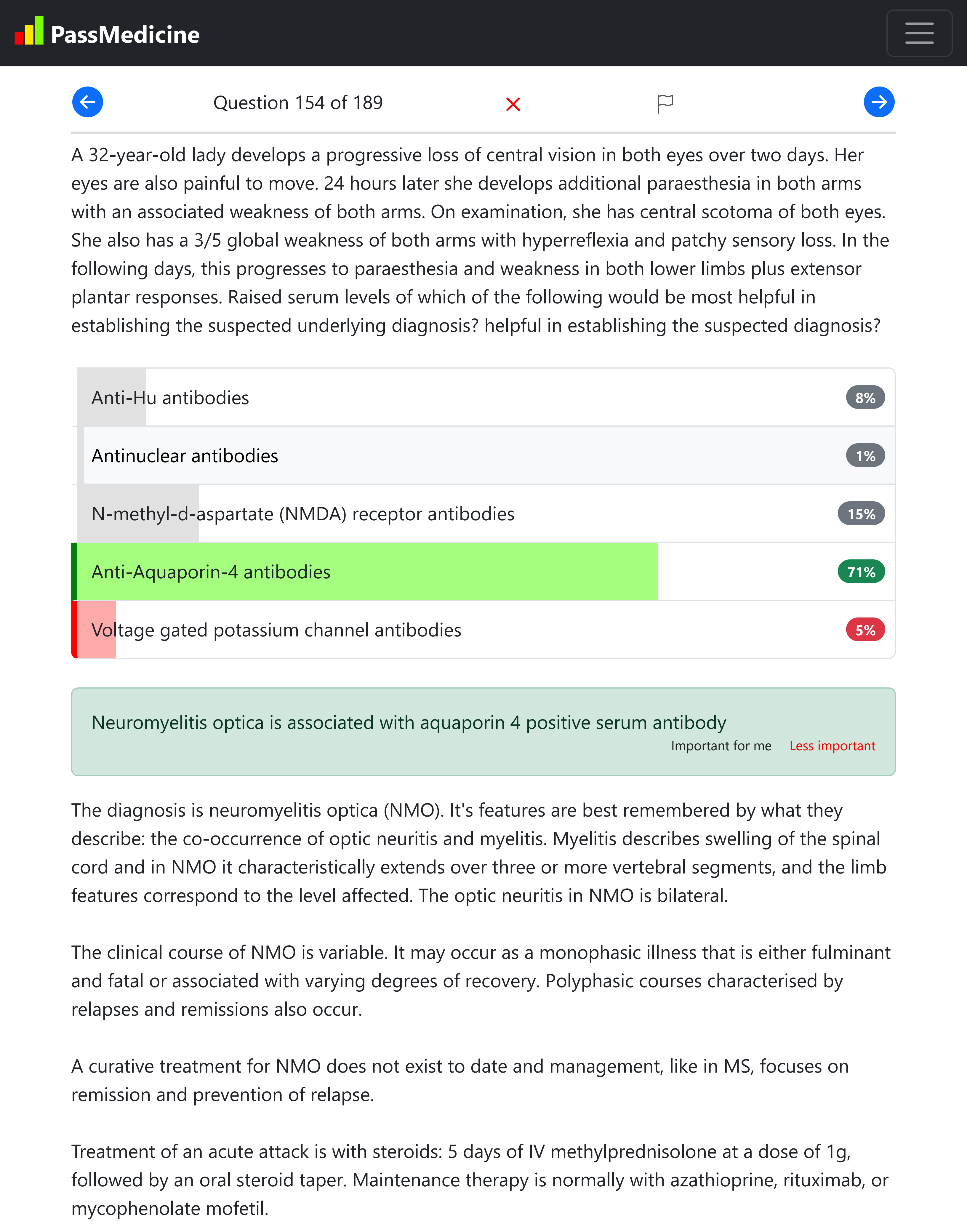

A 32-year-old lady develops a progressive loss of central vision in both eyes over two days. Her eyes are also painful to move. 24 hours later she develops additional paraesthesia in both arms with an associated weakness of both arms. On examination, she has central scotoma of both eyes. She also has a 3/5 global weakness of both arms with hyperreflexia and patchy sensory loss. In the following days, this progresses to paraesthesia and weakness in both lower limbs plus extensor plantar responses. Raised serum levels of which of the following would be most helpful in establishing the suspected underlying diagnosis? helpful in establishing the suspected diagnosis?

Anti-Hu antibodies	8%
Antinuclear antibodies	1%
N-methyl-d-aspartate (NMDA) receptor antibodies	15%
Anti-Aquaporin-4 antibodies	71%
Voltage gated potassium channel antibodies	5%

Neuromyelitis optica is associated with aquaporin 4 positive serum antibody

Important for me Less important

The diagnosis is neuromyelitis optica (NMO). It's features are best remembered by what they describe: the co-occurrence of optic neuritis and myelitis. Myelitis describes swelling of the spinal cord and in NMO it characteristically extends over three or more vertebral segments, and the limb features correspond to the level affected. The optic neuritis in NMO is bilateral.

The clinical course of NMO is variable. It may occur as a monophasic illness that is either fulminant and fatal or associated with varying degrees of recovery. Polyphasic courses characterised by relapses and remissions also occur.

A curative treatment for NMO does not exist to date and management, like in MS, focuses on remission and prevention of relapse.

Treatment of an acute attack is with steroids: 5 days of IV methylprednisolone at a dose of 1g, followed by an oral steroid taper. Maintenance therapy is normally with azathioprine, rituximab, or mycophenolate mofetil.

Discuss (4) Improve

Next question

Neuromyelitis optica ★

Neuromyelitis optica (NMO) is a monophasic or relapsing-remitting demyelinating CNS disorder Although previously thought to be a variant of multiple sclerosis, it is now recognised to be a distinct disease, particularly prevalent in Asian populations. It typically involves the optic nerves and cervical spine, with imaging of the brain frequently normal. Vomiting is also a common presenting complaint.

Diagnosis requires bilateral optic neuritis, myelitis and 2 of the following 3 criteria:

- 1. Spinal cord lesion involving 3 or more spinal levels
- 2. Initially normal MRI brain
- 3. Aquaporin 4 positive serum antibody

Management

- immunosuppressant e.g. with anti-CD20 agent rituximab)

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **20.6%**

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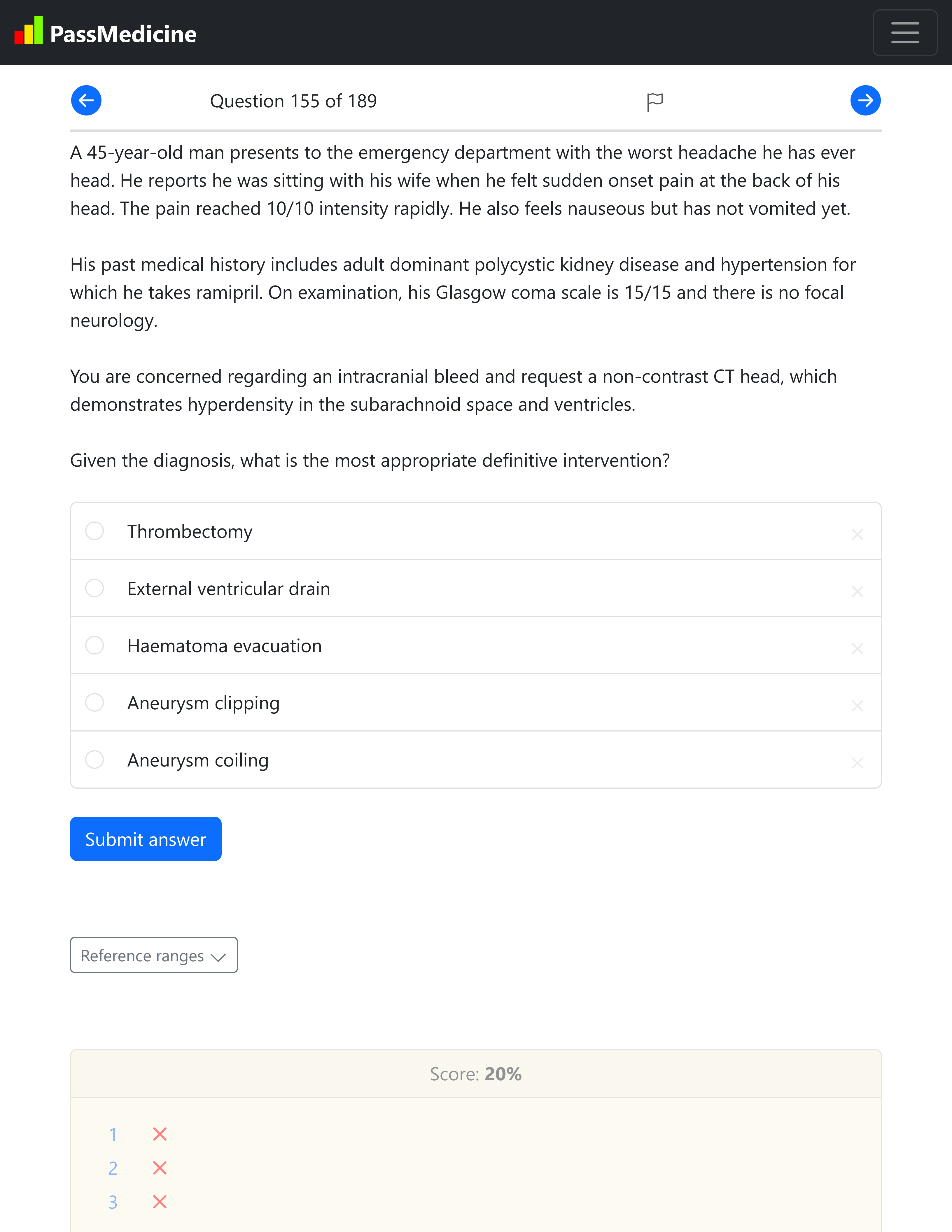

A 45-year-old man presents to the emergency department with the worst headache he has ever head. He reports he was sitting with his wife when he felt sudden onset pain at the back of his head. The pain reached 10/10 intensity rapidly. He also feels nauseous but has not vomited yet.

His past medical history includes adult dominant polycystic kidney disease and hypertension for which he takes ramipril. On examination, his Glasgow coma scale is 15/15 and there is no focal neurology.

You are concerned regarding an intracranial bleed and request a non-contrast CT head, which demonstrates hyperdensity in the subarachnoid space and ventricles.

Given the diagnosis, what is the most appropriate definitive intervention?

○ Thrombectomy

○ External ventricular drain

○ Haematoma evacuation

○ Aneurysm clipping

○ Aneurysm coiling

Score: **20%**

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You are concerned regarding an intracranial bleed and request a non-contrast CT head, which demonstrates hyperdensity in the subarachnoid space and ventricles.

Given the diagnosis, what is the most appropriate definitive intervention?

Thrombectomy	1%
External ventricular drain	2%
Haematoma evacuation	2%
Aneurysm clipping	33%
Aneurysm coiling	63%

Following a subarachnoid haemorrhage, most intracranial aneurysms are now treated with a coil by an interventional neuroradiologist

Important for me Less important

The description given is of a thunderclap headache which is synonymous with a subarachnoid haemorrhage. Further evidence to support this is the CT findings of fresh blood in the subarachnoid space. The likely aetiology is an aneurysmal bleed given the background of ADPKD which is associated with Berry aneurysms. Most aneurysms are now managed by interventional radiology with coils, therefore is the most appropriate definitive intervention.

Thrombectomy is used in at management of acute ischaemic stroke.

External ventricular drains are used to treat complications such as hydrocephalus and therefore is not treating the aneurysm itself.

Haematoma evacuation via craniotomy is a neurosurgical procedure used in the management of moderate to large-sized subdural haematomas.

Aneurysm clipping by neurosurgery is also used to manage aneurysmal bleeds but is not the first-line treatment option.

👍 👎 Discuss (7) Improve

Next question >

Subarachnoid haemorrhage ★

A subarachnoid haemorrhage (SAH) is an intracranial haemorrhage that is defined as the presence of blood within the subarachnoid space, i.e. deep to the subarachnoid layer of the meninges.

The most common cause of SAH is head injury and this is called *traumatic SAH* . In the absence of trauma, SAH is termed *spontaneous SAH* . The rest of this note focuses on spontaneous SAH.

Causes of spontaneous SAH include:

- intracranial aneurysm (saccular 'berry' aneurysms)
  - accounts for around 85% of cases
  - conditions associated with berry aneurysms include hypertension,adult polycystic kidney disease, Ehlers-Danlos syndrome and coarctation of the aorta
- arteriovenous malformation
- pituitary apoplexy
- mycotic (infective) aneurysms

Classical presenting features include:

- headache
  - usually of sudden-onset ('thunderclap' or 'hit with a baseball bat')
  - severe ('worst of my life')
  - occipital
  - typically peaking in intensity within 1 to 5 minutes
  - there may be a history of a less-severe 'sentinel' headache in the weeks prior to presentation
- nausea and vomiting

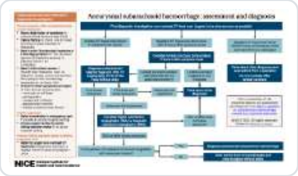
- meningism (photophobia, neck stiffness)
- coma
- seizures
- ECG changes including ST elevation may be seen
    - this may be secondary to either autonomic neural stimulation from the hypothalamus or elevated levels of circulating catecholamines

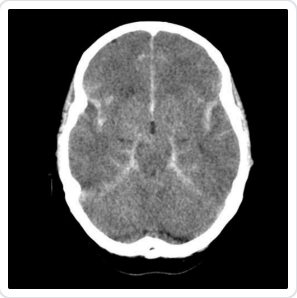
Investigation

- non-contrast CT head is the first-line investigation of choice
    - acute blood (hyperdense/bright on CT) is typically distributed in the basal cisterns, sulci and in severe cases the ventricular system.
- if CT head is done within 6 hours of symptom onset and is normal
    - new guidelines suggest **not** doing a lumbar puncture
    - consider an alternative diagnosis
- if CT head is done more than 6 hours after symptom onset and is normal
    - do a lumber puncture (LP)
    - timing wise the LP should be performed at least 12 hours following the onset of symptoms to allow the development of xanthochromia (the result of red blood cell breakdown).
    - xanthochromia helps to distinguish true SAH from a 'traumatic tap' (blood introduced by the LP procedure).
    - as well as xanthochromia, CSF findings consistent with subarachnoid haemorrhage include a normal or raised opening pressure
- if the CT shows evidence of a SAH
    - referral to neurosurgery to be made as soon as SAH is confirmed

After spontaneous SAH is confirmed, the aim of investigation is to identify a causative pathology that needs urgent treatment:

- CT intracranial angiogram (to identify a vascular lesion e.g. aneurysm or AVM)
- +/- digital subtraction angiogram (catheter angiogram)





Management of a confirmed aneurysmal subarachnoid haemorrhage

- supportive
    - bed rest

    - analgesia
    - venous thromboembolism prophylaxis
    - discontinuation of antithrombotics (reversal of anticoagulation if present)
- vasospasm is prevented using a course of oral nimodipine
- intracranial aneurysms are at risk of rebleeding and therefore require prompt intervention, preferably within 24 hours
    - most intracranial aneurysms are now treated with a coil by interventional neuroradiologists, but a minority require a craniotomy and clipping by a neurosurgeon

Complications of aneurysmal SAH:

- re-bleeding
    - happens in around 10% of cases and most common in the first 12 hours
    - if rebleeding is suspected (e.g. sudden worsening of neurological symptoms) then a repeat CT should be arranged
    - associated with a high mortality (up to 70%)
- hydrocephalus
    - hydrocephalus is temporarily treated with an external ventricular drain (CSF diverted into a bag at the bedside) or, if required, a long-term ventriculoperitoneal shunt
- vasospasm (also termed delayed cerebral ischaemia), typically 7-14 days after onset
    - ensure euvolaemia (normal blood volume)
    - consider treatment with a vasopressor if symptoms persist
- hyponatraemia (most typically due to syndrome inappropriate anti-diuretic hormone (SIADH))
- seizures

Important predictive factors in SAH:

- conscious level on admission
- age
- amount of blood visible on CT head

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia 👍 15 👎 15

Subarachnoid haemorrhage

Life in the Fast Lane 👍 19 👎 10

Example ECG changes in subarachnoid haemorrhage

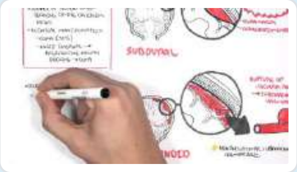
NICE 👍 9 👎 4

2022 Subarachnoid haemorrhage caused by a ruptured aneurysm: diagnosis and management

Suggest link

Report broken link

Media



Intracranial Haemorrhage Types

Armando Hasudungan - YouTube 👍 2 👎 0

Arteriovenous malformation

Osmosis - YouTube 👍 0 👎 0

NEUROLOGY
NEUROSURGERY
SUBARACHNOID
HAEMORRHAGE

Subarachnoid Haemorrhage / pathophysiology, complications and management

Armando Hasudungan - YouTube 👍 0 👎 0

Report broken media

Score: **20.6%**

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A 44-year-old man presents with a fever. He states that it came on a couple of days ago. Today he developed a headache. The headache is frontal, dull in character and was gradual in onset. He explains that he generally keeps well however did experience an episode of fever with associated joint pain a few weeks ago. On examination he has nuchal rigidity. You also note the presence of sensory loss within the right ulnar nerve distribution.

Blood results are as follows:

Hb	134 g/l	Na$^+$	143 mmol/l
Platelets	388 * 10^9/l	K$^+$	3.7 mmol/l
WBC	14.2 * 10^9/l	Urea	5.8 mmol/l
Neuts	7.4 * 10^9/l	Creatinine	51 µmol/l
Lymphs	4.7 * 10^9/l	CRP	82 mg/l
Glucose	6.2 mmol/l		

An ECG was performed:

12 lead ECG	First degree heart block

CSF results are as follows:

White cell count	104/mm*^{3}
White cell differential	95% lymphocytes
Glucose	2.8 mmol/l
Proteins	1250 mg/L (normal 15-450)

What is the most likely pathogen?

○ *Neisseria meningitidis*

○ *Streptococcus pneumoniae*

○ *Borrelia burgdorferi*

○ *Cryptococcus neoformans* ×

○ *Mycobacterium tuberculosis* ×

Submit answer

Reference ranges

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A 44-year-old man presents with a fever. He states that it came on a couple of days ago. Today he developed a headache. The headache is frontal, dull in character and was gradual in onset. He explains that he generally keeps well however did experience an episode of fever with associated joint pain a few weeks ago. On examination he has nuchal rigidity. You also note the presence of sensory loss within the right ulnar nerve distribution.

Blood results are as follows:

Hb	134 g/l	Na^+	143 mmol/l
Platelets	388 * 10^9/l	K^+	3.7 mmol/l
WBC	14.2 * 10^9/l	Urea	5.8 mmol/l
Neuts	7.4 * 10^9/l	Creatinine	51 µmol/l
Lymphs	4.7 * 10^9/l	CRP	82 mg/l
Glucose	6.2 mmol/l		

An ECG was performed:

12 lead ECG	First degree heart block

CSF results are as follows:

White cell count	104/mm*^{3}
White cell differential	95% lymphocytes
Glucose	2.8 mmol/l
Proteins	1250 mg/L (normal 15-450)

What is the most likely pathogen?

Neisseria meningitidis	7%
Streptococcus pneumoniae	6%
Borrelia burgdorferi	62%

Cryptococcus neoformans 7%

Mycobacterium tuberculosis 17%

Central nervous system Lyme disease can be diagnosed with CSF analysis. The results are similar to the 'typical' bacterial picture with low glucose and high protein, however in Lyme disease we would expect a lymphocytic pleocytosis

Important for me Less important

The patients CSF results are suggestive of a bacterial infection with a glucose < 50% of plasma and a very high protein. Common bacterial infections (such as Pneumococcus and Meningococcus) usually result in a neutrophilic pleocytosis. However in this case the patient has a lymphocytic pleocytosis. Causes of this type of picture include partially treated bacterial infection, Lyme disease, tuberculosis, and Cryptococcus.

The extra-CNS clinical features of arthralgia, peripheral neuropathy and first degree heart block are suggestive of Lyme disease. This diagnosis is further supported by the CSF results. *Borrelia burgdorferi* is the bacteria which causes Lyme disease. Intravenous administration of ceftriaxone is the treatment of choice for disseminated Lyme disease.

A partially treated bacterial infection (e.g. Pneumococcus and Meningococcus) could results in the CSF results given, however this patient has yet to be started on treatment.

Mycobacterium tuberculosis and *Cryptococcus neoformans* could also result in this CSF picture, however they are less common generally, and tend to occur in specific patient cohorts with certain risk factors such as HIV. They are less likely diagnoses in this case.

👍 👎 Discuss (5) Improve

Next question >

Cerebrospinal fluid: raised lymphocytes ★

Normal values of cerebrospinal fluid (CSF) are as follows:

- pressure = 60-150 mm (patient recumbent)
- protein = 0.2-0.4 g/l
- glucose = > 2/3 blood glucose
- cells: red cells = 0, white cells < 5/mm^3

The following conditions are associated with raised lymphocytes

- viral meningitis/encephalitis
- TB meningitis
- partially treated bacterial meningitis
- Lyme disease
- Behcet's, SLE
- lymphoma, leukaemia

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20.6%**

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189 ✓

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Question 157 of 189

A 67-year-old woman presents to her GP with progressive numbness and difficulty walking. Furthermore her daughter who was present mentions that she has been behaving strange over the past few months.

She is otherwise fit and well, apart from a ileal resection for treatment-resistant Crohn's disease 9 years ago.

Laboratory tests showed a low haematocrit and mean corpuscular volume of 110 fL. Blood smear analysis noted macrocytic red blood cells with hypersegmented neutrophils.

Which of the following is the most likely cause of the patient's presentation?

○ Ferrochelatase deficiency ×

○ Folate deficiency ×

○ Intrinsic factor deficiency ×

○ Iron deficiency anaemia ×

○ Cobalamin deficiency ×

Submit answer

Reference ranges

Score: **20%**

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A 67-year-old woman presents to her GP with progressive numbness and difficulty walking. Furthermore her daughter who was present mentions that she has been behaving strange over the past few months.

She is otherwise fit and well, apart from a ileal resection for treatment-resistant Crohn's disease 9 years ago.

Laboratory tests showed a low haematocrit and mean corpuscular volume of 110 fL. Blood smear analysis noted macrocytic red blood cells with hypersegmented neutrophils.

Which of the following is the most likely cause of the patient's presentation?

Ferrochelatase deficiency	1%
Folate deficiency	13%
Intrinsic factor deficiency	24%
Iron deficiency anaemia	1%
Cobalamin deficiency	61%

The ileal resection suggests that the patient is not absorbing the vitamin B12-intrinsic factor complex, leading to vitamin B12 deficiency (aka. cobalamin deficiency), and subsequently subacute combined degeneration. This is further supported by the macrocytic anaemia with hypersegmented neutrophils. Folate deficiency causes the same blood film picture but does not present with neurological symptoms.

Discuss (4) | Improve

Next question

Subacute combined degeneration of the spinal cord

Subacute combined degeneration of the spinal cord is due to vitamin B12 deficiency resulting in impairment of the dorsal columns, lateral corticospinal tracts and spinocerebellar tracts..

Recreational nitrous oxide inhalation may also result in vitamin B12 deficiency → subacute combined degeneration of the spinal cord.

Features

- dorsal column involvement
  - distal tingling/burning/sensory loss is symmetrical and tends to affect the legs more than the arms
  - impaired proprioception and vibration sense
- lateral corticospinal tract involvement
  - muscle weakness, hyperreflexia, and spasticity
  - upper motor neuron signs typically develop in the legs first
  - brisk knee reflexes
  - absent ankle jerks
  - extensor plantars
- spinocerebellar tract involvement
  - sensory ataxia → gait abnormalities
  - positive Romberg's sign

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20.6%**

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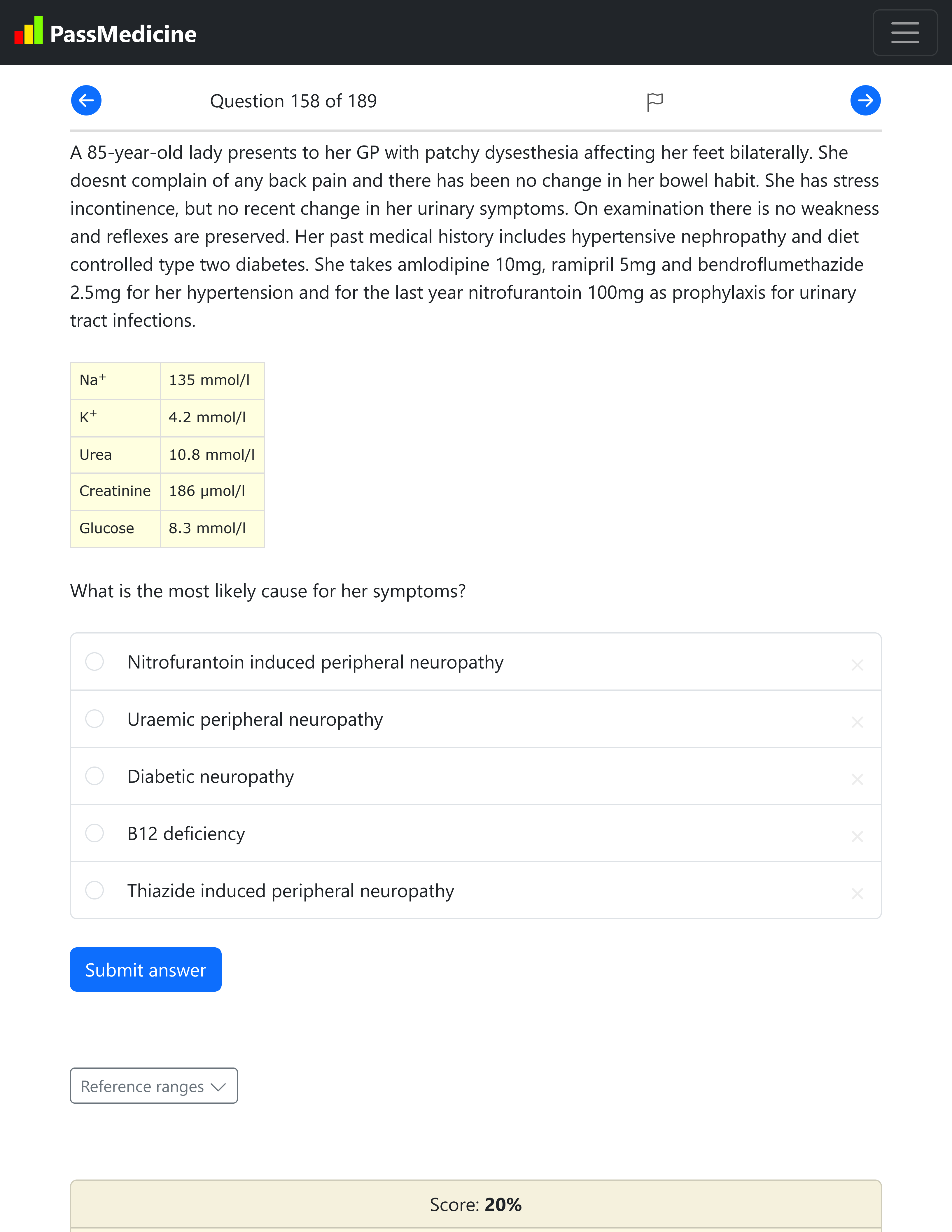

A 85-year-old lady presents to her GP with patchy dysesthesia affecting her feet bilaterally. She doesnt complain of any back pain and there has been no change in her bowel habit. She has stress incontinence, but no recent change in her urinary symptoms. On examination there is no weakness and reflexes are preserved. Her past medical history includes hypertensive nephropathy and diet controlled type two diabetes. She takes amlodipine 10mg, ramipril 5mg and bendroflumethazide 2.5mg for her hypertension and for the last year nitrofurantoin 100mg as prophylaxis for urinary tract infections.

Na^+	135 mmol/l
K^+	4.2 mmol/l
Urea	10.8 mmol/l
Creatinine	186 µmol/l
Glucose	8.3 mmol/l

What is the most likely cause for her symptoms?

○ Nitrofurantoin induced peripheral neuropathy

○ Uraemic peripheral neuropathy

○ Diabetic neuropathy

○ B12 deficiency

○ Thiazide induced peripheral neuropathy

Score: **20%**

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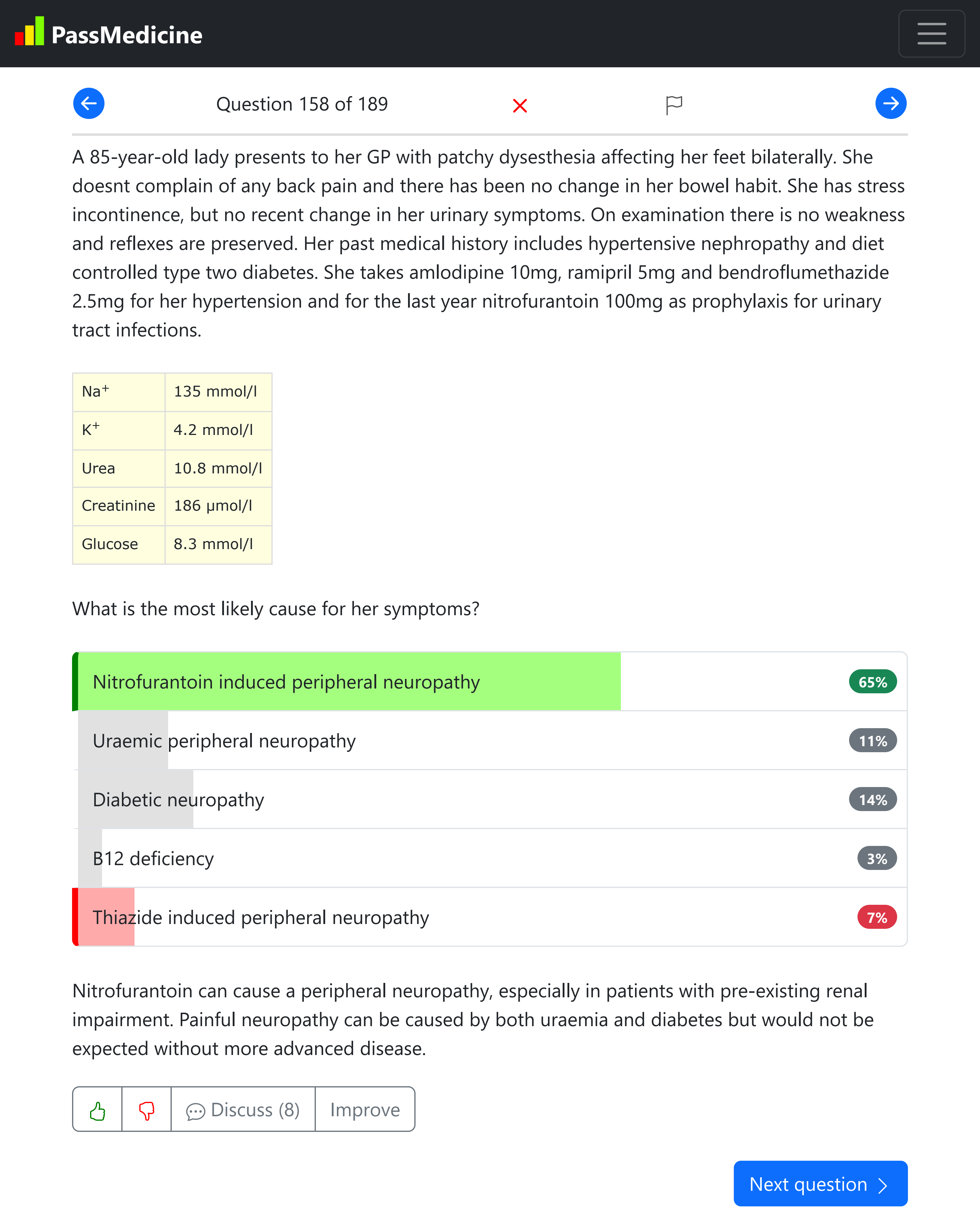

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K^+	4.2 mmol/l
Urea	10.8 mmol/l
Creatinine	186 µmol/l
Glucose	8.3 mmol/l

What is the most likely cause for her symptoms?

Nitrofurantoin induced peripheral neuropathy	65%
Uraemic peripheral neuropathy	11%
Diabetic neuropathy	14%
B12 deficiency	3%
Thiazide induced peripheral neuropathy	7%

Nitrofurantoin can cause a peripheral neuropathy, especially in patients with pre-existing renal impairment. Painful neuropathy can be caused by both uraemia and diabetes but would not be expected without more advanced disease.

Drugs causing peripheral neuropathy ★

Drugs causing a peripheral neuropathy

- amiodarone
- isoniazid
- vincristine
- nitrofurantoin
- metronidazole

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20.6%**

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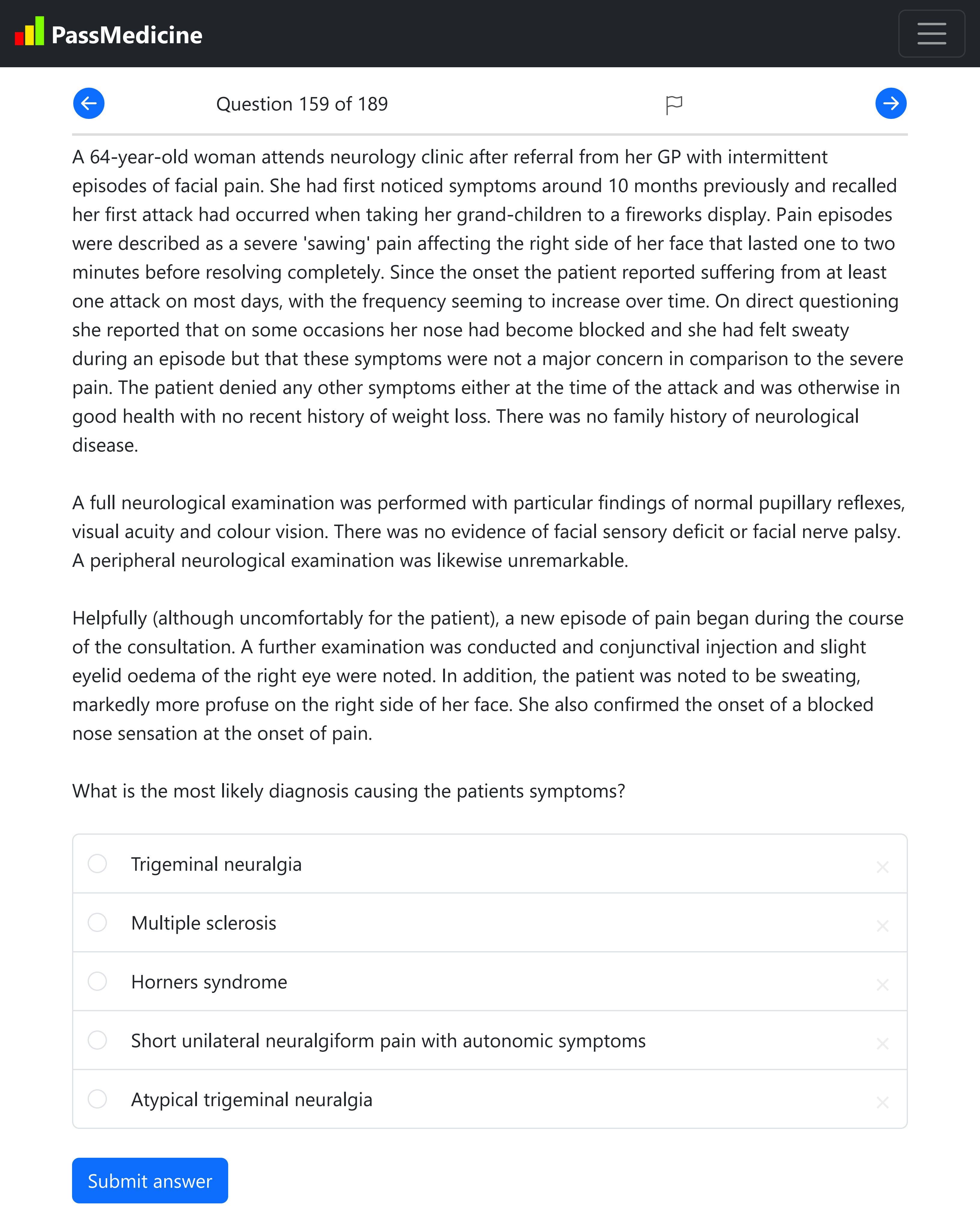

Question 159 of 189

A 64-year-old woman attends neurology clinic after referral from her GP with intermittent episodes of facial pain. She had first noticed symptoms around 10 months previously and recalled her first attack had occurred when taking her grand-children to a fireworks display. Pain episodes were described as a severe 'sawing' pain affecting the right side of her face that lasted one to two minutes before resolving completely. Since the onset the patient reported suffering from at least one attack on most days, with the frequency seeming to increase over time. On direct questioning she reported that on some occasions her nose had become blocked and she had felt sweaty during an episode but that these symptoms were not a major concern in comparison to the severe pain. The patient denied any other symptoms either at the time of the attack and was otherwise in good health with no recent history of weight loss. There was no family history of neurological disease.

A full neurological examination was performed with particular findings of normal pupillary reflexes, visual acuity and colour vision. There was no evidence of facial sensory deficit or facial nerve palsy. A peripheral neurological examination was likewise unremarkable.

Helpfully (although uncomfortably for the patient), a new episode of pain began during the course of the consultation. A further examination was conducted and conjunctival injection and slight eyelid oedema of the right eye were noted. In addition, the patient was noted to be sweating, markedly more profuse on the right side of her face. She also confirmed the onset of a blocked nose sensation at the onset of pain.

What is the most likely diagnosis causing the patients symptoms?

○ Trigeminal neuralgia

○ Multiple sclerosis

○ Horners syndrome

○ Short unilateral neuralgiform pain with autonomic symptoms

○ Atypical trigeminal neuralgia

Reference ranges

Score: **20%**

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What is the most likely diagnosis causing the patients symptoms?

Trigeminal neuralgia	13%
Multiple sclerosis	1%
Horners syndrome	4%
Short unilateral neuralgiform pain with autonomic symptoms	71%
Atypical trigeminal neuralgia	12%

The patients history certainly has features suggestive of trigeminal neuralgia, in particular the character and episodic nature of the pain and that the first episode was particularly memorable. However, the presence of autonomic features associated with attacks points strongly towards the variant short unilateral neuralgiform pain with autonomic symptoms (SUNA) as the correct diagnosis. The distinction is clinically relevant as individuals with autonomic symptoms achieved less benefit from microvascular decompression compared to individuals with trigeminal neuralgia.

Atypical trigeminal neuralgia is the diagnosis made when an individual suffers from persistent lower intensity pain between exacerbations, in contrast to the complete resolution of pain seen in trigeminal neuralgia. Horners syndrome would not cause episodic autonomic symptoms and is not normally associated with pain. Multiple sclerosis is an important differential for trigeminal neuralgia but the patients normal baseline neurological examination and prominent autonomic symptoms make this less likely. If uncertainty persists after clinical assessment, MRI scanning to assess for demyelination plaques is the appropriate investigation.

Zakrzewska J, Linskey M. Trigeminal neuralgia. BMJ 2014;348:g474.

Discuss (6) | Improve

Next question ›

Trigeminal autonomic cephalalgias ★

Trigeminal autonomic cephalalgias (TACs)

- cluster headache
- paroxysmal hemicrania
- hemicrania continua
- short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT syndrome)
- short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms (SUNA)

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Media

NEUROLOGY
TRIGEMINAL AUTONOMIC
CEPHALALGIAS

Severe headaches - Understanding Trigeminal Autonomic Cephalgias - types, pathophysiology, treatment

Armando Hasudungan - YouTube 👍 10 👎 0

Report broken media

Score: **20.6%**

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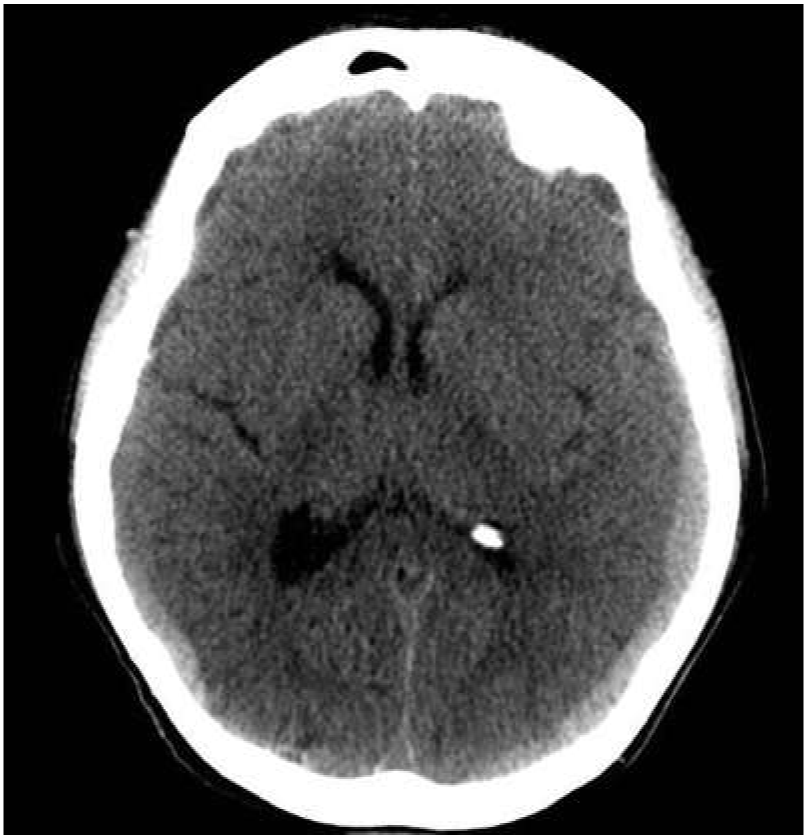
An 81-year-old woman attends the emergency department following a mechanical fall. She has sustained a head injury and a CT head is arranged as part of her trauma survey. Her past medical history includes osteoarthritis of the right hip, recurrent falls, and atrial fibrillation for which she is anticoagulated.

On examination, her respiratory rate is 16/min and is saturating at 96% on air. Her heart rate is 81/min and her blood pressure is 91/64mmHg. Neurological examination elicits a Glasgow coma score of 14 due to confusion but there are no focal neurological deficits. She has no spinal, limb, chest, or abdominal tenderness, and a full painless range of movement in all four limbs.

Her CT head is displayed below:



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What is the diagnosis in this instance?

○ Extradural haematoma

○ Intracerebral haematoma

○ Normal appearances

○ Subarachnoid haemorrhage

○ Subdural haematoma

Submit answer

Reference ranges

Score: **20%**

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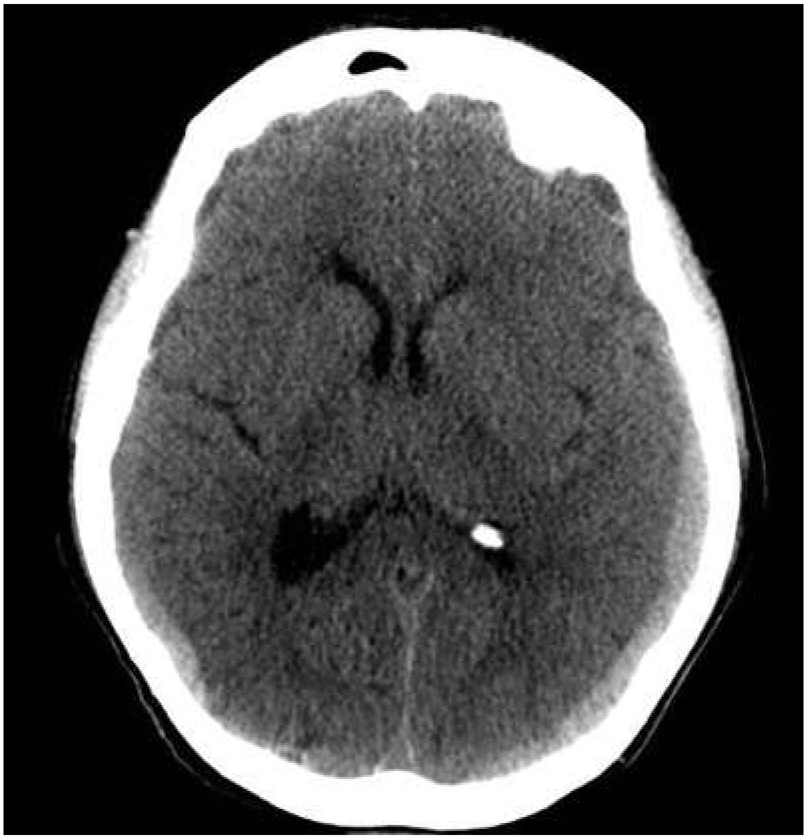

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What is the diagnosis in this instance?

Extradural haematoma 8%

Intracerebral haematoma 4%

Normal appearances	6%
Subarachnoid haemorrhage	7%
Subdural haematoma	75%

This woman has sustained a subdural haematoma. She is at increased risk of sustaining such an insult due to her advanced age and anticoagulation. Note that alcohol excess is also considered a risk factor. The diagnosis is confirmed on the CT scan which shows a concave bleed posteriorly.

An extradural haematoma typically presents with a convex-shaped bleed as it requires more force to detach the dura mater from the cranium. This CT shows a concave shape and is therefore consistent with a subdural rather than an extradural haematoma.

An intracerebral haematoma, as the name suggests, refers to a bleed within the brain parenchyma. As the bleed in this CT is of convex shape along the edge of the cranium, this is not indicative of an intracerebral haematoma.

This CT does not have normal appearances as it displays a haematoma posteriorly. This is therefore an incorrect answer.

A subarachnoid haemorrhage is not the correct answer here. CT images in subarachnoid haemorrhage show blood within the sulci of the brain parenchyma which is not the case here where the bleed is confined to the meninges.

👍 👎 Discuss (3) Improve

Next question >

Head injury: types of traumatic brain injury

Basics
- primary brain injury may be focal (contusion/haematoma) or diffuse (diffuse axonal injury)
- diffuse axonal injury occurs as a result of mechanical shearing following deceleration, causing disruption and tearing of axons
- intra-cranial haematomas can be extradural, subdural or intracerebral, while contusions may occur adjacent to (coup) or contralateral (contre-coup) to the side of impact
- secondary brain injury occurs when cerebral oedema, ischaemia, infection, tonsillar or tentorial herniation exacerbates the original injury. The normal cerebral auto regulatory

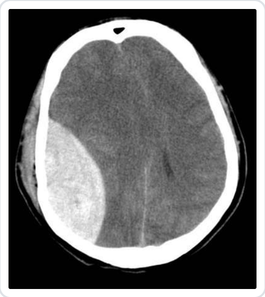
processes are disrupted following trauma rendering the brain more susceptible to blood flow changes and hypoxia
- the Cushings reflex (hypertension and bradycardia) often occurs late and is usually a pre terminal event

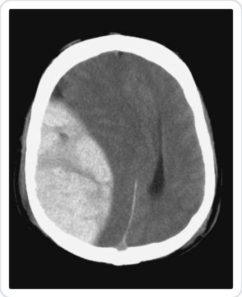
Type of injury	**Notes**
Extradural (epidural) haematoma	Bleeding into the space between the dura mater and the skull. Often results from acceleration-deceleration trauma or a blow to the side of the head. The majority of epidural haematomas occur in the temporal region where skull fractures cause a rupture of the middle meningeal artery. Features • features of raised intracranial pressure • some patients may exhibit a lucid interval
Subdural haematoma	Bleeding into the outermost meningeal layer. Most commonly occur around the frontal and parietal lobes. Risk factors include old age, alcoholism and anticoagulation. Slower onset of symptoms than a epidural haematoma. There may be fluctuating confusion/consciousness
Subarachnoid haemorrhage	Classically causes a sudden occipital headache. Usually occurs spontaneously in the context of a ruptured cerebral aneurysm but may be seen in association with other injuries when a patient has sustained a traumatic brain injury
Intracerebral haematoma	An intracerebral (or intraparenchymal) haemorrhage is a collection of blood within the substance of the brain. Causes / risk factors include: hypertension, vascular lesion (e.g. aneurysm or arteriovenous malformation), cerebral amyloid angiopathy, trauma, brain tumour or infarct (particularly in stroke patients undergoing thrombolysis). Patients will present similarly to an ischaemic stroke (which is why it is crucial to obtain a CT in head in all stroke patients prior to thrombolysis) or with a decrease in consciousness. CT imaging will show a hyperdensity (bright lesion) within the substance of the brain.

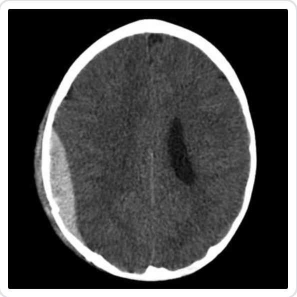
Type of injury	Notes
	Treatment is often conservative under the care of stroke physicians, but large clots in patients with impaired consciousness may warrant surgical evacuation.

Image gallery

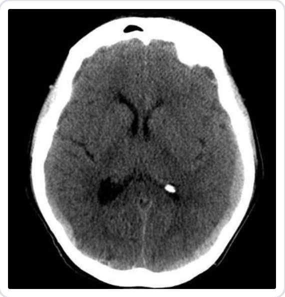
Extradural (epidural) haematoma:

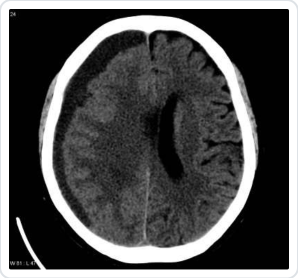


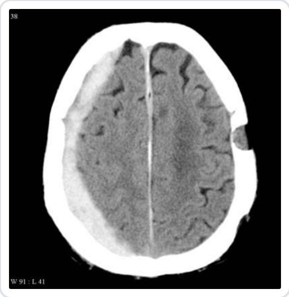




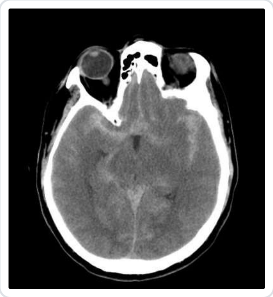
Subdural haematoma:

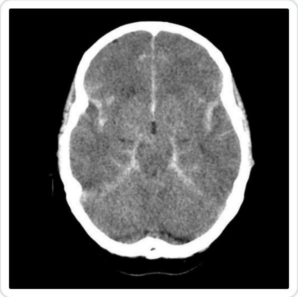






Subarachnoid haemorrhage:





Next question

Textbooks

High-yield textbook

Extended textbook

Links

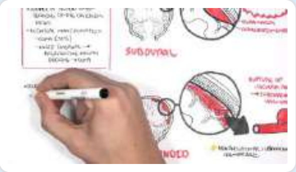
NICE 👍 11 👎 2

2014 Head injury guidelines

Suggest link

Report broken link

Media



Intracranial Haemorrhage Types

Armando Hasudungan - YouTube 👍 6 👎 0

Intracranial
Bleeds
subdural
&
extradural

Intracranial bleeds - Subdural and Extradural haematoma

Soton Brain Hub - YouTube 👍 4 👎 0

TRAUMATIC
BRAIN INJURY
~ PATHOLOGY REVIEW ~
Osmosis.org

Traumatic brain injury: pathology review

Osmosis - YouTube 👍 4 👎 1

Report broken media

Score: **20.6%**

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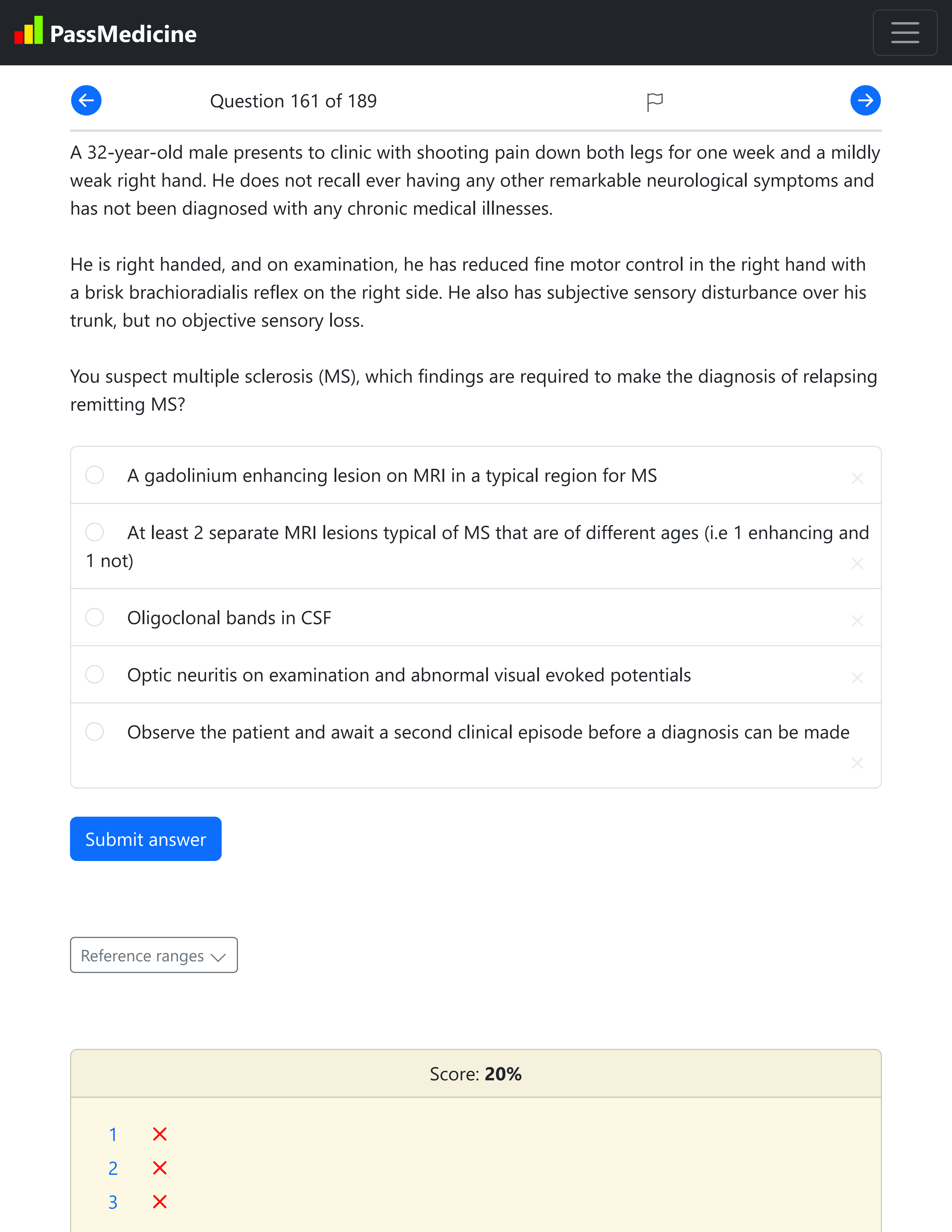

Question 161 of 189

A 32-year-old male presents to clinic with shooting pain down both legs for one week and a mildly weak right hand. He does not recall ever having any other remarkable neurological symptoms and has not been diagnosed with any chronic medical illnesses.

He is right handed, and on examination, he has reduced fine motor control in the right hand with a brisk brachioradialis reflex on the right side. He also has subjective sensory disturbance over his trunk, but no objective sensory loss.

You suspect multiple sclerosis (MS), which findings are required to make the diagnosis of relapsing remitting MS?

○ A gadolinium enhancing lesion on MRI in a typical region for MS

○ At least 2 separate MRI lesions typical of MS that are of different ages (i.e 1 enhancing and 1 not)

○ Oligoclonal bands in CSF

○ Optic neuritis on examination and abnormal visual evoked potentials

○ Observe the patient and await a second clinical episode before a diagnosis can be made

Score: **20%**

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A 32-year-old male presents to clinic with shooting pain down both legs for one week and a mildly weak right hand. He does not recall ever having any other remarkable neurological symptoms and has not been diagnosed with any chronic medical illnesses.

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You suspect multiple sclerosis (MS), which findings are required to make the diagnosis of relapsing remitting MS?

A gadolinium enhancing lesion on MRI in a typical region for MS	6%
At least 2 separate MRI lesions typical of MS that are of different ages (i.e 1 enhancing and 1 not)	75%
Oligoclonal bands in CSF	5%
Optic neuritis on examination and abnormal visual evoked potentials	2%
Observe the patient and await a second clinical episode before a diagnosis can be made	11%

McDonald's criteria are MRI criteria used in the diagnosis of multiple sclerosis. Introduced in 2001, revised in 2005 and again recently in 2010. The latest revision improves sensitivity from 46-74% with a slight trade-off in specificity (slight deterioration from 94-92%)

The diagnosis of multiple sclerosis requires establishing disease disseminated in both space and time.

Dissemination in space:

Dissemination in space requires at least 1 T2 bright lesion in two or more of the following locations:

- periventricular
- juxtacortical
- infratentorial

- spinal cord

Dissemination in time:

Dissemination in time can be established in one of two ways:

i) A new lesion when compared to a previous scan (irrespective of timing)
- T2 bright lesion and/or gadolinium-enhancing lesion

ii) Presence of an asymptomatic enhancing lesion and a non-enhancing T2 bright lesion on any one scan (i.e 2 lesions of differing ages that satisfy the dissemination in space criteria)

👍 | 👎 | Discuss (5) | Improve

Next question ›

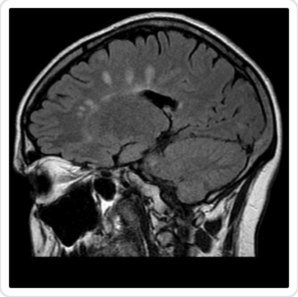
Multiple sclerosis: investigation

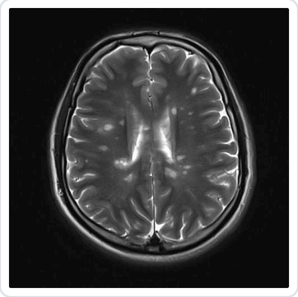
Diagnosis requires demonstration of lesions disseminated in time and space

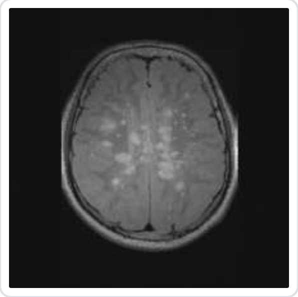
MRI
- high signal T2 lesions
- periventricular plaques
- Dawson fingers: often seen on FLAIR images - hyperintense lesions penpendicular to the corpus callosum

CSF
- oligoclonal bands (and not in serum)
- increased intrathecal synthesis of IgG

Visual evoked potentials
- delayed, but well preserved waveform







123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

DEMYELINATION
T-CELLS
PLAQUES
MULTIPLE SCLEROSIS
OLIGO CLONAL BANDS
RELAPSING-REMITTING

Multiple sclerosis

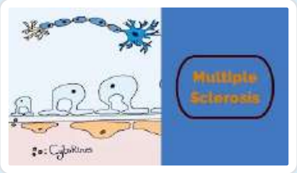
Rhesus Medicine - YouTube 👍 1 👎 0

MULTIPLE SCLEROSIS

Multiple sclerosis - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube

👍 6 👎 1


Multiple Sclerosis

Multiple sclerosis

Soton Brain Hub - YouTube 👍 2 👎 1

Multiple sclerosis

Podmedics - YouTube 👍 1 👎 3

Report broken media

Score: **20.6%**

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Question 162 of 189

A 28-year-old lady presents to the emergency department with a 4-day history of generalised headache that is worse on lying down. She reports it has gradually become worse and she has also noticed blurred vision since yesterday. Her past medical history includes chronic back pain, acne and anxiety. On examination, she is noted to be overweight. Fundoscopy shows papilloedema. Further investigations point to a diagnosis of idiopathic intracranial hypertension. Which of the following of her medications is associated with this condition?

- ○ Tramadol
- ○ Tetracycline antibiotics
- ○ Diazepam
- ○ Amitriptyline
- ○ Ibuprofen

Submit answer

Reference ranges

Score: **20%**

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Tramadol	5%
Tetracycline antibiotics	76%
Diazepam	1%
Amitriptyline	13%
Ibuprofen	4%

Associated medications include, but are not limited to:

- tetracycline antibiotics
- isotretinoin
- contraceptives
- steroids
- levothyroxine
- lithium
- cimetidine

Discuss (2) Improve

Next question ›

Idiopathic intracranial hypertension

Idiopathic intracranial hypertension (also known as pseudotumour cerebri and formerly benign intracranial hypertension) is a condition classically seen in young, overweight females.

Risk factors

- obesity
- female sex
- pregnancy
- drugs
  - combined oral contraceptive pill
  - steroids
  - tetracyclines
  - retinoids (isotretinoin, tretinoin) / vitamin A
  - lithium

Features

- headache
- blurred vision
- papilloedema (usually present)
- enlarged blind spot
- sixth nerve palsy may be present

Management

- weight loss
  - whilst diet and exercise are important, medications such as semaglitide and topiramate may be considered by specialists. Topiramate is particularly beneficial as it also inhibits carbonic anhydrase
- carbonic anhydrase inhibitors e.g. acetazolamide
- topiramate is also used, and has the added benefit of causing weight loss in most patients
- repeated lumbar puncture may be used as a temporary measure but is not suitable for longer-term management
- surgery: optic nerve sheath decompression and fenestration may be needed to prevent damage to the optic nerve. A lumboperitoneal or ventriculoperitoneal shunt may also be performed to reduce intracranial pressure

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 12 👎 13

Idiopathic intracranial hypertension review

Suggest link Report broken link

Score: **20.6%**

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Back to top

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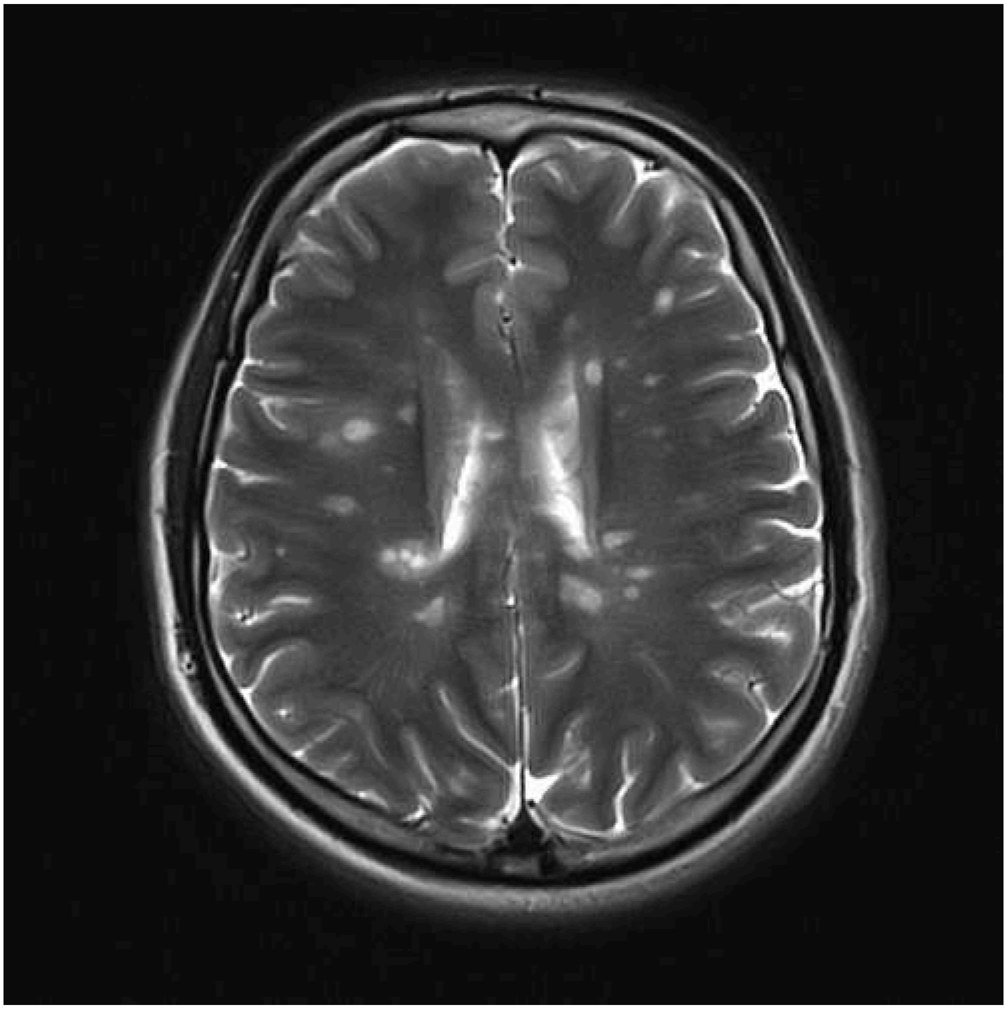
A MRI head is performed. The T2 images are shown below:



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What is the most likely diagnosis?

○ Creutzfeldt-Jakob disease ×

○ Multiple sclerosis ×

○ Adrenoleucodystrophy ×

○ Cerebral toxoplasmosis ×

○ Gliolastoma multiforme ×

Submit answer

Reference ranges ⌵

Score: **20%**

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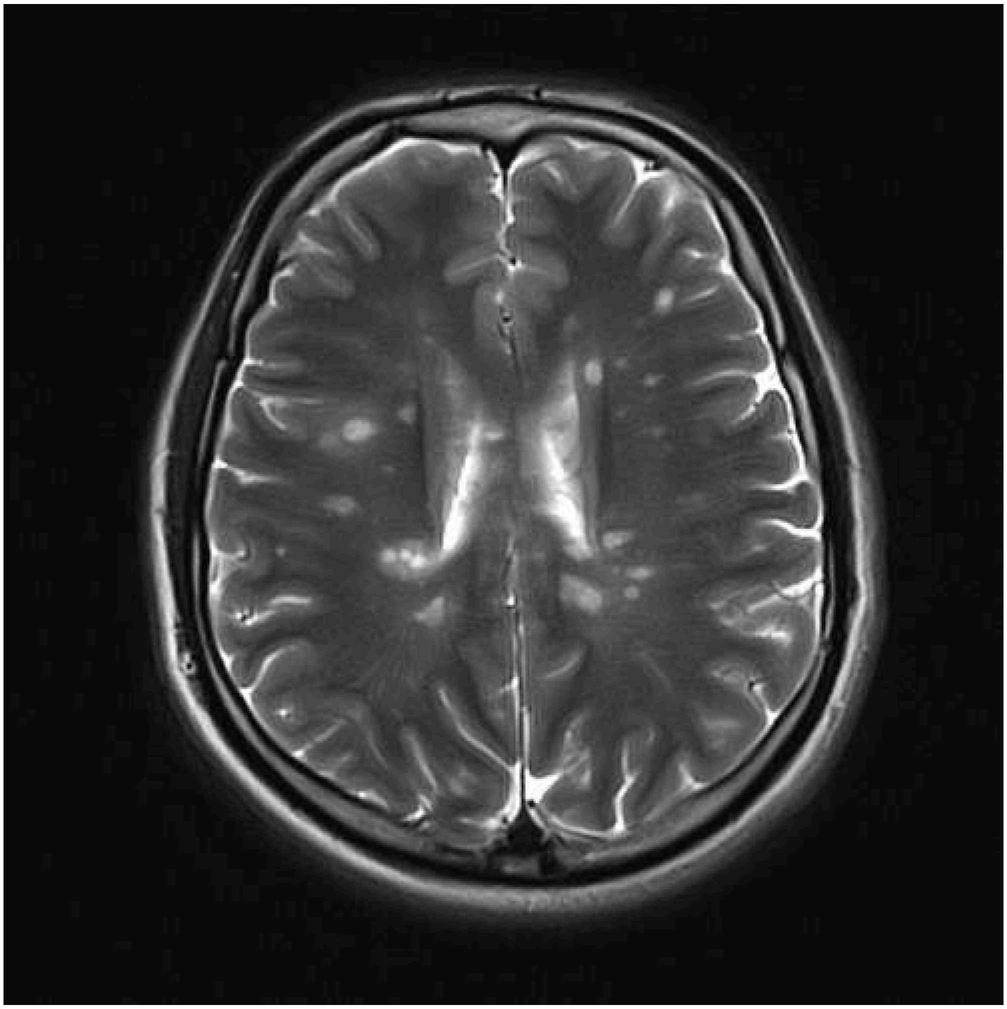

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What is the most likely diagnosis?

Creutzfeldt-Jakob disease	9%
Multiple sclerosis	64%
Adrenoleucodystrophy	14%
Cerebral toxoplasmosis	10%
Gliolastoma multiforme	3%

The MRI scan is consistent with multiple sclerosis. Widespread periventricular, juxtacortical, post fossa and upper cervical cord high T2 regions are noted. Note the difference in the lesions with varying degrees of contrast enhancement and restricted diffusion indicating active/recent demyelination. This satisfies the diagnostic criteria in terms of separation in terms of time space.

👍 👎 Discuss (9) Improve

Next question ›

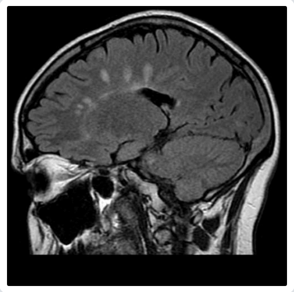
Multiple sclerosis: investigation ★

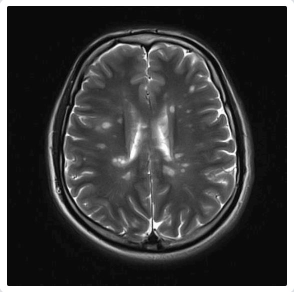
Diagnosis requires demonstration of lesions disseminated in time and space

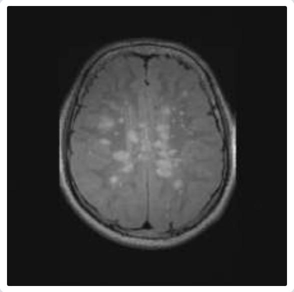
MRI
- high signal T2 lesions
- periventricular plaques
- Dawson fingers: often seen on FLAIR images - hyperintense lesions penpendicular to the corpus callosum

CSF
- oligoclonal bands (and not in serum)
- increased intrathecal synthesis of IgG

Visual evoked potentials
- delayed, but well preserved waveform







123

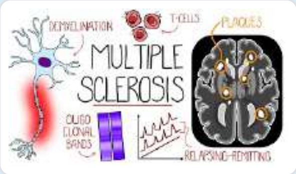
Next question ›

Textbooks

High-yield textbook

Extended textbook

Media


DEMYELINATION
T-CELLS
PLAQUES
MULTIPLE SCLEROSIS
OLIGO CLONAL BANDS
RELAPSING-REMITTING

Multiple sclerosis

Rhesus Medicine - YouTube 👍 1 👎 0

MULTIPLE SCLEROSIS

Multiple sclerosis - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube

👍 6 👎 1

Multiple Sclerosis

Multiple sclerosis

Soton Brain Hub - YouTube 👍 2 👎 1

Multiple sclerosis

Podmedics - YouTube 👍 1 👎 3

Report broken media

Score: **20.6%**

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Back to top

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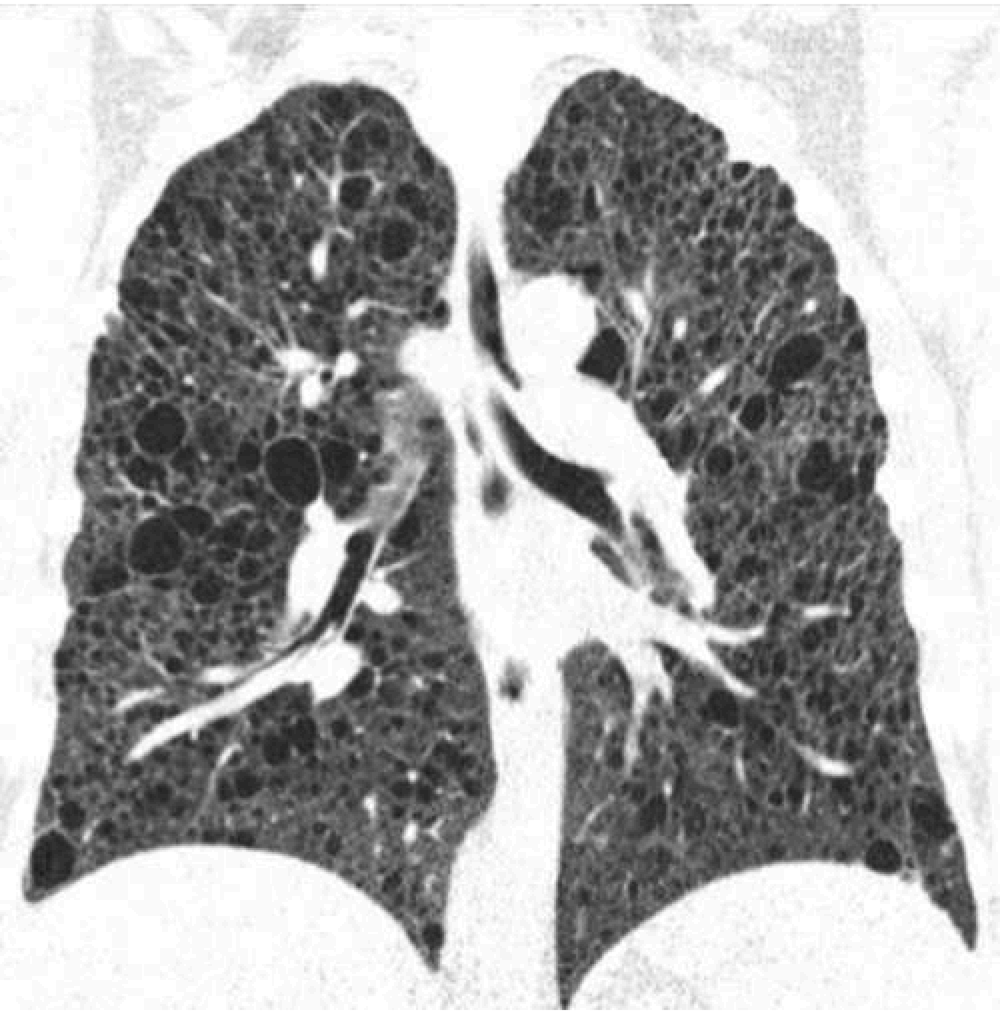
The CT scan is shown below:



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What complication has developed?

○ Metastatic angiomyolipoma

○ Lymphangioleiomyomatosis

○ Lung rhabdomyomas

○ Lung angiofibromas ×

○ Subependymal giant cell astrocytoma ×

Submit answer

Reference ranges

Score: **20%**

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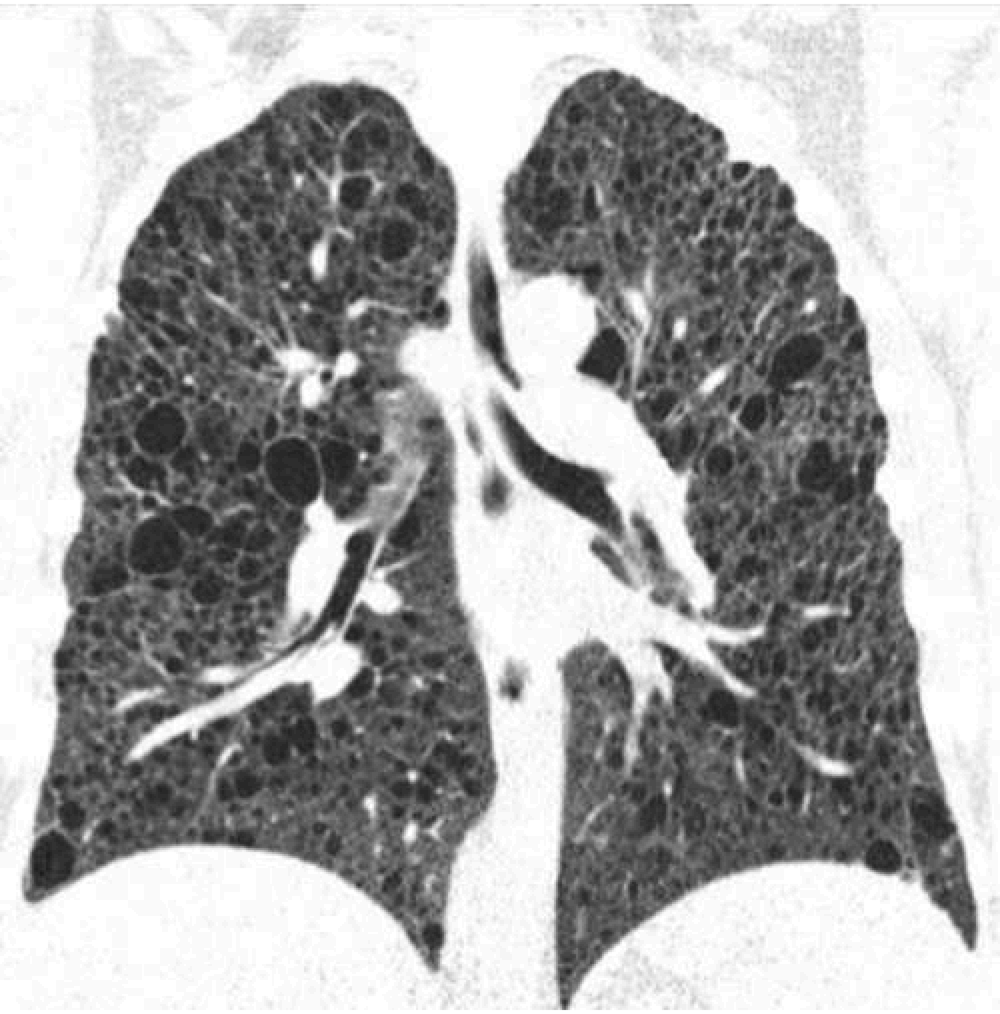

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What complication has developed?

Metastatic angiomyolipoma	4%
Lymphangioleiomyomatosis	63%
Lung rhabdomyomas	5%

Lung angiofibromas 25%

Subependymal giant cell astrocytoma 3%

The CT demonstrate innumerable small regular lung cysts diffusely distributed throughout the lungs, the typical appearance of lymphangioleiomyomatosis (LAM). This disorder may occur alone on in association with tuberous sclerosis.

👍 👎 Discuss (4) Improve

Next question >

Tuberous sclerosis ★

Tuberous sclerosis (TS) is a genetic condition of autosomal dominant inheritance. Like neurofibromatosis, the majority of features seen in TS are neurocutaneous.

Cutaneous features

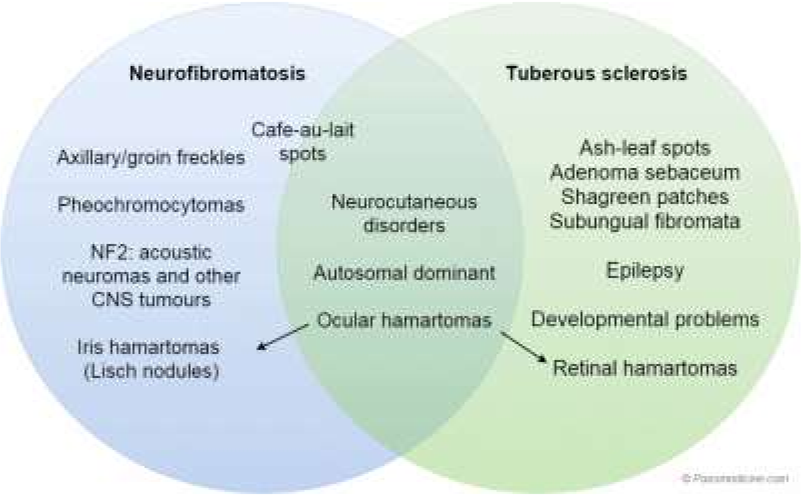
- depigmented 'ash-leaf' spots which fluoresce under UV light
- roughened patches of skin over lumbar spine (Shagreen patches)
- adenoma sebaceum (angiofibromas): butterfly distribution over nose
- fibromata beneath nails (subungual fibromata)
- cafe-au-lait spots* may be seen

Neurological features

- developmental delay
- epilepsy (infantile spasms or partial)
- intellectual impairment

Also

- retinal hamartomas: dense white areas on retina (phakomata)
- rhabdomyomas of the heart
- gliomatous changes can occur in the brain lesions
- polycystic kidneys, renal angiomyolipomata
- lymphangioleiomyomatosis: multiple lung cysts


Neurofibromatosis
Tuberous sclerosis
Axillary/groin freckles
Cafe-au-lait spots
Pheochromocytomas
NF2: acoustic neuromas and other CNS tumours
Iris hamartomas (Lisch nodules)
Neurocutaneous disorders
Autosomal dominant
Ocular hamartomas
Ash-leaf spots
Adenoma sebaceum
Shagreen patches
Subungual fibromata
Epilepsy
Developmental problems
Retinal hamartomas
© Passmedicine.com

Comparison of neurofibromatosis and tuberous sclerosis. Note that whilst they are both autosomal dominant neurocutaneous disorders there is little overlap otherwise

*these of course are more commonly associated with neurofibromatosis. However a 1998 study of 106 children with TS found cafe-au-lait spots in 28% of patients

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 6 12

Tuberous sclerosis review

Suggest link Report broken link

Media

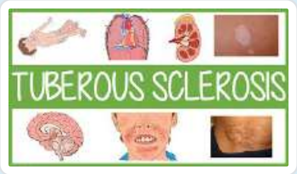
TUBEROUS SCLEROSIS
Osmosis.org

Tuberous sclerosis

Osmosis - YouTube 👍 11 👎 0

Angiomyolipoma

Osmosis - YouTube 👍 3 👎 0


TUBEROUS SCLEROSIS

Tuberous Sclerosis Complex

Townsend Teaching - YouTube 👍 3 👎 0

Tuberous
Sclerosis

Tuberous Sclerosis Complex (TSC)

Pixorize - YouTube 👍 2 👎 2

Report broken media

Score: **20.6%**

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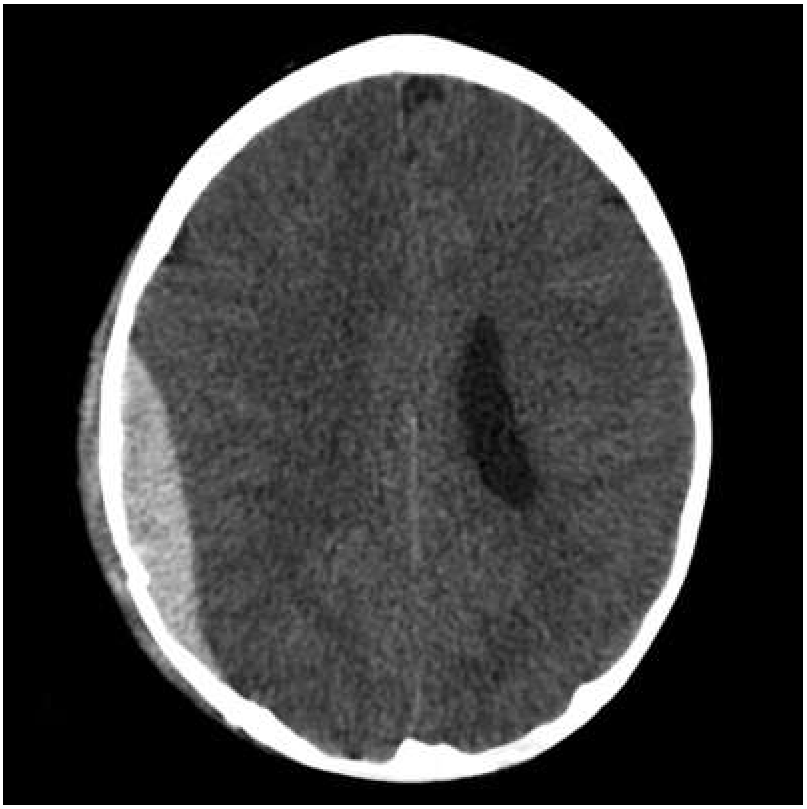
Back to top

A 7-year-old boy falls from the top of a slide and is taken to the Emergency Department. He is crying, complaining of a severe headache and regularly vomiting. A CT head is performed:



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What does the scan show?

○ Subarachnoid haemorrhage

○ Brain contusion

○ Extradural haemorrhage

○ Subdural haematoma

○ Meningioma

Submit answer

Reference ranges ﹀

Score: **20%**

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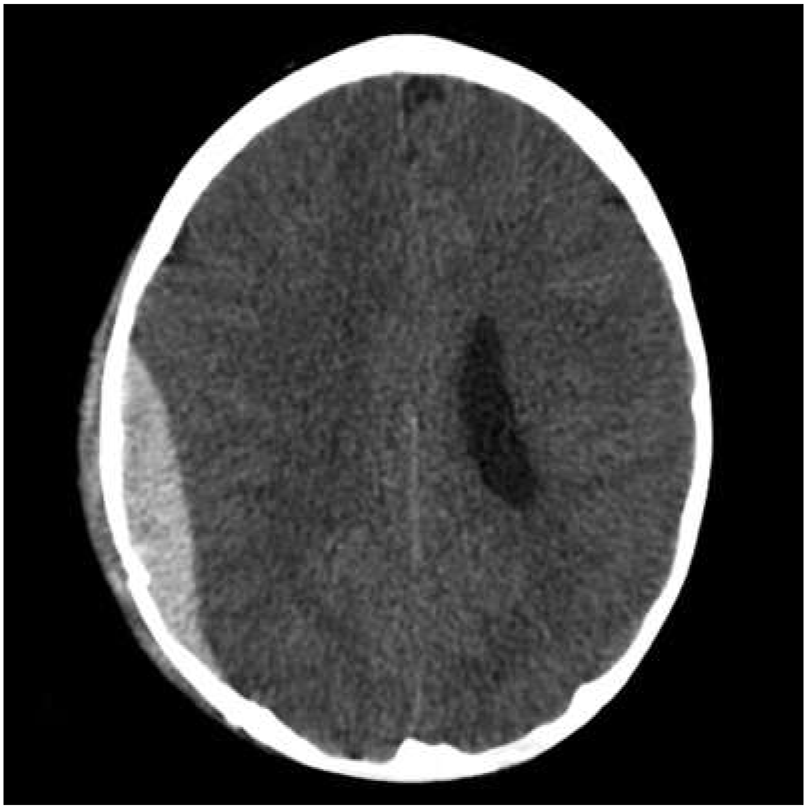

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What does the scan show?

Subarachnoid haemorrhage	1%
Brain contusion	1%
Extradural haematoma	80%
Subdural haematoma	19%
Meningioma	0%

The CT scan demonstrates a bi-convex (lentiform) extra-axial hyperdense collection consistent with an extradural haematoma.

👍 👎 Discuss (3) Improve

Next question >

Head injury: types of traumatic brain injury ★

Basics

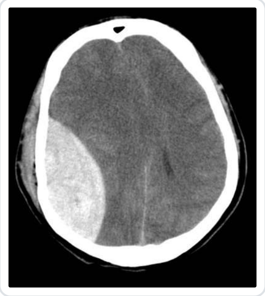
- primary brain injury may be focal (contusion/haematoma) or diffuse (diffuse axonal injury)
- diffuse axonal injury occurs as a result of mechanical shearing following deceleration, causing disruption and tearing of axons
- intra-cranial haematomas can be extradural, subdural or intracerebral, while contusions may occur adjacent to (coup) or contralateral (contre-coup) to the side of impact
- secondary brain injury occurs when cerebral oedema, ischaemia, infection, tonsillar or tentorial herniation exacerbates the original injury. The normal cerebral auto regulatory processes are disrupted following trauma rendering the brain more susceptible to blood flow changes and hypoxia
- the Cushings reflex (hypertension and bradycardia) often occurs late and is usually a pre terminal event

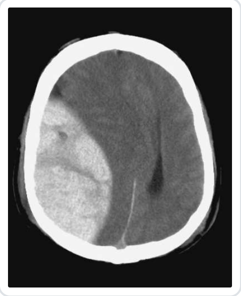
Type of injury	**Notes**
Extradural (epidural) haematoma	Bleeding into the space between the dura mater and the skull. Often results from acceleration-deceleration trauma or a blow to the side of the head. The majority of epidural haematomas occur in the temporal region where skull fractures cause a rupture of the middle meningeal artery. Features • features of raised intracranial pressure • some patients may exhibit a lucid interval
Subdural haematoma	Bleeding into the outermost meningeal layer. Most commonly occur around the frontal and parietal lobes. Risk factors include old age, alcoholism and anticoagulation. Slower onset of symptoms than a epidural haematoma. There may be fluctuating confusion/consciousness
Subarachnoid haemorrhage	Classically causes a sudden occipital headache. Usually occurs spontaneously in the context of a ruptured cerebral aneurysm but may be

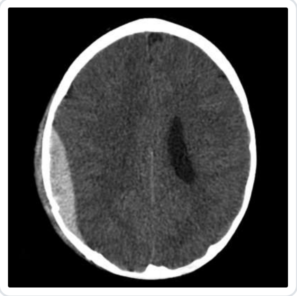
Type of injury	Notes
	seen in association with other injuries when a patient has sustained a traumatic brain injury
Intracerebral haematoma	An intracerebral (or intraparenchymal) haemorrhage is a collection of blood within the substance of the brain. Causes / risk factors include: hypertension, vascular lesion (e.g. aneurysm or arteriovenous malformation), cerebral amyloid angiopathy, trauma, brain tumour or infarct (particularly in stroke patients undergoing thrombolysis). Patients will present similarly to an ischaemic stroke (which is why it is crucial to obtain a CT in head in all stroke patients prior to thrombolysis) or with a decrease in consciousness. CT imaging will show a hyperdensity (bright lesion) within the substance of the brain. Treatment is often conservative under the care of stroke physicians, but large clots in patients with impaired consciousness may warrant surgical evacuation.

Image gallery

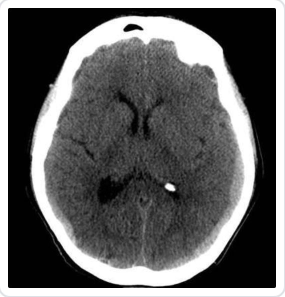
Extradural (epidural) haematoma:

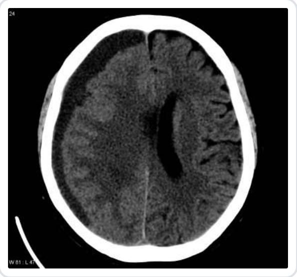


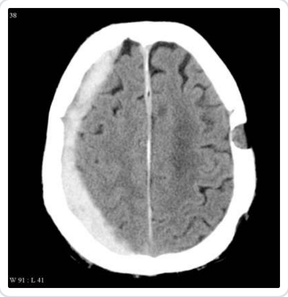




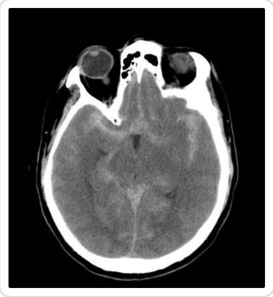
Subdural haematoma:

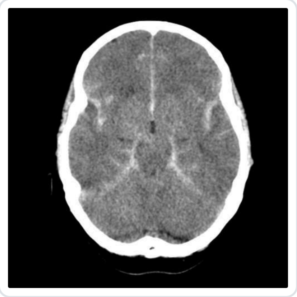






Subarachnoid haemorrhage:





123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

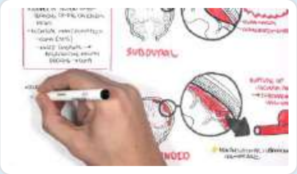
NICE 11 2

2014 Head injury guidelines

Suggest link

Report broken link

Media



Intracranial Haemorrhage Types

Armando Hasudungan - YouTube 👍 6 👎 0

Intracranial
Bleeds
subdural
&
extradural

Intracranial bleeds - Subdural and Extradural haematoma

Soton Brain Hub - YouTube 👍 4 👎 0

TRAUMATIC
BRAIN INJURY
~ PATHOLOGY REVIEW ~
Osmosis.org

Traumatic brain injury: pathology review

Osmosis - YouTube 👍 4 👎 1

Report broken media

Score: **20.6%**

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A 62-year-old woman comes to the gastroenterology clinic for review. She has a history of watery diarrhoea over the course of the past 6 months, where she is opening her bowels some 4-6 times per day. There is other past medical history of hypertension, ischaemic heart disease, Type 2 diabetes and depression.

Investigations

Hb	11.5 g/l	Na^+	139 mmol/l	Bilirubin	12 µmol/l
Platelets	$207 * 10^9/l$	K^+	3.9 mmol/l	ALP	95 u/l
WBC	$8.9 * 10^9/l$	Urea	7.2 mmol/l	ALT	23 u/l
Neuts	$5.6 * 10^9/l$	Creatinine	100 µmol/l	γGT	56 u/l
Lymphs	$1.8 * 10^9/l$			Albumin	38 g/l
Eosin	$0.5 * 10^9/l$				

Colonoscopy: Mild mucosal oedema only, biopsy reveals lymphocytic infiltration

Which of the following agents is most likely to be the cause of her colonoscopy findings?

○ Amlodipine

○ Atorvastatin

○ Lisinopril

○ Metformin

○ Sertraline

Submit answer

Reference ranges

Score: **20%**

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Colonoscopy: Mild mucosal oedema only, biopsy reveals lymphocytic infiltration

Which of the following agents is most likely to be the cause of her colonoscopy findings?

Amlodipine	4%
Atorvastatin	4%
Lisinopril	6%
Metformin	20%
Sertraline	66%

Sertraline is recognised as highly likely to be associated with lymphocytic colitis, (microscopic colitis associated with lymphocytic infiltration). This conclusion results from a mix of association / epidemiology studies, and case series where removal and re-introduction of medication has been correlated with symptoms and changes in biopsy appearance.

Ref: Munch A, Aust D, Bohr J, et al. Microscopic colitis: Current status, present and future challenges: statements of the European Microscopic Colitis Group. J Crohns Colitis 2012; 6:932

Calcium channel antagonists are not thought to be associated with microscopic colitis, but are uncommonly reported as leading to change in bowel habit including diarrhoea. Metformin leads to bile salt malabsorption which may result in diarrhoea. Statins may be associated with microscopic colitis, although withdrawal and re-challenge evidence is less strong than for sertraline. ACE inhibitors are not known to be associated with microscopic colitis. Diarrhoea was a commonly reported adverse event in lisinopril clinical trials, but this is not thought to be due to microscopic colitis.

Discuss (3) Improve

Next question ›

Selective serotonin reuptake inhibitors

Selective serotonin reuptake inhibitors (SSRIs) are considered first-line treatment for the majority of patients with depression.

- citalopram (although see below re: QT interval) and fluoxetine are currently the preferred SSRIs
- sertraline is useful post myocardial infarction as there is more evidence for its safe use in this situation than other antidepressants
- SSRIs should be used with caution in children and adolescents. Fluoxetine is the drug of choice when an antidepressant is indicated

Adverse effects

- gastrointestinal symptoms are the most common side-effect
- there is an increased risk of gastrointestinal bleeding in patients taking SSRIs. A proton pump inhibitor should be prescribed if a patient is also taking a NSAID
- patients should be counselled to be vigilant for increased anxiety and agitation after starting a SSRI
- fluoxetine and paroxetine have a higher propensity for drug interactions

Citalopram and the QT interval

- the Medicines and Healthcare products Regulatory Agency (MHRA) released a warning on the use of citalopram in 2011
- it advised that citalopram and escitalopram are associated with dose-dependent QT interval prolongation and should not be used in those with: congenital long QT syndrome; known pre-existing QT interval prolongation; or in combination with other medicines that prolong the QT interval

- the maximum daily dose is now 40 mg for adults; 20 mg for patients older than 65 years; and 20 mg for those with hepatic impairment

Interactions
- NSAIDs: NICE guidelines advise 'do not normally offer SSRIs', but if given co-prescribe a proton pump inhibitor
- warfarin / heparin: NICE guidelines recommend avoiding SSRIs and considering mirtazapine
- aspirin
- triptans - increased risk of serotonin syndrome
- monoamine oxidase inhibitors (MAOIs) - increased risk of serotonin syndrome

Following the initiation of antidepressant therapy patients should normally be reviewed by a doctor after 2 weeks. For patients under the age of 25 years or at increased risk of suicide should be reviewed after 1 week. If a patient makes a good response to antidepressant therapy they should continue on treatment for at least 6 months after remission as this reduces the risk of relapse.

When stopping a SSRI the dose should be gradually reduced over a 4 week period (this is not necessary with fluoxetine). Paroxetine has a higher incidence of discontinuation symptoms.

Discontinuation symptoms
- increased mood change
- restlessness
- difficulty sleeping
- unsteadiness
- sweating
- gastrointestinal symptoms: pain, cramping, diarrhoea, vomiting
- paraesthesia

SSRIs and pregnancy
- BNF says to weigh up benefits and risk when deciding whether to use in pregnancy.
- Use during the first trimester gives a small increased risk of congenital heart defects
- Use during the third trimester can result in persistent pulmonary hypertension of the newborn
- Paroxetine has an increased risk of congenital malformations, particularly in the first trimester

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 29 👎 43

2022 Depression in adults: treatment and management

Suggest link

Report broken link

Media

Antidepressants

Speed Pharmacology - YouTube 👍 6 👎 0

ANTIDEPRESSANTS
PHARMACOLOGY

Antidepressants Pharmacology

Soton Brain Hub - YouTube 👍 0 👎 0

Report broken media

Score: **20.6%**

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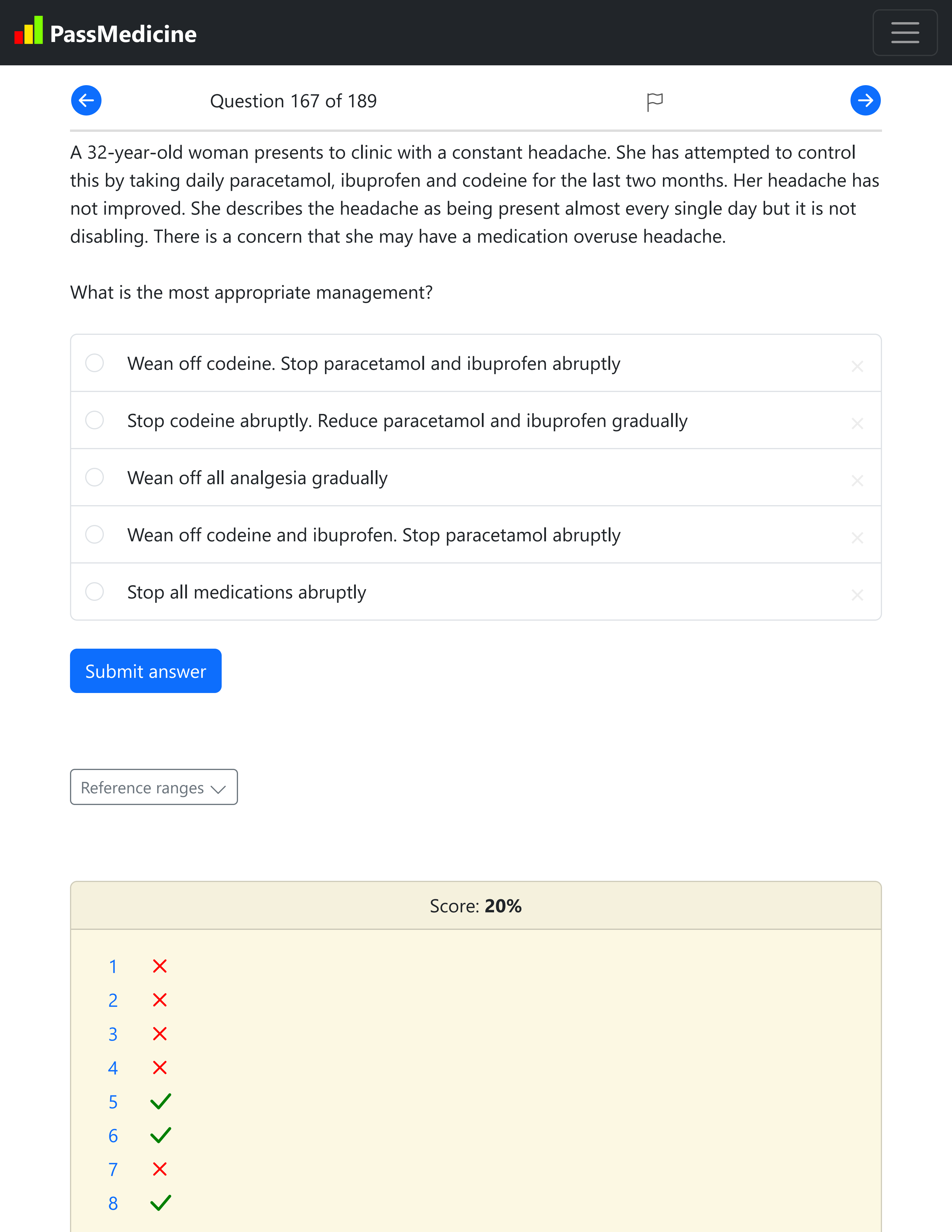

Question 167 of 189

A 32-year-old woman presents to clinic with a constant headache. She has attempted to control this by taking daily paracetamol, ibuprofen and codeine for the last two months. Her headache has not improved. She describes the headache as being present almost every single day but it is not disabling. There is a concern that she may have a medication overuse headache.

What is the most appropriate management?

- ○ Wean off codeine. Stop paracetamol and ibuprofen abruptly
- ○ Stop codeine abruptly. Reduce paracetamol and ibuprofen gradually
- ○ Wean off all analgesia gradually
- ○ Wean off codeine and ibuprofen. Stop paracetamol abruptly
- ○ Stop all medications abruptly

Score: **20%**

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Question 167 of 189 ✗

A 32-year-old woman presents to clinic with a constant headache. She has attempted to control this by taking daily paracetamol, ibuprofen and codeine for the last two months. Her headache has not improved. She describes the headache as being present almost every single day but it is not disabling. There is a concern that she may have a medication overuse headache.

What is the most appropriate management?

Wean off codeine. Stop paracetamol and ibuprofen abruptly	78%
Stop codeine abruptly. Reduce paracetamol and ibuprofen gradually	3%
Wean off all analgesia gradually	6%
Wean off codeine and ibuprofen. Stop paracetamol abruptly	3%
Stop all medications abruptly	10%

The duration of the headache and regular use of codeine makes the diagnosis of medication overuse headache most likely.

The correct answer is to stop paracetamol and ibuprofen abruptly, but gradually wean off codeine due to the adverse effects of acute opioid withdrawal. Whilst codeine and triptans are the most common cause, other types of analgesia can contribute. Improvement is often seen within a few days.

Discuss (2) | Improve

Next question >

Medication overuse headache ★

Medication overuse headache is one of the most common causes of chronic daily headache. It may affect up to 1 in 50 people

Features

- present for 15 days or more per month
- developed or worsened whilst taking regular symptomatic medication
- patients using opioids and triptans are at most risk
- may be psychiatric co-morbidity

Management (from 2008 SIGN guidelines)

- simple analgesics and triptans should be withdrawn abruptly (may initially worsen headaches)
- opioid analgesics should be gradually withdrawn

Withdrawal symptoms such as vomiting, hypotension, tachycardia, restlessness, sleep disturbances and anxiety may occur when medication is stopped

+ Q 123

Next question ›

B *I* A

Textbooks

High-yield textbook

Extended textbook

Score: **20.6%**

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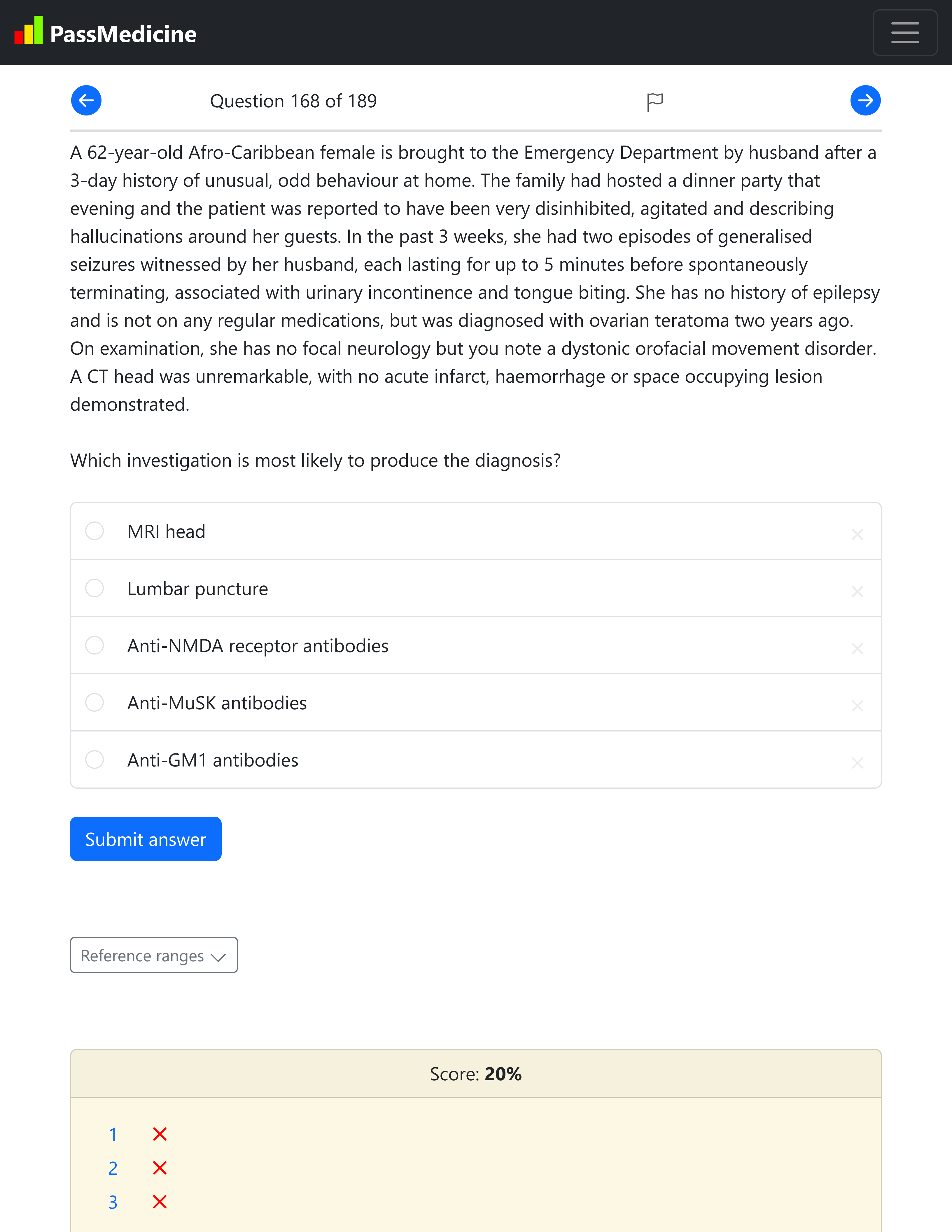

Question 168 of 189

A 62-year-old Afro-Caribbean female is brought to the Emergency Department by husband after a 3-day history of unusual, odd behaviour at home. The family had hosted a dinner party that evening and the patient was reported to have been very disinhibited, agitated and describing hallucinations around her guests. In the past 3 weeks, she had two episodes of generalised seizures witnessed by her husband, each lasting for up to 5 minutes before spontaneously terminating, associated with urinary incontinence and tongue biting. She has no history of epilepsy and is not on any regular medications, but was diagnosed with ovarian teratoma two years ago. On examination, she has no focal neurology but you note a dystonic orofacial movement disorder. A CT head was unremarkable, with no acute infarct, haemorrhage or space occupying lesion demonstrated.

Which investigation is most likely to produce the diagnosis?

- ○ MRI head
- ○ Lumbar puncture
- ○ Anti-NMDA receptor antibodies
- ○ Anti-MuSK antibodies
- ○ Anti-GM1 antibodies

Score: **20%**

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Question 168 of 189 ✗

A 62-year-old Afro-Caribbean female is brought to the Emergency Department by husband after a 3-day history of unusual, odd behaviour at home. The family had hosted a dinner party that evening and the patient was reported to have been very disinhibited, agitated and describing hallucinations around her guests. In the past 3 weeks, she had two episodes of generalised seizures witnessed by her husband, each lasting for up to 5 minutes before spontaneously terminating, associated with urinary incontinence and tongue biting. She has no history of epilepsy and is not on any regular medications, but was diagnosed with ovarian teratoma two years ago. On examination, she has no focal neurology but you note a dystonic orofacial movement disorder. A CT head was unremarkable, with no acute infarct, haemorrhage or space occupying lesion demonstrated.

Which investigation is most likely to produce the diagnosis?

MRI head	8%
Lumbar puncture	4%
Anti-NMDA receptor antibodies	80%
Anti-MuSK antibodies	5%
Anti-GM1 antibodies	4%

The syndrome described is strongly suggestive of an autoimmune-mediated limbic encephalitis.

Discuss (3) Improve

Next question ›

Anti-NMDA receptor encephalitis

Anti-NMDA receptor encephalitis is a paraneoplastic syndrome, presenting as prominent psychiatric features including agitation, hallucinations, delusions and disordered thinking; seizures, insomnia, dyskinesias and autonomic instability. Ovarian teratomas are detected in up to half of all female adult patients, particularly prevalent in Afro-Caribbean patients. MRI head can be normal but abnormalities can be visualised on FLAIR sequences in the deep subcortical limbic structures. CSF may demonstrate pleocytosis but can be normal initially. Anti-MuSK is an autoantibody specific to muscle kinase in myasthenia gravis with no evidence of a thymoma and without antibodies to acetylcholine receptors. Anti-GM1 is an autoantibody specific to acute inflammatory demyelinating polyneuropathy (AIDP) variant of Guillain-Barre syndrome.

Treatment of anti-NMDA encephalitis is based of immunosuppression with intravenous steroids, immunoglobulins, rituximab, cyclophosphamide or plasma exchange, alone or in combination. Resection of teratoma is also therapeutic.

AI + Q 123

Next question >

B *I*

Textbooks

High-yield textbook

Extended textbook

Score: **20.6%**

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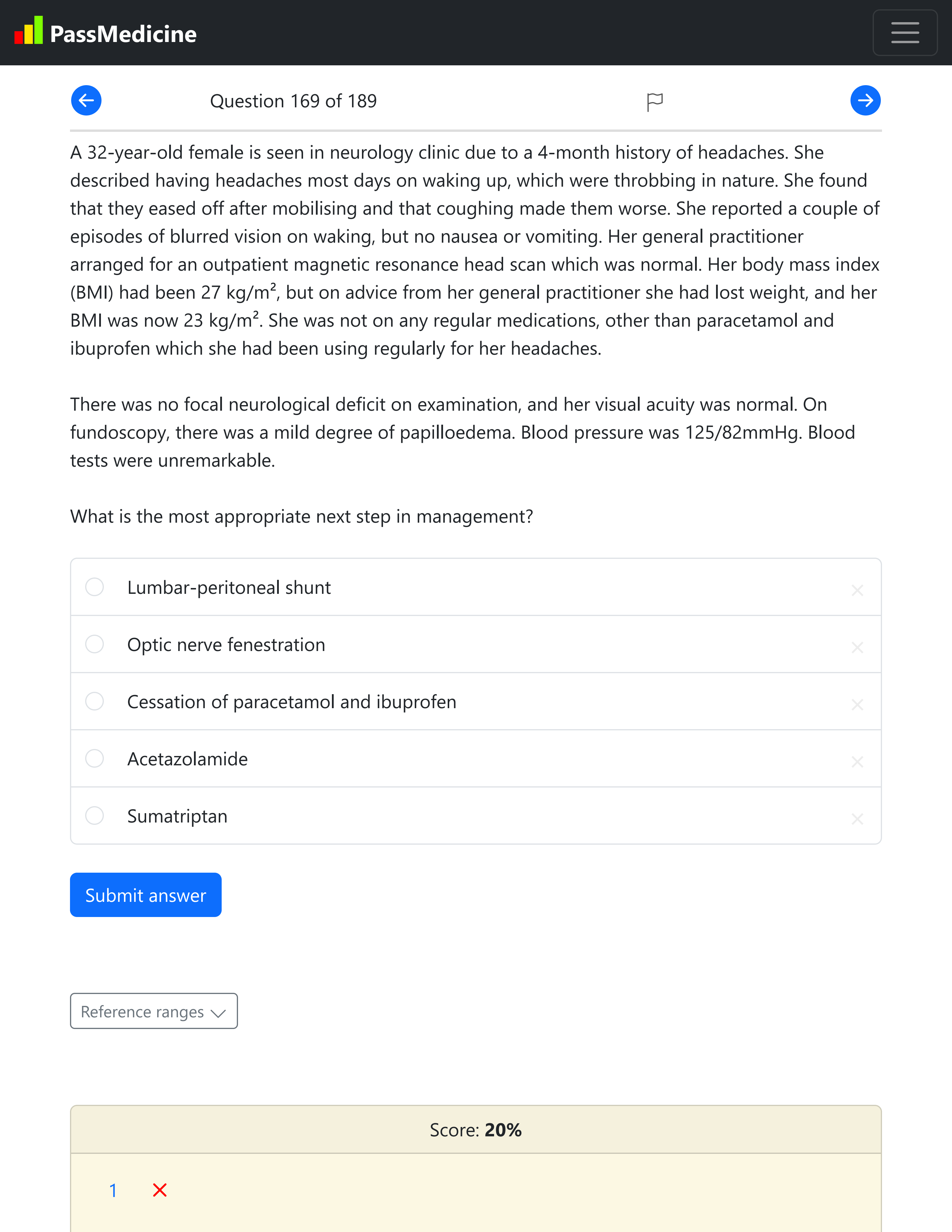

Question 169 of 189

A 32-year-old female is seen in neurology clinic due to a 4-month history of headaches. She described having headaches most days on waking up, which were throbbing in nature. She found that they eased off after mobilising and that coughing made them worse. She reported a couple of episodes of blurred vision on waking, but no nausea or vomiting. Her general practitioner arranged for an outpatient magnetic resonance head scan which was normal. Her body mass index (BMI) had been 27 kg/m^2, but on advice from her general practitioner she had lost weight, and her BMI was now 23 kg/m^2. She was not on any regular medications, other than paracetamol and ibuprofen which she had been using regularly for her headaches.

There was no focal neurological deficit on examination, and her visual acuity was normal. On fundoscopy, there was a mild degree of papilloedema. Blood pressure was 125/82mmHg. Blood tests were unremarkable.

What is the most appropriate next step in management?

○ Lumbar-peritoneal shunt ×

○ Optic nerve fenestration ×

○ Cessation of paracetamol and ibuprofen ×

○ Acetazolamide ×

○ Sumatriptan ×

Score: **20%**

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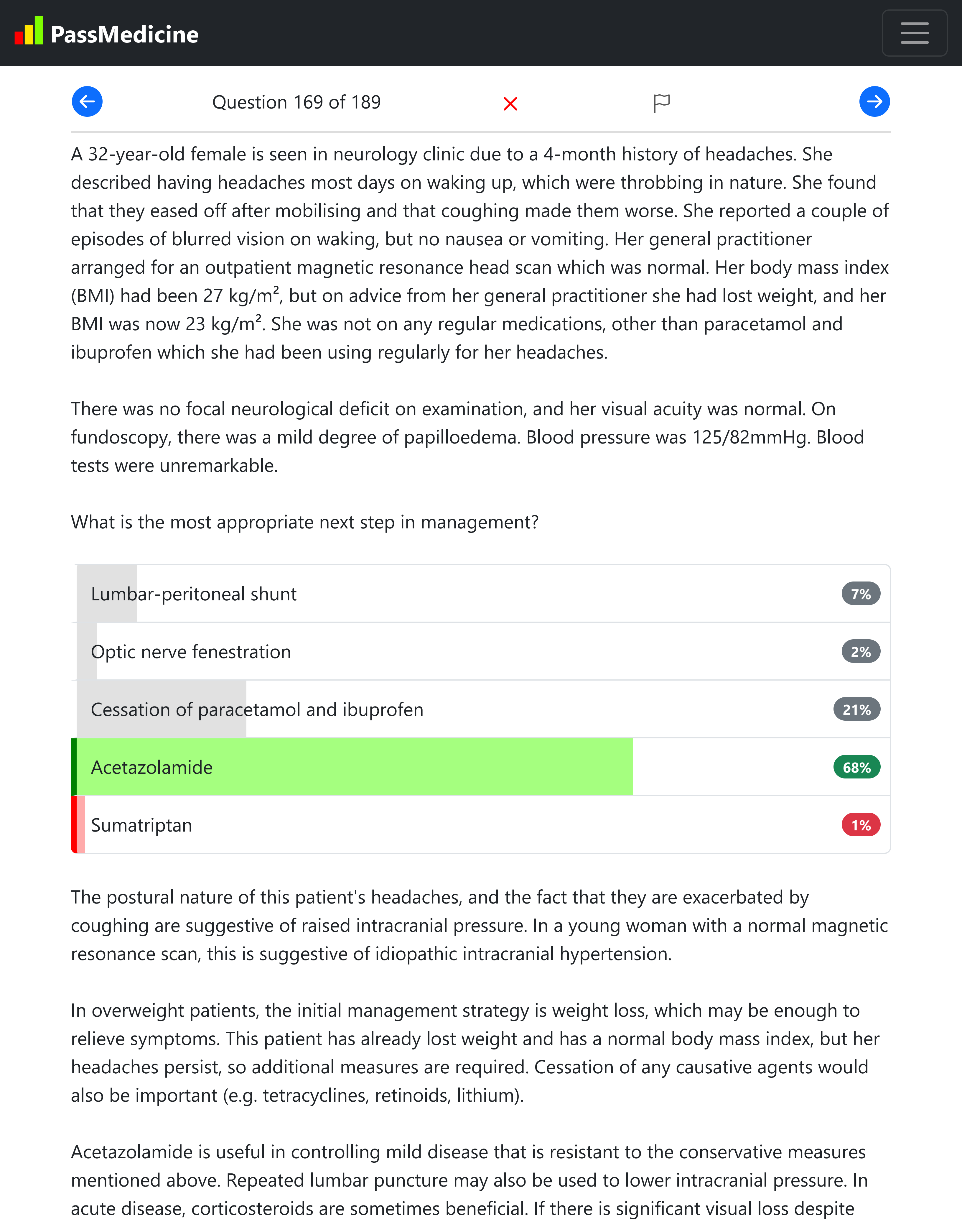

A 32-year-old female is seen in neurology clinic due to a 4-month history of headaches. She described having headaches most days on waking up, which were throbbing in nature. She found that they eased off after mobilising and that coughing made them worse. She reported a couple of episodes of blurred vision on waking, but no nausea or vomiting. Her general practitioner arranged for an outpatient magnetic resonance head scan which was normal. Her body mass index (BMI) had been 27 kg/m², but on advice from her general practitioner she had lost weight, and her BMI was now 23 kg/m². She was not on any regular medications, other than paracetamol and ibuprofen which she had been using regularly for her headaches.

There was no focal neurological deficit on examination, and her visual acuity was normal. On fundoscopy, there was a mild degree of papilloedema. Blood pressure was 125/82mmHg. Blood tests were unremarkable.

What is the most appropriate next step in management?

Lumbar-peritoneal shunt	7%
Optic nerve fenestration	2%
Cessation of paracetamol and ibuprofen	21%
Acetazolamide	68%
Sumatriptan	1%

The postural nature of this patient's headaches, and the fact that they are exacerbated by coughing are suggestive of raised intracranial pressure. In a young woman with a normal magnetic resonance scan, this is suggestive of idiopathic intracranial hypertension.

In overweight patients, the initial management strategy is weight loss, which may be enough to relieve symptoms. This patient has already lost weight and has a normal body mass index, but her headaches persist, so additional measures are required. Cessation of any causative agents would also be important (e.g. tetracyclines, retinoids, lithium).

Acetazolamide is useful in controlling mild disease that is resistant to the conservative measures mentioned above. Repeated lumbar puncture may also be used to lower intracranial pressure. In acute disease, corticosteroids are sometimes beneficial. If there is significant visual loss despite medical therapy surgical interventions such as optic nerve sheath fenestration or lumbar-peritoneal shunt insertion may be necessary.

Analgesia-induced headache and migraine are important causes of recurrent headache, but would not cause the postural association seen in this case, so cessation of analgesic agents and commencement of sumatriptan are not appropriate treatments.

👍 👎 Discuss (4) Improve

Next question ›

Idiopathic intracranial hypertension

Idiopathic intracranial hypertension (also known as pseudotumour cerebri and formerly benign intracranial hypertension) is a condition classically seen in young, overweight females.

Risk factors
- obesity
- female sex
- pregnancy
- drugs
    - combined oral contraceptive pill
    - steroids
    - tetracyclines
    - retinoids (isotretinoin, tretinoin) / vitamin A
    - lithium

Features
- headache
- blurred vision
- papilloedema (usually present)
- enlarged blind spot
- sixth nerve palsy may be present

Management
- weight loss
    - whilst diet and exercise are important, medications such as semaglitide and topiramate may be considered by specialists. Topiramate is particularly beneficial as it also inhibits carbonic anhydrase
- carbonic anhydrase inhibitors e.g. acetazolamide

- topiramate is also used, and has the added benefit of causing weight loss in most patients
- repeated lumbar puncture may be used as a temporary measure but is not suitable for longer-term management
- surgery: optic nerve sheath decompression and fenestration may be needed to prevent damage to the optic nerve. A lumboperitoneal or ventriculoperitoneal shunt may also be performed to reduce intracranial pressure

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 12 13

Idiopathic intracranial hypertension review

Suggest link Report broken link

Score: **20.6%**

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Question 170 of 189

A 35 year old mechanic attends the emergency department following an injury at work. He has suffered a serious laceration to the upper arm. While suturing, the doctor notices multiple cuts and burns on both arms.

On examination there is marked wasting of brachioradialis and the small muscles in both hands, with mild hyporeflexia of the biceps and brachioradialis tendons. He is weak in both arms, distally more so. His lower limb and cranial nerve examination is unremarkable. On testing upper limb sensation, vibration and proprioception are intact but there appears to be reduced pain and temperature sensation over the C3/C4/C5 deramatomes. What is the most useful investigation?

○ Lumbar puncture ×

○ Nerve conduction studies ×

○ MRI Brain ×

○ Electromyography ×

○ MRI cervico-thoracic spine ×

Submit answer

Reference ranges

Score: **20%**

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On examination there is marked wasting of brachioradialis and the small muscles in both hands, with mild hyporeflexia of the biceps and brachioradialis tendons. He is weak in both arms, distally more so. His lower limb and cranial nerve examination is unremarkable. On testing upper limb sensation, vibration and proprioception are intact but there appears to be reduced pain and temperature sensation over the C3/C4/C5 deramatomes. What is the most useful investigation?

Lumbar puncture	0%
Nerve conduction studies	13%
MRI Brain	4%
Electromyography	4%
MRI cervico-thoracic spine	79%

The presence of loss of pain and temperature sensation in a 'cape like' distribution is highly suggestive of syringomyelia. A fluid filled cavity within the cord develops, typically between C2 to T9, compressing the cord from inside to out. Consequently the spinothalamic tracts are the first to be affected in a symmetrical distribution.

There are a number of aetiologies, but it should be noted that therw is an association with Arnold Chiari malformation Type 1. It is sensible to investigate for a compressive cord lesion in the first instance; imaging of the brain may be considered if syringomyelia is detected. An MRI cervice-thoracic cord would be the most sensitive method of detecting syringomyelia.

National Institute of neurological disease and stroke: syringomyelia fact sheet
http://www.ninds.nih.gov/disorders/syringomyelia/detailsyringomyelia.htm

Discuss (5) Improve

Next question ›

Syringomyelia ★

Syringomyelia ('syrinx' for short) describes a collection of cerebrospinal fluid within the spinal cord.

Syringobulbia is a similar phenomenon in which there is a fluid-filled cavity within the medulla of the brainstem. This is often an extension of the syringomyelia but in rare cases can be an isolated finding.

Causes include:
- a Chiari malformation: strong association
- trauma
- tumours
- idiopathic

Features
- a 'cape-like' (neck, shoulders and arms)
  - loss of sensation to temperature but the preservation of light touch, proprioception and vibration
  - classic examples are of patients who accidentally burn their hands without realising
  - this is due to the crossing spinothalamic tracts in the anterior commissure of the spinal cord being the first tracts to be affected
- spastic weakness (predominantly of the lower limbs)
- neuropathic pain
- upgoing plantars
- autonomic features:
- Horner's syndrome due to compression of the sympathetic chain, but this is rare
  - bowel and bladder dysfunction
- scoliosis will occur over a matter of years if the syrinx is not treated

Investigations
- full spine MRI with contrast to exclude a tumour or tethered cord
- a brain MRI is also needed to exclude a Chiari malformation

Treatment will be directed at treating the cause of the syrinx. In patients with a persistent or symptomatic syrinx, a shunt into the syrinx can be placed.

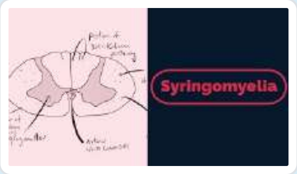
Next question >

Textbooks

High-yield textbook

Extended textbook

Media



Syringomyelia

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Report broken media

Score: **20.6%**

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Question 171 of 189

A 78-year-old man is brought to see you in clinic by his daughter. He has a diagnosis of Alzheimer's dementia and although currently coping well, has significantly impaired short-term memory.

Currently, his daughter attends daily for all meals, cleaning, and shopping. He continues to live in his own home and is alone overnight. She has, however, become concerned as he continues to drive 5 miles three times a week and she is unsure if this is safe. Which of the following is the best course of action regarding his driving licence?

- ○ He is disqualified from driving
- ○ He is allowed to drive, but only if daughter is present
- ○ There is no need to contact the DVLA, continue to drive
- ○ Assess his risk factors, report to the DVLA and await advice
- ○ He is safe if his mini mental state examination score >11

Submit answer

Reference ranges

Score: **20%**

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Question 171 of 189 ✗

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Currently, his daughter attends daily for all meals, cleaning, and shopping. He continues to live in his own home and is alone overnight. She has, however, become concerned as he continues to drive 5 miles three times a week and she is unsure if this is safe. Which of the following is the best course of action regarding his driving licence?

He is disqualified from driving	24%
He is allowed to drive, but only if daughter is present	5%
There is no need to contact the DVLA, continue to drive	8%
Assess his risk factors, report to the DVLA and await advice	57%
He is safe if his mini mental state examination score >11	6%

The DVLA guidance states this is a difficult decision to make. The presence of impaired short-term memory, disorientation or loss of insight suggests someone with dementia is not fit to drive.

In this case, the correct answer would be to contact the DVLA, assess the risk factors and take further advice. He should not be immediately disqualified; he warrants a more careful assessment and decision with support from the DVLA. The factors that suggest someone with dementia may not be safe to drive include significantly impaired short-term memory, poor attention and concentration, and impairment of planning.

DVLA guidance: Dementia or any organic brain syndrome.
https://www.gov.uk/current-medical-guidelines-dvla-guidance-for-professionals-conditions-d-to-f

Discuss (5) | Improve

Next question ›

DVLA: neurological disorders

The guidelines below relate to car/motorcycle use unless specifically stated. For obvious reasons, the rules relating to drivers of heavy goods vehicles tend to be much stricter

Epilepsy/seizures - all patient must not drive and must inform the DVLA

- first unprovoked/isolated seizure: 6 months off if there are no relevant structural abnormalities on brain imaging and no definite epileptiform activity on EEG. If these conditions are not met then this is increased to 12 months
- for patients with established epilepsy or those with multiple unprovoked seizures:
  - may qualify for a driving licence if they have been free from any seizure for 12 months
  - if there have been no seizures for 5 years (with medication if necessary) a 'til 70 licence is usually restored
- withdrawawl of epilepsy medication: should not drive whilst anti-epilepsy medication is being withdrawn and for 6 months after the last dose

Syncope

- simple faint: no restriction
- single episode, explained and treated: 4 weeks off
- single episode, unexplained: 6 months off
- two or more episodes: 12 months off

Other conditions

- stroke or TIA: 1 month off driving, may not need to inform DVLA if no residual neurological deficit
- multiple TIAs over short period of times: 3 months off driving and inform DVLA
- craniotomy e.g. For meningioma: 1 year off driving*
- pituitary tumour: craniotomy: 6 months; trans-sphenoidal surgery 'can drive when there is no debarring residual impairment likely to affect safe driving'
- narcolepsy/cataplexy: cease driving on diagnosis, can restart once 'satisfactory control of symptoms'
- chronic neurological disorders e.g. multiple sclerosis, motor neuron disease: DVLA should be informed, complete PK1 form (application for driving licence holders state of health)

*if the tumour is a benign meningioma and there is no seizure history, licence can be reconsidered 6 months after surgery if remains seizure free

Next question

Textbooks

High-yield textbook

Extended textbook

Links

DVLA 31 68

Fitness to drive guidelines

Suggest link Report broken link

Score: **20.6%**

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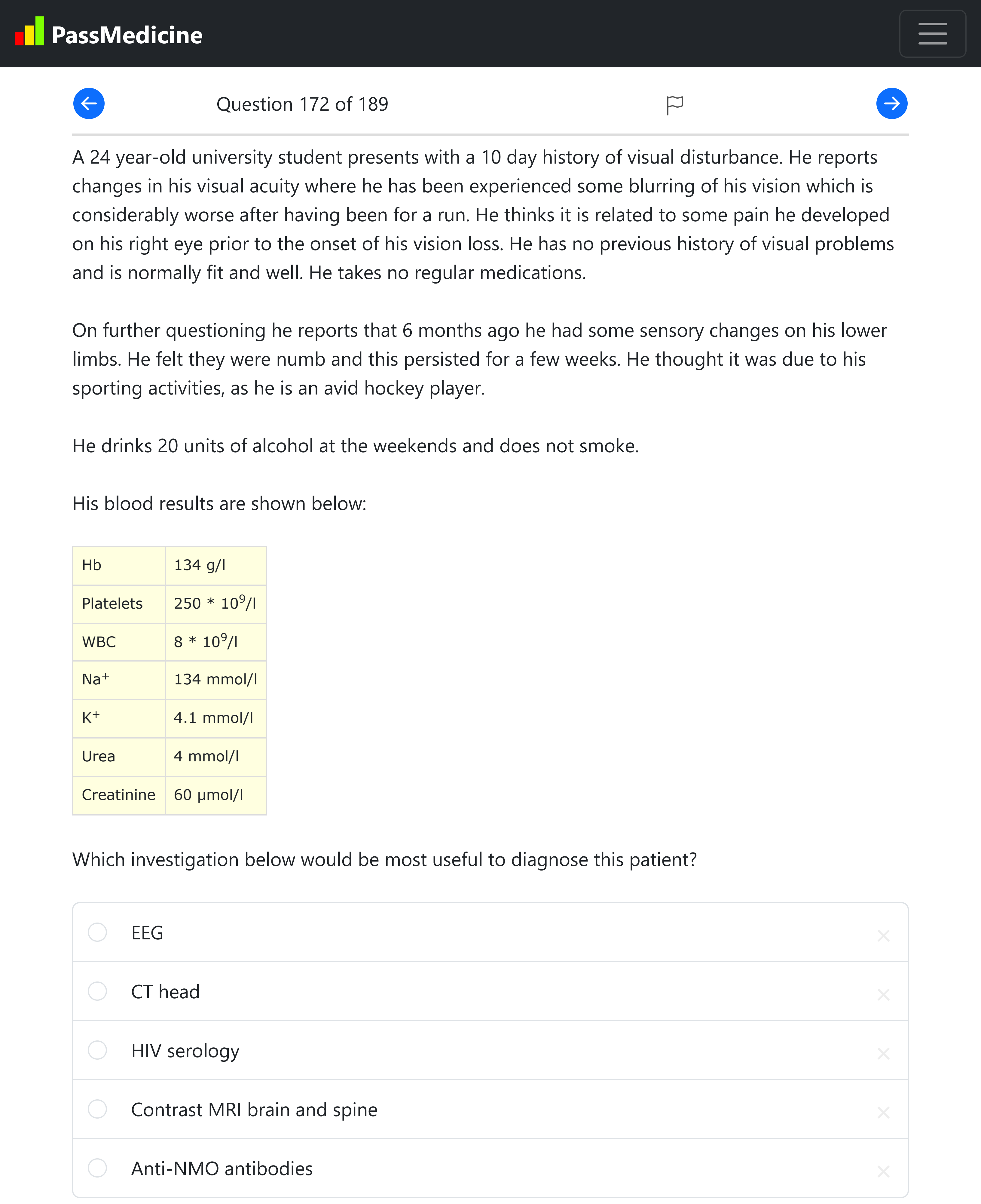

A 24 year-old university student presents with a 10 day history of visual disturbance. He reports changes in his visual acuity where he has been experienced some blurring of his vision which is considerably worse after having been for a run. He thinks it is related to some pain he developed on his right eye prior to the onset of his vision loss. He has no previous history of visual problems and is normally fit and well. He takes no regular medications.

On further questioning he reports that 6 months ago he had some sensory changes on his lower limbs. He felt they were numb and this persisted for a few weeks. He thought it was due to his sporting activities, as he is an avid hockey player.

He drinks 20 units of alcohol at the weekends and does not smoke.

His blood results are shown below:

Hb	134 g/l
Platelets	250 * 10^9/l
WBC	8 * 10^9/l
Na^+	134 mmol/l
K^+	4.1 mmol/l
Urea	4 mmol/l
Creatinine	60 µmol/l

Which investigation below would be most useful to diagnose this patient?

- ○ EEG
- ○ CT head
- ○ HIV serology
- ○ Contrast MRI brain and spine
- ○ Anti-NMO antibodies

Submit answer

Reference ranges

Score: **20%**

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Question 172 of 189 ✗

A 24 year-old university student presents with a 10 day history of visual disturbance. He reports changes in his visual acuity where he has been experienced some blurring of his vision which is considerably worse after having been for a run. He thinks it is related to some pain he developed on his right eye prior to the onset of his vision loss. He has no previous history of visual problems and is normally fit and well. He takes no regular medications.

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Na^+	134 mmol/l
K^+	4.1 mmol/l
Urea	4 mmol/l
Creatinine	60 µmol/l

Which investigation below would be most useful to diagnose this patient?

EEG	1%
CT head	1%
HIV serology	1%
Contrast MRI brain and spine	71%
Anti-NMO antibodies	26%

This young man presents with the classic symptoms of optic neuritis. Changes in vision(usually blurriness but can occasionally present as loss of red colour vision). He describes Uhthoff's phenomenon.

His history of a previous episode of paraesthesia is consistent with a previous first CIN episode. This gentleman requires urgent investigations. He should have a lumbar puncture which would show oligoclonal bands- which should be paired with a serum sample for comparison.

The correct answer is contrast MRI brain and spine and this will show a gadolinium enhancing consistent with an acute demyelinating lesion. Most likely it would also show an old lesion in his cord which would be diagnostic of MS.
http://emedicine.medscape.com/article/1146199-workup

Neuromyelitis optica is a demyelinating condition associated with NMO antibodies (Aquaporin 4 antibodies) which presents with demyelination of the optic nerve and spinal cord. It follows a more severe course than traditional MS. In this instance, an MRI with contrast will be the most appropriate investigation to yield a diagnosis.

👍 👎 Discuss (5) Improve

Next question ›

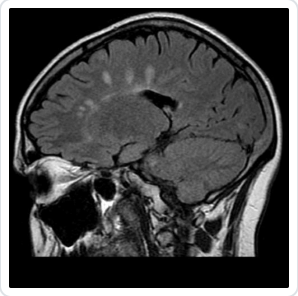
Multiple sclerosis: investigation ★

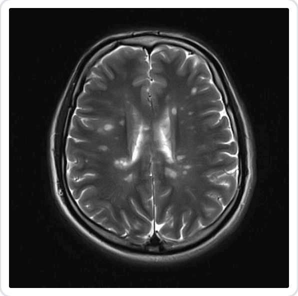
Diagnosis requires demonstration of lesions disseminated in time and space

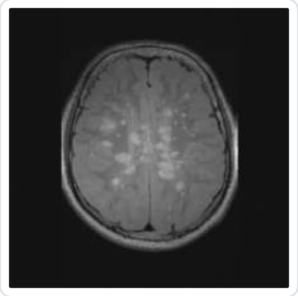
MRI
- high signal T2 lesions
- periventricular plaques
- Dawson fingers: often seen on FLAIR images - hyperintense lesions penpendicular to the corpus callosum

CSF
- oligoclonal bands (and not in serum)
- increased intrathecal synthesis of IgG

Visual evoked potentials
- delayed, but well preserved waveform







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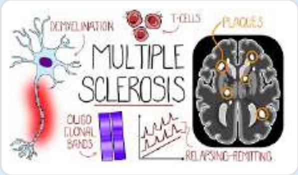
Next question >

Textbooks

High-yield textbook

Extended textbook

Media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Multiple sclerosis

Rhesus Medicine - YouTube 1 0

MULTIPLE SCLEROSIS

Multiple sclerosis - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube

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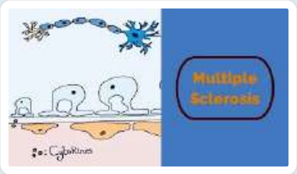
Multiple Sclerosis

Multiple sclerosis

Soton Brain Hub - YouTube 👍 2 👎 1

Multiple sclerosis

Podmedics - YouTube 👍 1 👎 3

Report broken media

Score: **20.6%**

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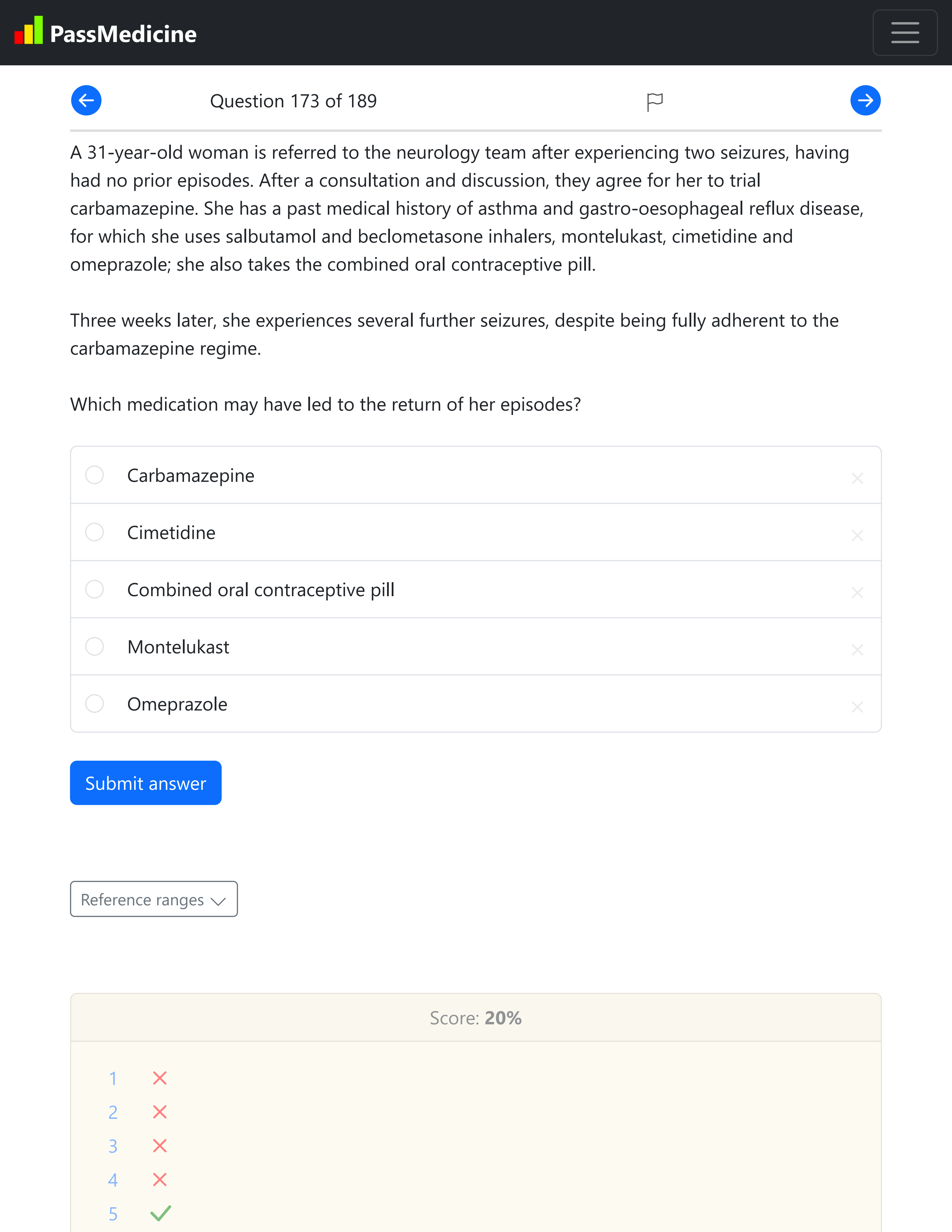

Question 173 of 189

A 31-year-old woman is referred to the neurology team after experiencing two seizures, having had no prior episodes. After a consultation and discussion, they agree for her to trial carbamazepine. She has a past medical history of asthma and gastro-oesophageal reflux disease, for which she uses salbutamol and beclometasone inhalers, montelukast, cimetidine and omeprazole; she also takes the combined oral contraceptive pill.

Three weeks later, she experiences several further seizures, despite being fully adherent to the carbamazepine regime.

Which medication may have led to the return of her episodes?

- ○ Carbamazepine ×
- ○ Cimetidine ×
- ○ Combined oral contraceptive pill ×
- ○ Montelukast ×
- ○ Omeprazole ×

Score: **20%**

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A 31-year-old woman is referred to the neurology team after experiencing two seizures, having had no prior episodes. After a consultation and discussion, they agree for her to trial carbamazepine. She has a past medical history of asthma and gastro-oesophageal reflux disease, for which she uses salbutamol and beclometasone inhalers, montelukast, cimetidine and omeprazole; she also takes the combined oral contraceptive pill.

Three weeks later, she experiences several further seizures, despite being fully adherent to the carbamazepine regime.

Which medication may have led to the return of her episodes?

Carbamazepine	47%
Cimetidine	17%
Combined oral contraceptive pill	22%
Montelukast	4%
Omeprazole	10%

Carbamazepine is known to exhibit autoinduction. hence when patients start carbamazepine they may see a return of seizures after 3-4 weeks of treatment

Important for me Less important

Carbamazepine is a commonly-used antiepileptic medication. It is also used for trigeminal neuralgia and bipolar disorder. Crucially, it is known to induce the P450 enzyme system, which breaks down the drug itself, and so it is considered an auto-inducer. As such, 3-4 weeks after treatment, it may lead to the return of seizures.

Cimetidine is an antihistamine H2-receptor antagonist, used for the control of gastric acid production. It is a P450 inhibitor, not an inducer, and so would not reduce the effectiveness of carbamazepine.

The **combined oral contraceptive pill** is metabolised by the P450 system. It is not known to significantly induce or inhibit the P450 system and so is incorrect.

Like the pill, **montelukast** is also known to be metabolised by the P450 system. Similarly, it is not known to induce or inhibit the system and so is not the correct answer.

Omeprazole is considered a P450 inhibitor, rather than inducer. As such, it would not cause a reduction in carbamazepine levels and so would not be the cause of seizures returning.

👍 👎 Discuss (4) Improve

Next question ›

Carbamazepine ★

Carbamazepine is chemically similar to the tricyclic antidepressant drugs. It is most commonly used in the treatment of epilepsy, particularly partial seizures, where carbamazepine remains a first-line medication. Other uses include

- trigeminal neuralgia
- bipolar disorder

Mechanism of action

- binds to sodium channels increases their refractory period

Adverse effects

- P450 enzyme inducer
- dizziness and ataxia
- drowsiness
- headache
- visual disturbances (especially diplopia)
- Steven-Johnson syndrome
- leucopenia and agranulocytosis
- hyponatraemia secondary to syndrome of inappropriate ADH secretion

Carbamazepine is known to exhibit autoinduction, hence when patients start carbamazepine they may see a return of seizures after 3-4 weeks of treatment.

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

BNF 👍 9 👎 8

Carbamazepine

Suggest link Report broken link

Score: **20.6%**

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Question 174 of 189

A 51-year-old patient with a history of schizophrenia presents after being found on the floor of his sheltered accommodation home surrounded by many packets of medications. There is no collateral history. On examination, the patient is only verbally responsive to pain. There is evidence of vomitus around the oral cavity. You note that he is generally tremulous. Observations show heart rate 110/min, blood pressure 101/61 mmHg.

On the 12 lead ECG the P waves are present followed by QRS complexes. PR interval 0.12 seconds, QRS 0.12 seconds, corrected QT 0.48 seconds. What is the most likely causative agent?

- ○ Citalopram
- ○ Chlorpromazine
- ○ Diazepam
- ○ Digoxin
- ○ Cocaine

Submit answer

Reference ranges

Score: **20%**

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A 51-year-old patient with a history of schizophrenia presents after being found on the floor of his sheltered accommodation home surrounded by many packets of medications. There is no collateral history. On examination, the patient is only verbally responsive to pain. There is evidence of vomitus around the oral cavity. You note that he is generally tremulous. Observations show heart rate 110/min, blood pressure 101/61 mmHg.

On the 12 lead ECG the P waves are present followed by QRS complexes. PR interval 0.12 seconds, QRS 0.12 seconds, corrected QT 0.48 seconds. What is the most likely causative agent?

Citalopram	61%
Chlorpromazine	25%
Diazepam	6%
Digoxin	4%
Cocaine	3%

Other side effects of citalopram overdose to be aware of include:

- Nausea
- Vomiting
- Tremor
- Nystagmus
- Convulsions

Management of SSRI poisoning is generally supportive. Convulsions can be managed with benzodiazepines.

Further information can be found in the BNF.

Discuss (7) | Improve

Next question >

Selective serotonin reuptake inhibitors

Selective serotonin reuptake inhibitors (SSRIs) are considered first-line treatment for the majority of patients with depression.

- citalopram (although see below re: QT interval) and fluoxetine are currently the preferred SSRIs
- sertraline is useful post myocardial infarction as there is more evidence for its safe use in this situation than other antidepressants
- SSRIs should be used with caution in children and adolescents. Fluoxetine is the drug of choice when an antidepressant is indicated

Adverse effects

- gastrointestinal symptoms are the most common side-effect
- there is an increased risk of gastrointestinal bleeding in patients taking SSRIs. A proton pump inhibitor should be prescribed if a patient is also taking a NSAID
- patients should be counselled to be vigilant for increased anxiety and agitation after starting a SSRI
- fluoxetine and paroxetine have a higher propensity for drug interactions

Citalopram and the QT interval

- the Medicines and Healthcare products Regulatory Agency (MHRA) released a warning on the use of citalopram in 2011
- it advised that citalopram and escitalopram are associated with dose-dependent QT interval prolongation and should not be used in those with: congenital long QT syndrome; known pre-existing QT interval prolongation; or in combination with other medicines that prolong the QT interval
- the maximum daily dose is now 40 mg for adults; 20 mg for patients older than 65 years; and 20 mg for those with hepatic impairment

Interactions

- NSAIDs: NICE guidelines advise 'do not normally offer SSRIs', but if given co-prescribe a proton pump inhibitor
- warfarin / heparin: NICE guidelines recommend avoiding SSRIs and considering mirtazapine
- aspirin
- triptans - increased risk of serotonin syndrome
- monoamine oxidase inhibitors (MAOIs) - increased risk of serotonin syndrome

Following the initiation of antidepressant therapy patients should normally be reviewed by a doctor after 2 weeks. For patients under the age of 25 years or at increased risk of suicide should be reviewed after 1 week. If a patient makes a good response to antidepressant therapy they should continue on treatment for at least 6 months after remission as this reduces the risk of relapse.

When stopping a SSRI the dose should be gradually reduced over a 4 week period (this is not necessary with fluoxetine). Paroxetine has a higher incidence of discontinuation symptoms.

Discontinuation symptoms
- increased mood change
- restlessness
- difficulty sleeping
- unsteadiness
- sweating
- gastrointestinal symptoms: pain, cramping, diarrhoea, vomiting
- paraesthesia

SSRIs and pregnancy
- BNF says to weigh up benefits and risk when deciding whether to use in pregnancy.
- Use during the first trimester gives a small increased risk of congenital heart defects
- Use during the third trimester can result in persistent pulmonary hypertension of the newborn
- Paroxetine has an increased risk of congenital malformations, particularly in the first trimester

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 29 👎 43

2022 Depression in adults: treatment and management

Suggest link | Report broken link

Media

Antidepressants

Speed Pharmacology - YouTube 👍 6 👎 0

ANTIDEPRESSANTS
PHARMACOLOGY

Antidepressants Pharmacology

Soton Brain Hub - YouTube 👍 0 👎 0

Report broken media

Score: **20.6%**

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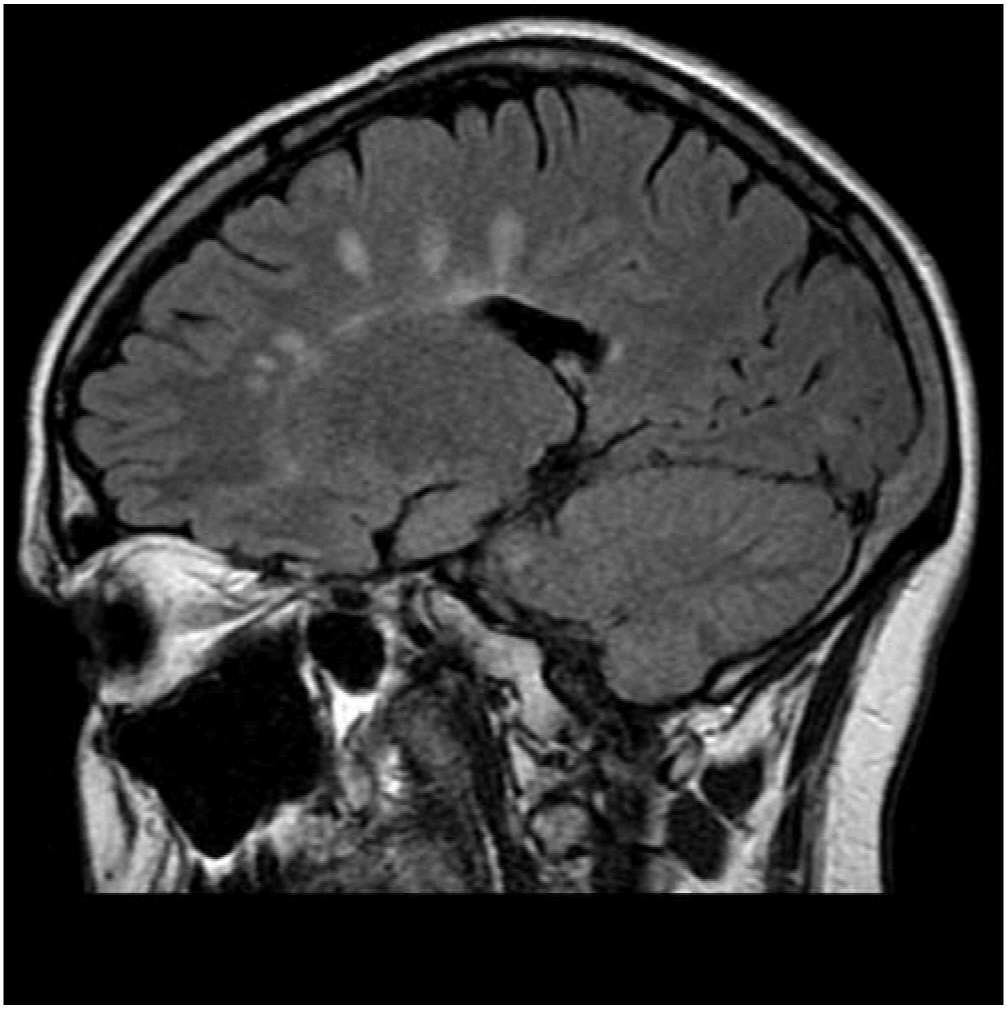
Back to top

A 31-year-old woman is reviewed in the neurology clinic. For the past 6 months she has been presenting to her GP with a variety of symptoms including lethargy, heat intolerance, pins/needles and limb numbness. For the past 4 weeks she has also been complaining of shooting pains in her right hand.

Her GP has arranged a number of blood tests including thyroid function tests and vitamin B12 which were reported as normal.

A neurological examination today demonstrates no consistent neurological findings.

A MRI is requested:



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What is the most likely diagnosis?

○ Multiple sclerosis ×

○ Pituitary tumour ×

○ Ependymoma ×

○ Acute disseminated encephalomyelitis (ADEM) ×

○ Systemic lupus erythematosus ×

Submit answer

Reference ranges ⌵

Score: **20%**

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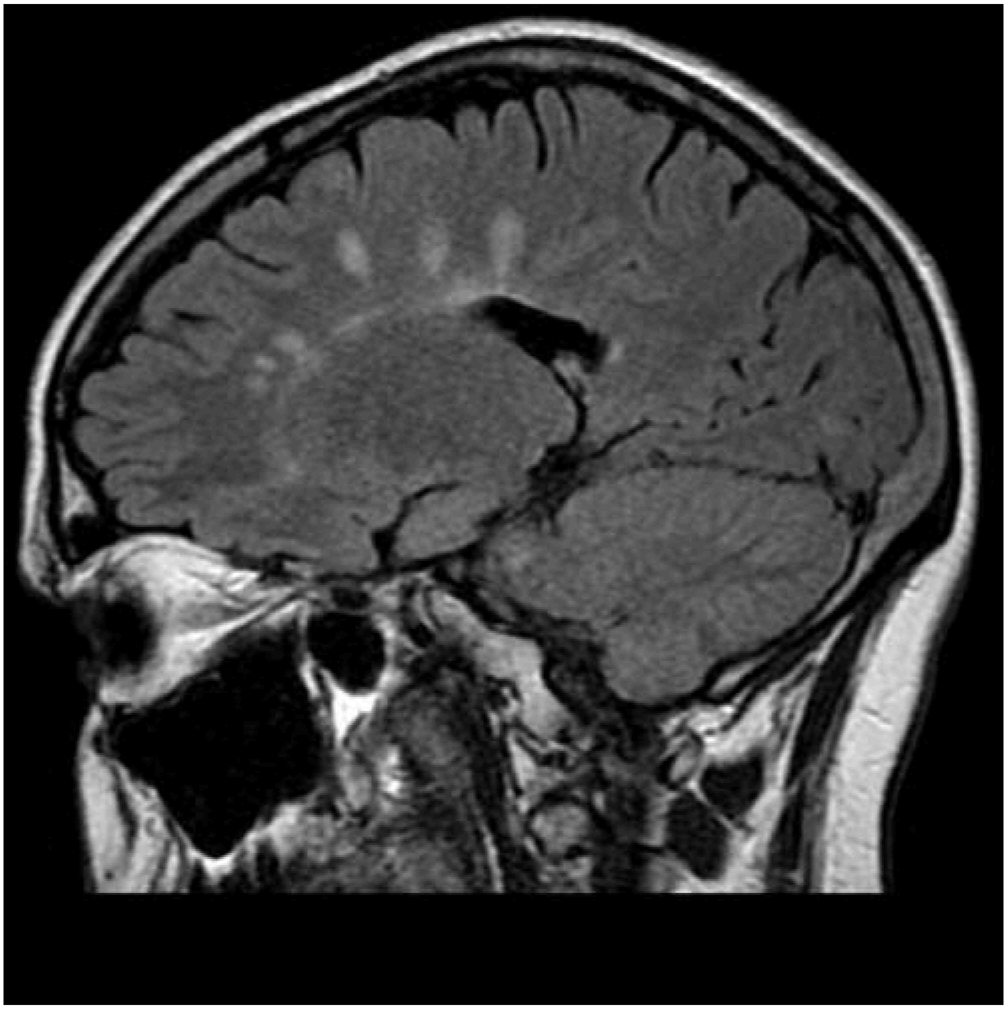

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What is the most likely diagnosis?

Multiple sclerosis	81%
Pituitary tumour	3%
Ependymoma	5%
Acute disseminated encephalomyelitis (ADEM)	10%
Systemic lupus erythematosus	0%

The MRI shows multiple white matter plaques penpendicular to the corpus callosum giving the appearance of Dawson fingers - a classic MRI finding in multiple sclerosis.

Lethargy is a clue in this question. It is very common feature in patients with multiple sclerosis. Other features such as pain and heat intolerance and also seen.

👍 👎 💬 Discuss (2) Improve

Next question ›

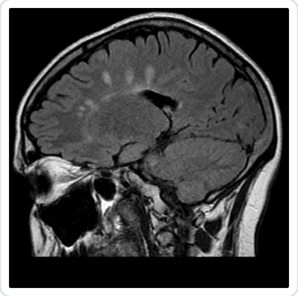
Multiple sclerosis: investigation ★

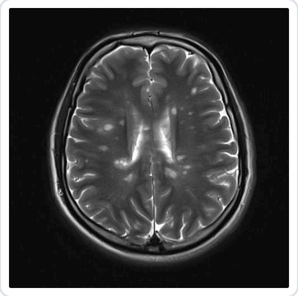
Diagnosis requires demonstration of lesions disseminated in time and space

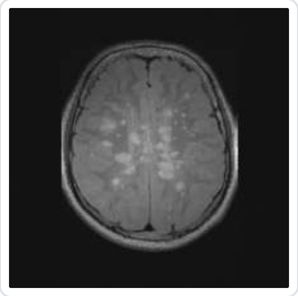
MRI
- high signal T2 lesions
- periventricular plaques
- Dawson fingers: often seen on FLAIR images - hyperintense lesions penpendicular to the corpus callosum

CSF
- oligoclonal bands (and not in serum)
- increased intrathecal synthesis of IgG

Visual evoked potentials
- delayed, but well preserved waveform







123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

DEMYELINATION
T-CELLS
PLAQUES
MULTIPLE SCLEROSIS
OLIGO CLONAL BANDS
RELAPSING-REMITTING

Multiple sclerosis

Rhesus Medicine - YouTube

👍 1 👎 0

MULTIPLE SCLEROSIS

Multiple sclerosis - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube

👍 6 👎 1

Multiple Sclerosis

Multiple sclerosis

Soton Brain Hub - YouTube 👍 2 👎 1

Multiple sclerosis

Podmedics - YouTube 👍 1 👎 3

Report broken media

Score: **20.6%**

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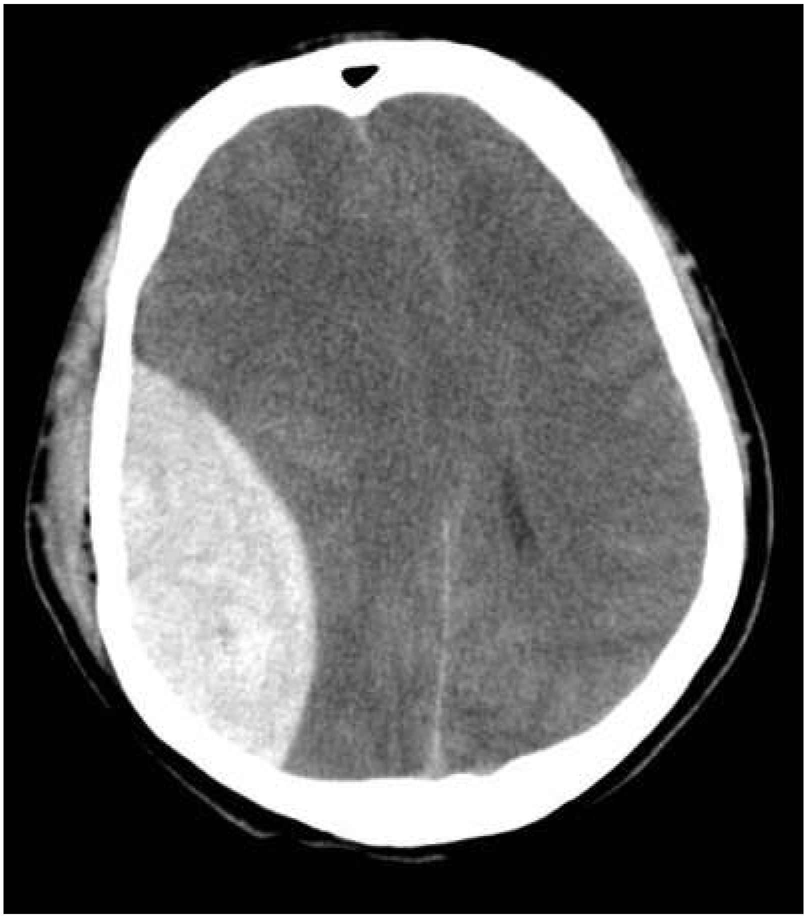

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What does the scan show:

○ Subdural haemotoma

○ Subarachnoid haemorrhage

○ Extradural haematoma

○ Brain contusion

○ Meningioma

Submit answer

Reference ranges ⌵

Score: **20%**

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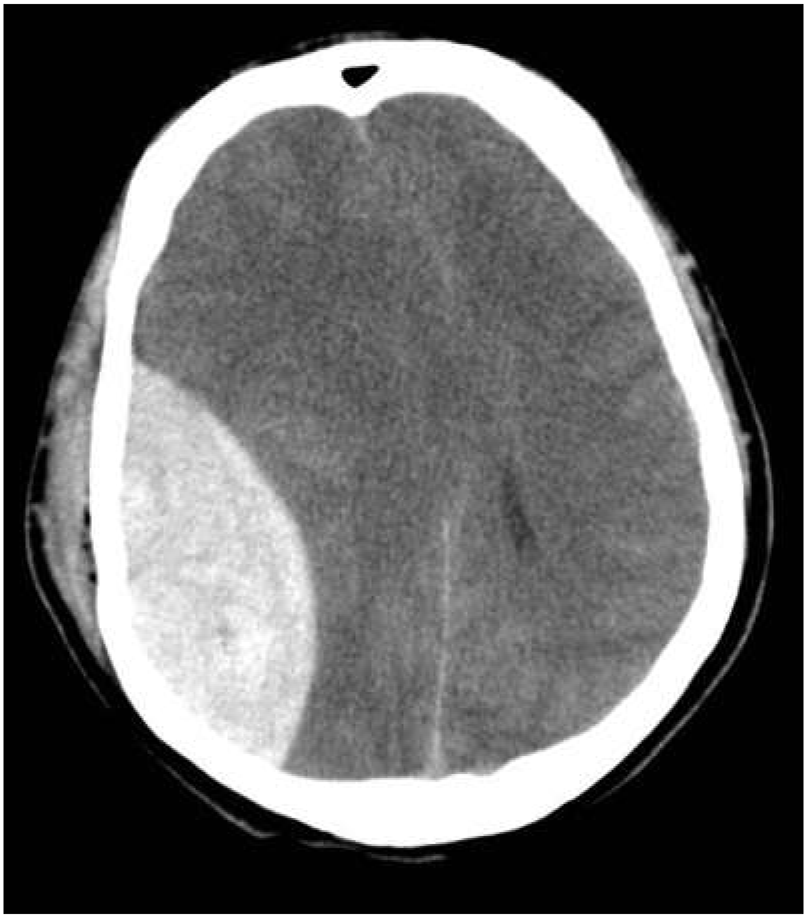

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What does the scan show:

Subdural haematoma	11%
Subarachnoid haemorrhage	1%
Extradural haematoma	88%
Brain contusion	0%
Meningioma	0%

The CT scan shows a extra-axial biconvex collection associated with fracture suggestive of extradural haematoma.

👍 👎 Discuss (5) Improve

Next question >

Head injury: types of traumatic brain injury ★

Basics

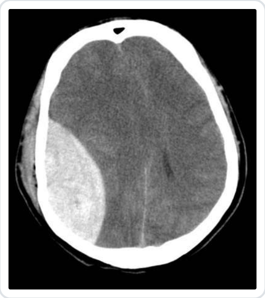
- primary brain injury may be focal (contusion/haematoma) or diffuse (diffuse axonal injury)
- diffuse axonal injury occurs as a result of mechanical shearing following deceleration, causing disruption and tearing of axons
- intra-cranial haematomas can be extradural, subdural or intracerebral, while contusions may occur adjacent to (coup) or contralateral (contre-coup) to the side of impact
- secondary brain injury occurs when cerebral oedema, ischaemia, infection, tonsillar or tentorial herniation exacerbates the original injury. The normal cerebral auto regulatory processes are disrupted following trauma rendering the brain more susceptible to blood flow changes and hypoxia
- the Cushings reflex (hypertension and bradycardia) often occurs late and is usually a pre terminal event

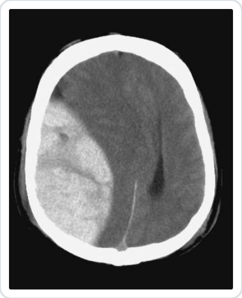
Type of injury	**Notes**
Extradural (epidural) haematoma	Bleeding into the space between the dura mater and the skull. Often results from acceleration-deceleration trauma or a blow to the side of the head. The majority of epidural haematomas occur in the temporal region where skull fractures cause a rupture of the middle meningeal artery. Features • features of raised intracranial pressure • some patients may exhibit a lucid interval
Subdural haematoma	Bleeding into the outermost meningeal layer. Most commonly occur around the frontal and parietal lobes. Risk factors include old age, alcoholism and anticoagulation.

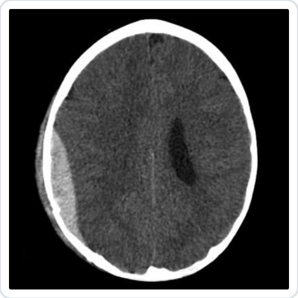
Type of injury	Notes
	Slower onset of symptoms than a epidural haematoma. There may be fluctuating confusion/consciousness
Subarachnoid haemorrhage	Classically causes a sudden occipital headache. Usually occurs spontaneously in the context of a ruptured cerebral aneurysm but may be seen in association with other injuries when a patient has sustained a traumatic brain injury
Intracerebral haematoma	An intracerebral (or intraparenchymal) haemorrhage is a collection of blood within the substance of the brain. Causes / risk factors include: hypertension, vascular lesion (e.g. aneurysm or arteriovenous malformation), cerebral amyloid angiopathy, trauma, brain tumour or infarct (particularly in stroke patients undergoing thrombolysis). Patients will present similarly to an ischaemic stroke (which is why it is crucial to obtain a CT in head in all stroke patients prior to thrombolysis) or with a decrease in consciousness. CT imaging will show a hyperdensity (bright lesion) within the substance of the brain. Treatment is often conservative under the care of stroke physicians, but large clots in patients with impaired consciousness may warrant surgical evacuation.

Image gallery

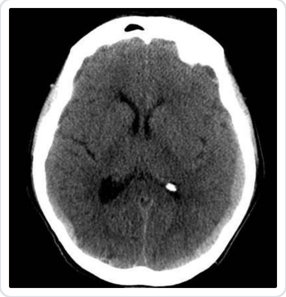
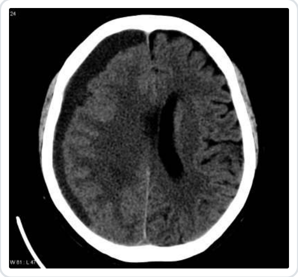
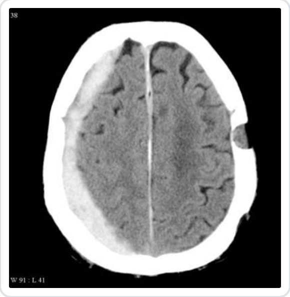
Extradural (epidural) haematoma:



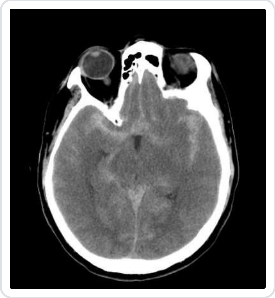
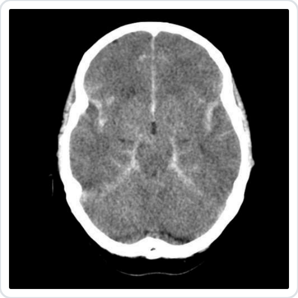




Subdural haematoma:

Subarachnoid haemorrhage:

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

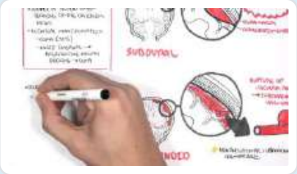
NICE 11 2

2014 Head injury guidelines

Suggest link

Report broken link

Media



Intracranial Haemorrhage Types

Armando Hasudungan - YouTube 👍 6 👎 0

Intracranial
Bleeds
subdural
&
extradural

Intracranial bleeds - Subdural and Extradural haematoma

Soton Brain Hub - YouTube 👍 4 👎 0

TRAUMATIC
BRAIN INJURY
~ PATHOLOGY REVIEW ~
Osmosis.org

Traumatic brain injury: pathology review

Osmosis - YouTube 👍 4 👎 1

Report broken media

Score: **20.6%**

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A 73-year-old man who is a chronic alcoholic is brought to the Emergency Department by an ambulance. He was found collapsed in the street. On admission his airway, breathing and circulation are satisfactory. His GCS is 13/15 (eyes = 3, verbal = 4, movement = 6) although his level of consciousness appears to be fluctuating. There are no obvious signs of external head injury.

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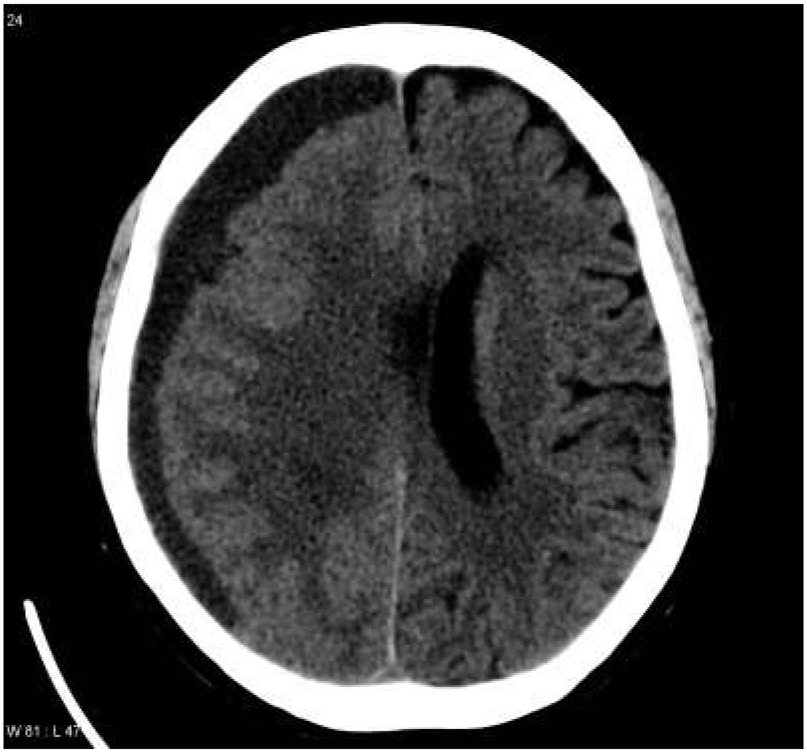

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What is the most likely diagnosis?

○ Subarachnoid haemorrhage

○ Wernicke's encephalopathy

○ Subdural haematoma

○ Meningioma

○ Extradural haematoma

Submit answer

Reference ranges

Score: **20%**

1	✗
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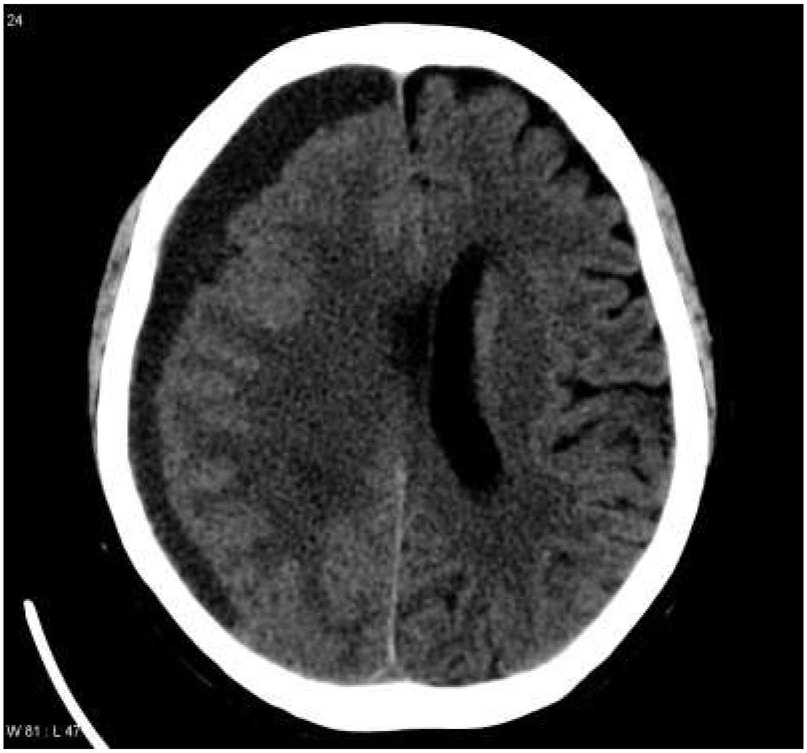

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What is the most likely diagnosis?

Subarachnoid haemorrhage	2%
Wernicke's encephalopathy	2%
Subdural haemorrhage	90%
Meningioma	0%
Extradural haematoma	6%

CT scan demonstrates a right sided chronic subdural haemorrhage resulting in midline shift.

👍 | 👎 | Discuss (3) | Improve

Next question ›

Head injury: types of traumatic brain injury ★

Basics

- primary brain injury may be focal (contusion/haematoma) or diffuse (diffuse axonal injury)
- diffuse axonal injury occurs as a result of mechanical shearing following deceleration, causing disruption and tearing of axons
- intra-cranial haematomas can be extradural, subdural or intracerebral, while contusions may occur adjacent to (coup) or contralateral (contre-coup) to the side of impact
- secondary brain injury occurs when cerebral oedema, ischaemia, infection, tonsillar or tentorial herniation exacerbates the original injury. The normal cerebral auto regulatory processes are disrupted following trauma rendering the brain more susceptible to blood flow changes and hypoxia
- the Cushings reflex (hypertension and bradycardia) often occurs late and is usually a pre terminal event

Type of injury	Notes
Extradural (epidural) haematoma	Bleeding into the space between the dura mater and the skull. Often results from acceleration-deceleration trauma or a blow to the side of the head. The majority of epidural haematomas occur in the temporal region where skull fractures cause a rupture of the middle meningeal artery. Features • features of raised intracranial pressure • some patients may exhibit a lucid interval
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Type of injury	Notes
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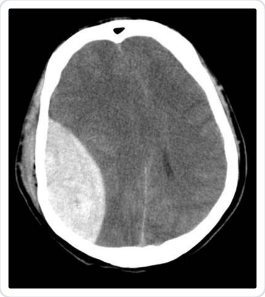
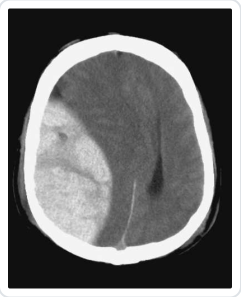
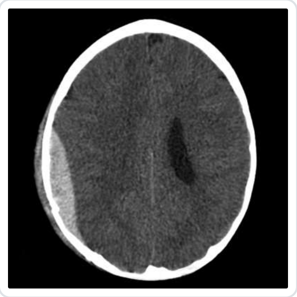
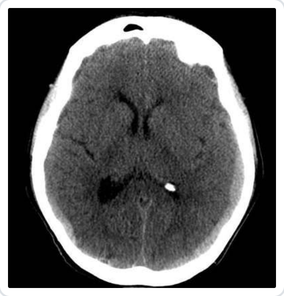
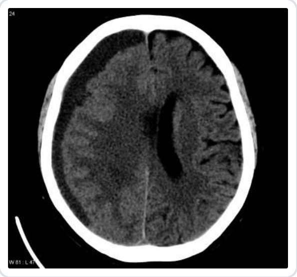
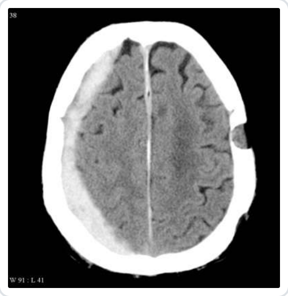
Image gallery

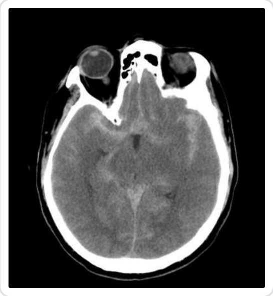
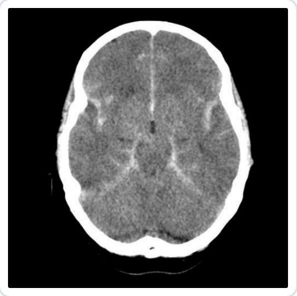
Extradural (epidural) haematoma:

Subdural haematoma:

Subarachnoid haemorrhage:

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

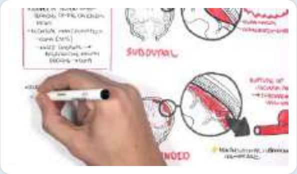
NICE 11 2

2014 Head injury guidelines

Suggest link

Report broken link

Media



Intracranial Haemorrhage Types

Armando Hasudungan - YouTube 👍 6 👎 0

Intracranial
Bleeds
subdural
&
extradural

Intracranial bleeds - Subdural and Extradural haematoma

Soton Brain Hub - YouTube 👍 4 👎 0

TRAUMATIC
BRAIN INJURY
~ PATHOLOGY REVIEW ~
Osmosis.org

Traumatic brain injury: pathology review

Osmosis - YouTube 👍 4 👎 1

Report broken media

Score: **20.6%**

1 ✗
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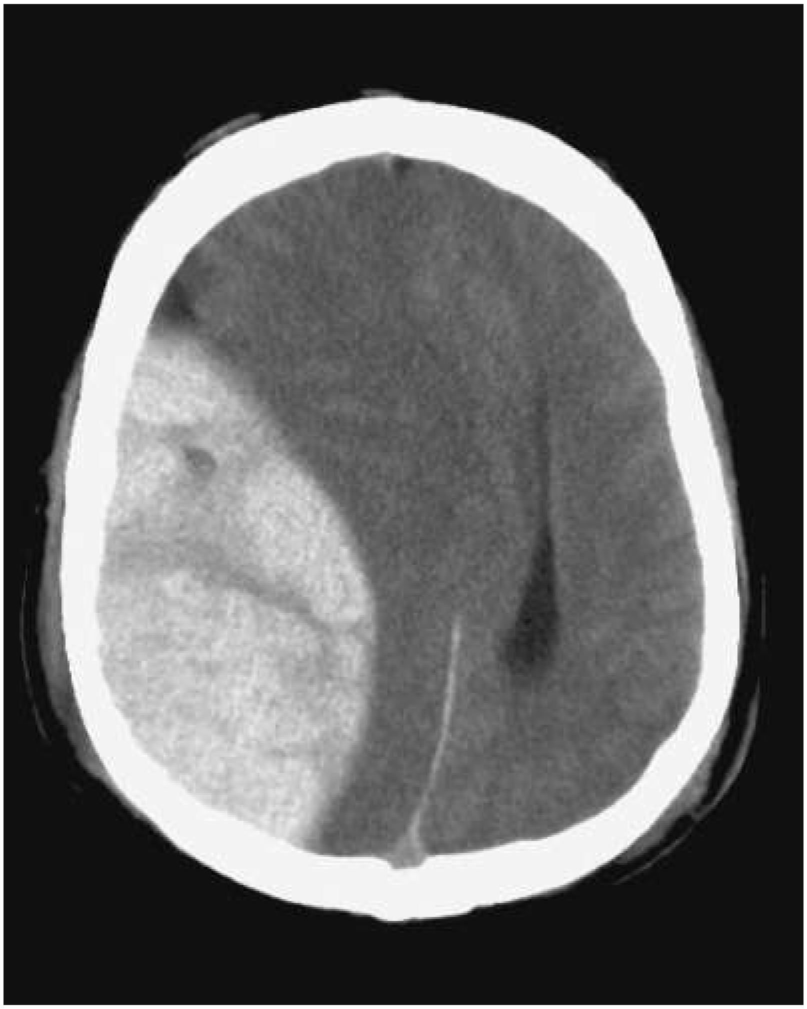
Back to top

A 30-year-old man who has been seriously assaulted is brought to the Emergency Department. His GCS is 5/15 on arrival and he is immediately intubated and transferred for a CT head (with contrast):



© Image used on license from Radiopaedia

Which blood vessel(s) is most likely to have ruptured?

○ Anterior communicating artery

○ Emissary veins

○ Temporal artery

○ Middle meningeal artery

○ Posterior communicating artery ✕

Submit answer

Reference ranges ⌄

Score: **20%**

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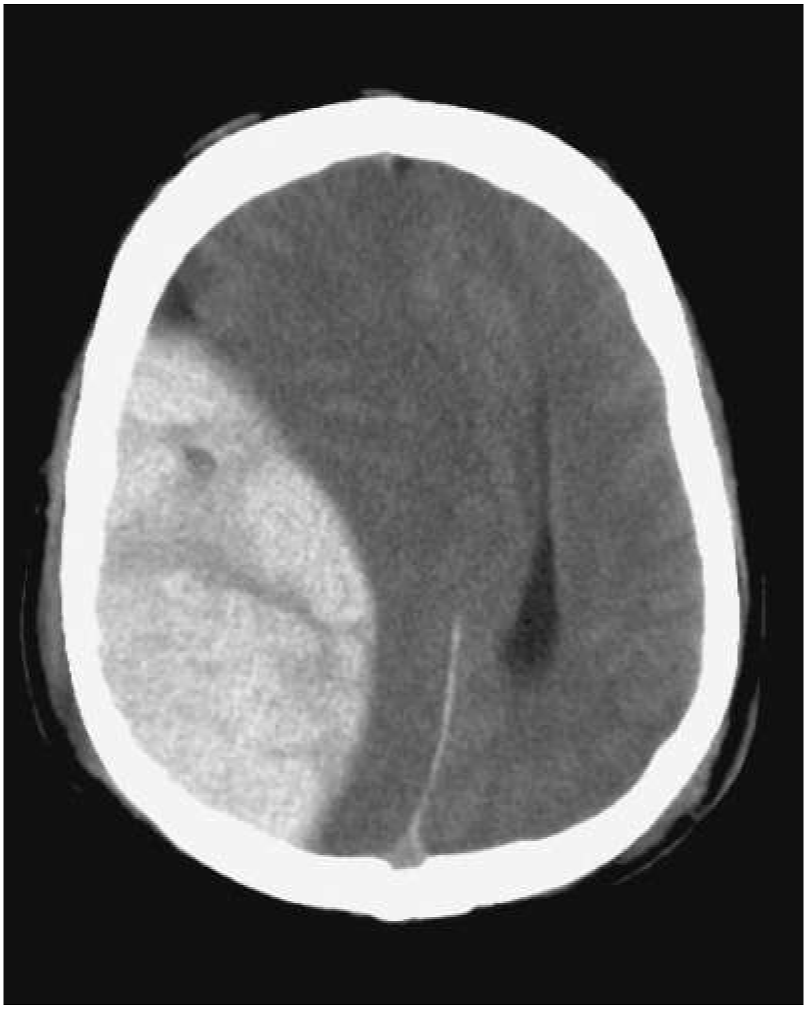
Question 178 of 189

A 30-year-old man who has been seriously assaulted is brought to the Emergency Department. His GCS is 5/15 on arrival and he is immediately intubated and transferred for a CT head (with contrast):



© Image used on license from Radiopaedia

Which blood vessel(s) is most likely to have ruptured?

Anterior communicating artery	2%
Emissary veins	6%
Temporal artery	5%
Middle meningeal artery	86%

Posterior communicating artery 2%

This scan shows a massive extradural haemorrhage, most likely caused by a ruptured middle meningeal artery.

👍 👎 💬 Discuss (2) Improve

Next question ›

Head injury: types of traumatic brain injury ★

Basics

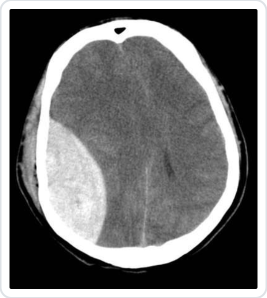
- primary brain injury may be focal (contusion/haematoma) or diffuse (diffuse axonal injury)
- diffuse axonal injury occurs as a result of mechanical shearing following deceleration, causing disruption and tearing of axons
- intra-cranial haematomas can be extradural, subdural or intracerebral, while contusions may occur adjacent to (coup) or contralateral (contre-coup) to the side of impact
- secondary brain injury occurs when cerebral oedema, ischaemia, infection, tonsillar or tentorial herniation exacerbates the original injury. The normal cerebral auto regulatory processes are disrupted following trauma rendering the brain more susceptible to blood flow changes and hypoxia
- the Cushings reflex (hypertension and bradycardia) often occurs late and is usually a pre terminal event

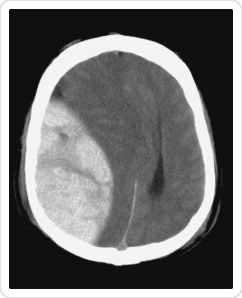
Type of injury	**Notes**
Extradural (epidural) haematoma	Bleeding into the space between the dura mater and the skull. Often results from acceleration-deceleration trauma or a blow to the side of the head. The majority of epidural haematomas occur in the temporal region where skull fractures cause a rupture of the middle meningeal artery. Features • features of raised intracranial pressure • some patients may exhibit a lucid interval
Subdural haematoma	Bleeding into the outermost meningeal layer. Most commonly occur around the frontal and parietal lobes. Risk factors include old age, alcoholism and anticoagulation.

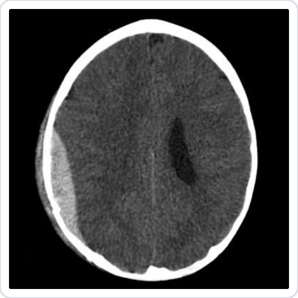
Type of injury	Notes
	Slower onset of symptoms than a epidural haematoma. There may be fluctuating confusion/consciousness
Subarachnoid haemorrhage	Classically causes a sudden occipital headache. Usually occurs spontaneously in the context of a ruptured cerebral aneurysm but may be seen in association with other injuries when a patient has sustained a traumatic brain injury
Intracerebral haematoma	An intracerebral (or intraparenchymal) haemorrhage is a collection of blood within the substance of the brain. Causes / risk factors include: hypertension, vascular lesion (e.g. aneurysm or arteriovenous malformation), cerebral amyloid angiopathy, trauma, brain tumour or infarct (particularly in stroke patients undergoing thrombolysis). Patients will present similarly to an ischaemic stroke (which is why it is crucial to obtain a CT in head in all stroke patients prior to thrombolysis) or with a decrease in consciousness. CT imaging will show a hyperdensity (bright lesion) within the substance of the brain. Treatment is often conservative under the care of stroke physicians, but large clots in patients with impaired consciousness may warrant surgical evacuation.

Image gallery

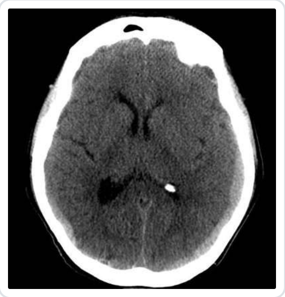
Extradural (epidural) haematoma:

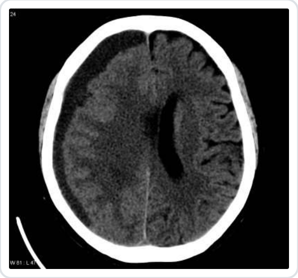


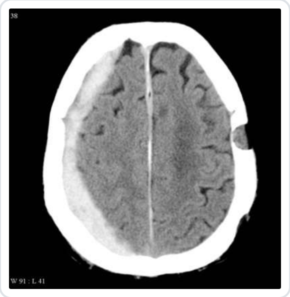




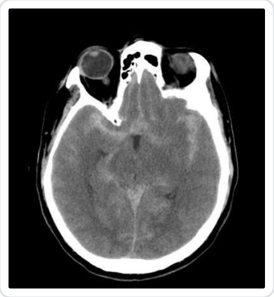
Subdural haematoma:

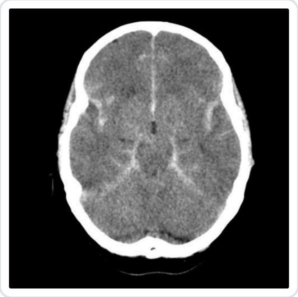






Subarachnoid haemorrhage:





123

Next question >

Textbooks

High-yield textbook

Extended textbook

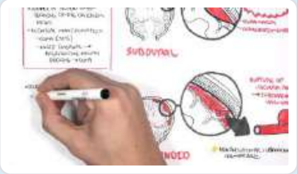
Links

NICE 👍 11 👎 2

2014 Head injury guidelines

Suggest link Report broken link

Media



Intracranial Haemorrhage Types

Armando Hasudungan - YouTube 👍 6 👎 0

Intracranial
Bleeds
subdural
&
extradural

Intracranial bleeds - Subdural and Extradural haematoma

Soton Brain Hub - YouTube 👍 4 👎 0

TRAUMATIC
BRAIN INJURY
~ PATHOLOGY REVIEW ~
Osmosis.org

Traumatic brain injury: pathology review

Osmosis - YouTube 👍 4 👎 1

Report broken media

Score: **20.6%**

1 ✗
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188 ✗

189 ✓

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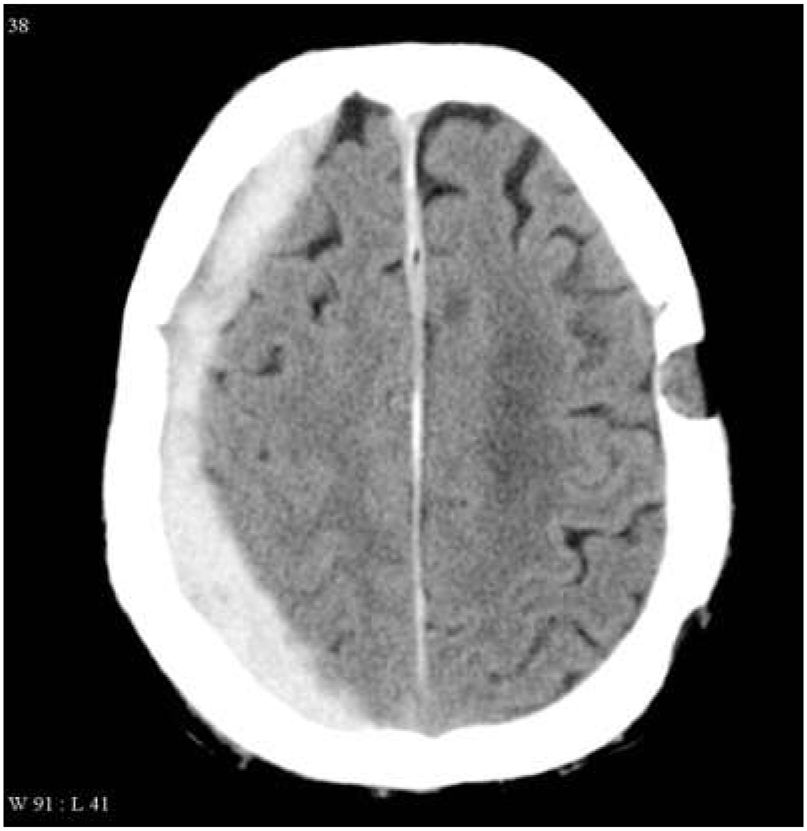
Back to top

An 85-year-old complains of severe headaches and lethargy over the past 3 weeks. His past medical history includes atrial fibrillation for which he takes warfarin. A CT head (with contrast) is performed:



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What is the most likely diagnosis?

○ Normal age-related changes

○ Extradural haematoma

○ Subdural haematoma

○ Meningioma

○ Subarachnoid haemorrhage

Submit answer

Reference ranges

Score: **20%**

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179 -

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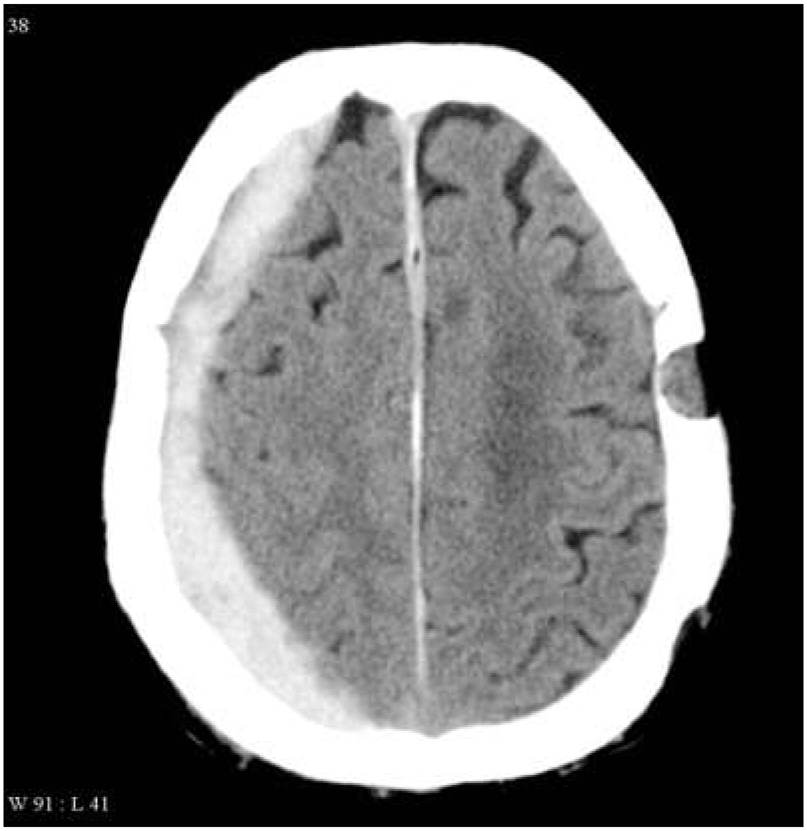
Question 179 of 189

An 85-year-old complains of severe headaches and lethargy over the past 3 weeks. His past medical history includes atrial fibrillation for which he takes warfarin. A CT head (with contrast) is performed:



© Image used on license from Radiopaedia

What is the most likely diagnosis?

Normal age-related changes	0%
Extradural haematoma	6%
Subdural haematoma	93%
Meningioma	0%
Subarachnoid haemorrhage	1%

The CT scan shows a crescent-shaped (concave surface away from the skull) extra-axial collection seen with subdural haematomas. A burr hole can also be seen on the left.

Discuss (1) Improve

Next question

Head injury: types of traumatic brain injury ★

Basics

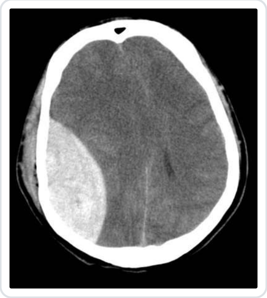
- primary brain injury may be focal (contusion/haematoma) or diffuse (diffuse axonal injury)
- diffuse axonal injury occurs as a result of mechanical shearing following deceleration, causing disruption and tearing of axons
- intra-cranial haematomas can be extradural, subdural or intracerebral, while contusions may occur adjacent to (coup) or contralateral (contre-coup) to the side of impact
- secondary brain injury occurs when cerebral oedema, ischaemia, infection, tonsillar or tentorial herniation exacerbates the original injury. The normal cerebral auto regulatory processes are disrupted following trauma rendering the brain more susceptible to blood flow changes and hypoxia
- the Cushings reflex (hypertension and bradycardia) often occurs late and is usually a pre terminal event

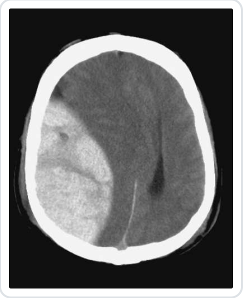
Type of injury	Notes
Extradural (epidural) haematoma	Bleeding into the space between the dura mater and the skull. Often results from acceleration-deceleration trauma or a blow to the side of the head. The majority of epidural haematomas occur in the temporal region where skull fractures cause a rupture of the middle meningeal artery. Features • features of raised intracranial pressure • some patients may exhibit a lucid interval
Subdural haematoma	Bleeding into the outermost meningeal layer. Most commonly occur around the frontal and parietal lobes. Risk factors include old age, alcoholism and anticoagulation. Slower onset of symptoms than a epidural haematoma. There may be fluctuating confusion/consciousness
Subarachnoid haemorrhage	Classically causes a sudden occipital headache. Usually occurs spontaneously in the context of a ruptured cerebral aneurysm but may be

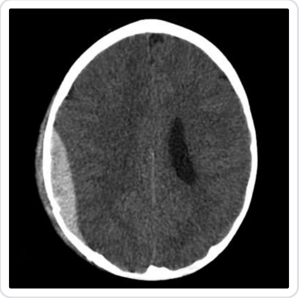
Type of injury	Notes
	seen in association with other injuries when a patient has sustained a traumatic brain injury
Intracerebral haematoma	An intracerebral (or intraparenchymal) haemorrhage is a collection of blood within the substance of the brain. Causes / risk factors include: hypertension, vascular lesion (e.g. aneurysm or arteriovenous malformation), cerebral amyloid angiopathy, trauma, brain tumour or infarct (particularly in stroke patients undergoing thrombolysis). Patients will present similarly to an ischaemic stroke (which is why it is crucial to obtain a CT in head in all stroke patients prior to thrombolysis) or with a decrease in consciousness. CT imaging will show a hyperdensity (bright lesion) within the substance of the brain. Treatment is often conservative under the care of stroke physicians, but large clots in patients with impaired consciousness may warrant surgical evacuation.

Image gallery

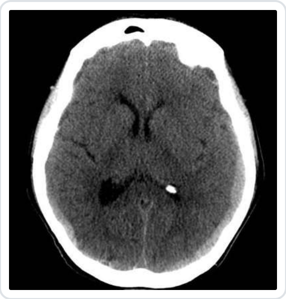
Extradural (epidural) haematoma:

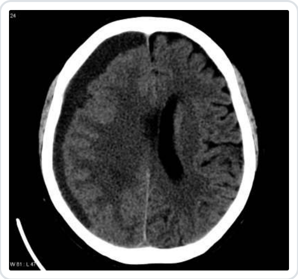


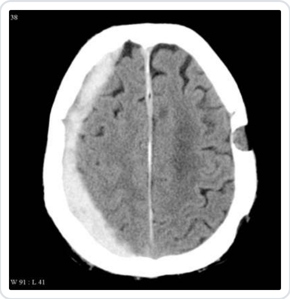




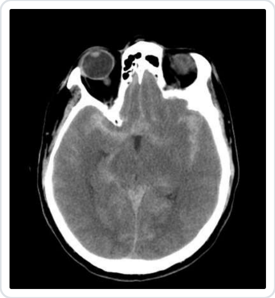
Subdural haematoma:

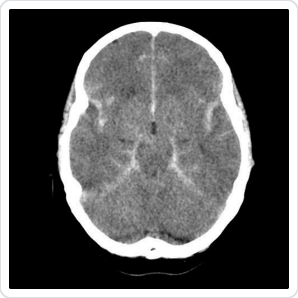






Subarachnoid haemorrhage:





AI + Q 123

Next question >

B *I* A

Textbooks

High-yield textbook

Extended textbook

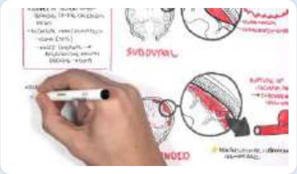
Links

NICE 11 2

2014 Head injury guidelines

Suggest link Report broken link

Media



Intracranial Haemorrhage Types

Armando Hasudungan - YouTube 👍 6 👎 0

Intracranial
Bleeds
subdural
&
extradural

Intracranial bleeds - Subdural and Extradural haematoma

Soton Brain Hub - YouTube 👍 4 👎 0

TRAUMATIC
BRAIN INJURY
~ PATHOLOGY REVIEW ~
Osmosis.org

Traumatic brain injury: pathology review

Osmosis - YouTube 👍 4 👎 1

Report broken media

Score: **20.6%**

1 ✗
2 ✗
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5 ✓
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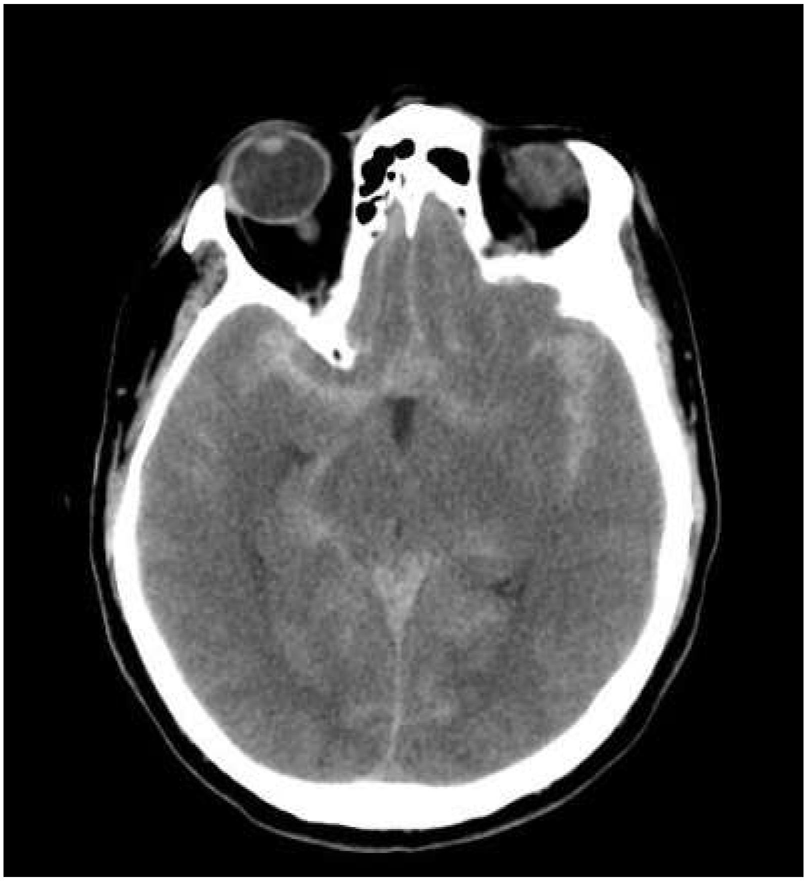
Back to top

A 33-year-old woman presents to the Emergency Department with a severe headache. She describes falling to the ground whilst walking to work and since having a severe occipital headache. Her GCS is 15/15 and her vital signs are stable, other than a low grade pyrexia of 37.5ºC. Her headache fails to improve with analgesia and a CT head with contrast is performed:



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What does the scan show?

- ○ Extradural haematoma
- ○ Brain contusion
- ○ Subdural haematoma
- ○ Meningioma
- ○ Subarachnoid haemorrhage

Submit answer

Reference ranges ⌵

Score: **20%**

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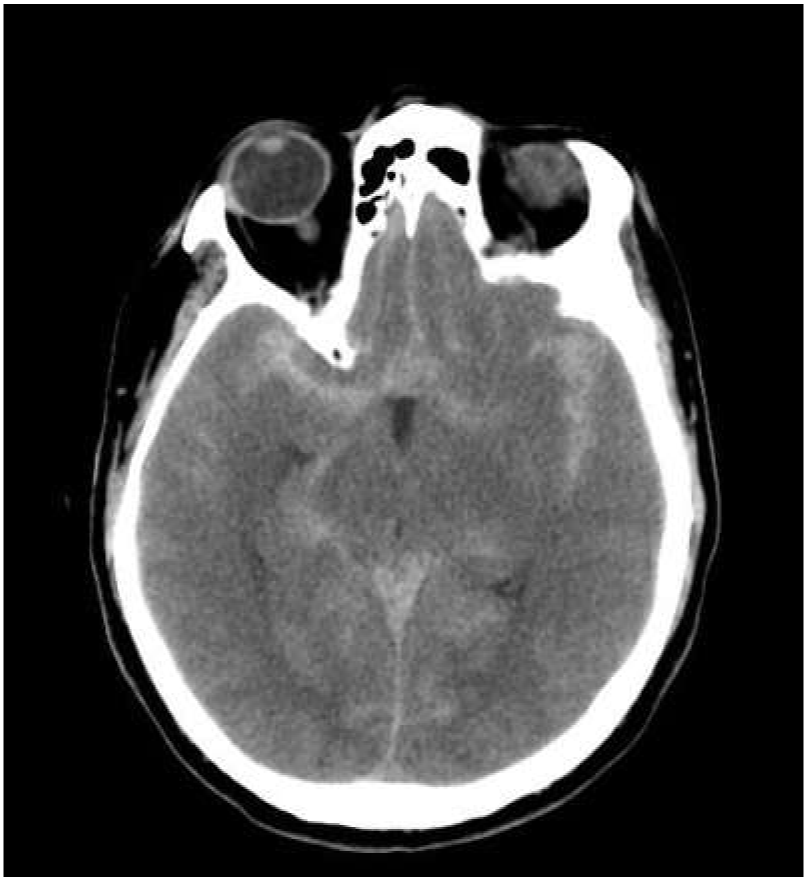
A 33-year-old woman presents to the Emergency Department with a severe headache. She describes falling to the ground whilst walking to work and since having a severe occipital headache. Her GCS is 15/15 and her vital signs are stable, other than a low grade pyrexia of 37.5ºC. Her headache fails to improve with analgesia and a CT head with contrast is performed:



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What does the scan show?

Extradural haematoma	2%
Brain contusion	11%
Subdural haematoma	3%
Meningioma	2%
Subarachnoid haemorrhage	83%

The correct answer is **Subarachnoid haemorrhage**. This condition typically presents with a sudden onset, severe headache (often described as a 'thunderclap' headache). The patient's symptoms of collapsing and having an occipital headache are consistent with this. A subarachnoid haemorrhage can also cause fever due to blood being irritating to the meninges, which could explain the patient's low-grade pyrexia. On CT imaging, subarachnoid haemorrhage appears as areas of high density in the subarachnoid space.

The first incorrect option is **Extradural Haematoma**. This usually results from trauma leading to bleeding between the dura mater and skull. It often presents with a lucid interval followed by rapid neurological deterioration. The CT appearance is typically a lens-shaped hyperdensity that does not cross suture lines, which does not match the image shown.

The second incorrect option is **Brain contusion**. This refers to bruising of brain tissue following head injury. The clinical presentation might include loss of consciousness at the time of injury and focal neurological deficits depending on the area affected. On CT imaging, brain contusions appear as irregular areas of low density due to oedema and high density due to haemorrhage; however, these findings are not evident in this case.

Subdural Haematoma, the third incorrect option, occurs when there is bleeding into the space between the dura mater and arachnoid membrane, most commonly due to tearing of bridging veins during trauma. Clinically, patients may present with fluctuating consciousness level or focal neurological deficits. On CT scan, it appears as a crescent-shaped hyperdensity that can cross suture lines but not dural reflections - again inconsistent with our image.

Lastly, **Meningioma**, another incorrect option, refers to benign tumours arising from meningeal layers surrounding the brain and spinal cord. Patients usually present with slowly progressive symptoms related to increased intracranial pressure or focal neurologic deficits based on tumour location. Meningiomas on CT scans appear as extra-axial masses that are typically homogenous and isodense or slightly hyperdense compared to brain parenchyma - features not seen in this case.

👍 👎 Discuss (5) Improve

Next question >

Head injury: types of traumatic brain injury ★

Basics

- primary brain injury may be focal (contusion/haematoma) or diffuse (diffuse axonal injury)

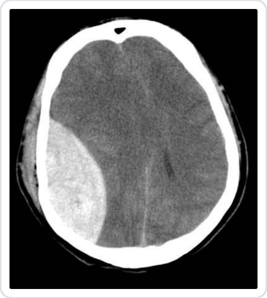
- diffuse axonal injury occurs as a result of mechanical shearing following deceleration, causing disruption and tearing of axons
- intra-cranial haematomas can be extradural, subdural or intracerebral, while contusions may occur adjacent to (coup) or contralateral (contre-coup) to the side of impact
- secondary brain injury occurs when cerebral oedema, ischaemia, infection, tonsillar or tentorial herniation exacerbates the original injury. The normal cerebral auto regulatory processes are disrupted following trauma rendering the brain more susceptible to blood flow changes and hypoxia
- the Cushings reflex (hypertension and bradycardia) often occurs late and is usually a pre terminal event

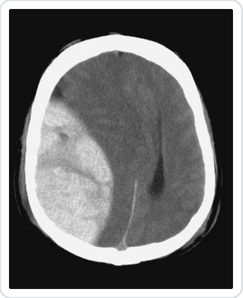
Type of injury	Notes
Extradural (epidural) haematoma	Bleeding into the space between the dura mater and the skull. Often results from acceleration-deceleration trauma or a blow to the side of the head. The majority of epidural haematomas occur in the temporal region where skull fractures cause a rupture of the middle meningeal artery. Features • features of raised intracranial pressure • some patients may exhibit a lucid interval
Subdural haematoma	Bleeding into the outermost meningeal layer. Most commonly occur around the frontal and parietal lobes. Risk factors include old age, alcoholism and anticoagulation. Slower onset of symptoms than a epidural haematoma. There may be fluctuating confusion/consciousness
Subarachnoid haemorrhage	Classically causes a sudden occipital headache. Usually occurs spontaneously in the context of a ruptured cerebral aneurysm but may be seen in association with other injuries when a patient has sustained a traumatic brain injury
Intracerebral haematoma	An intracerebral (or intraparenchymal) haemorrhage is a collection of blood within the substance of the brain. Causes / risk factors include: hypertension, vascular lesion (e.g. aneurysm or arteriovenous malformation), cerebral amyloid angiopathy, trauma, brain tumour or infarct (particularly in stroke patients undergoing thrombolysis).

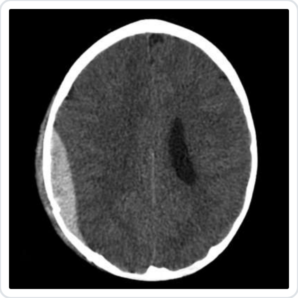
Type of injury	Notes
	Patients will present similarly to an ischaemic stroke (which is why it is crucial to obtain a CT in head in all stroke patients prior to thrombolysis) or with a decrease in consciousness. CT imaging will show a hyperdensity (bright lesion) within the substance of the brain. Treatment is often conservative under the care of stroke physicians, but large clots in patients with impaired consciousness may warrant surgical evacuation.

Image gallery

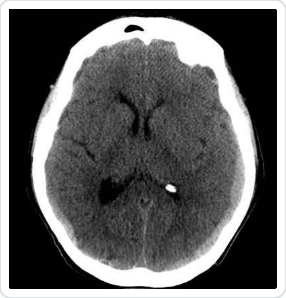
Extradural (epidural) haematoma:

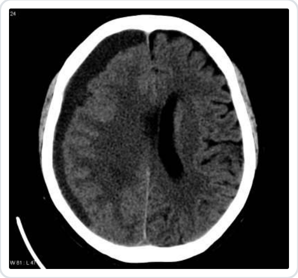


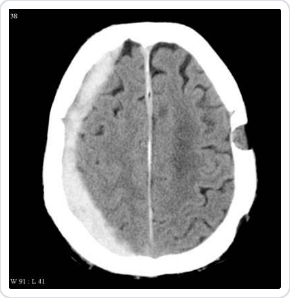




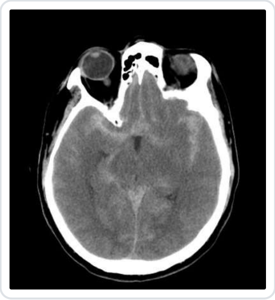
Subdural haematoma:

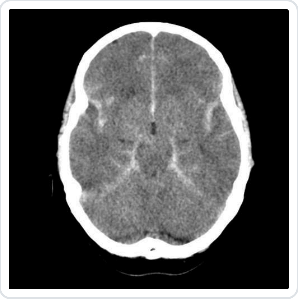






Subarachnoid haemorrhage:





+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

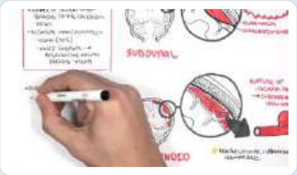
Links

NICE 👍 11 👎 2

2014 Head injury guidelines

Suggest link Report broken link

Media



Intracranial Haemorrhage Types

Armando Hasudungan - YouTube 👍 6 👎 0

Intracranial
Bleeds
subdural
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extradural

Intracranial bleeds - Subdural and Extradural haematoma

Soton Brain Hub - YouTube 👍 4 👎 0

TRAUMATIC
BRAIN INJURY
~ PATHOLOGY REVIEW ~
Osmosis.org

Traumatic brain injury: pathology review

Osmosis - YouTube 👍 4 👎 1

Report broken media

Score: **20.6%**

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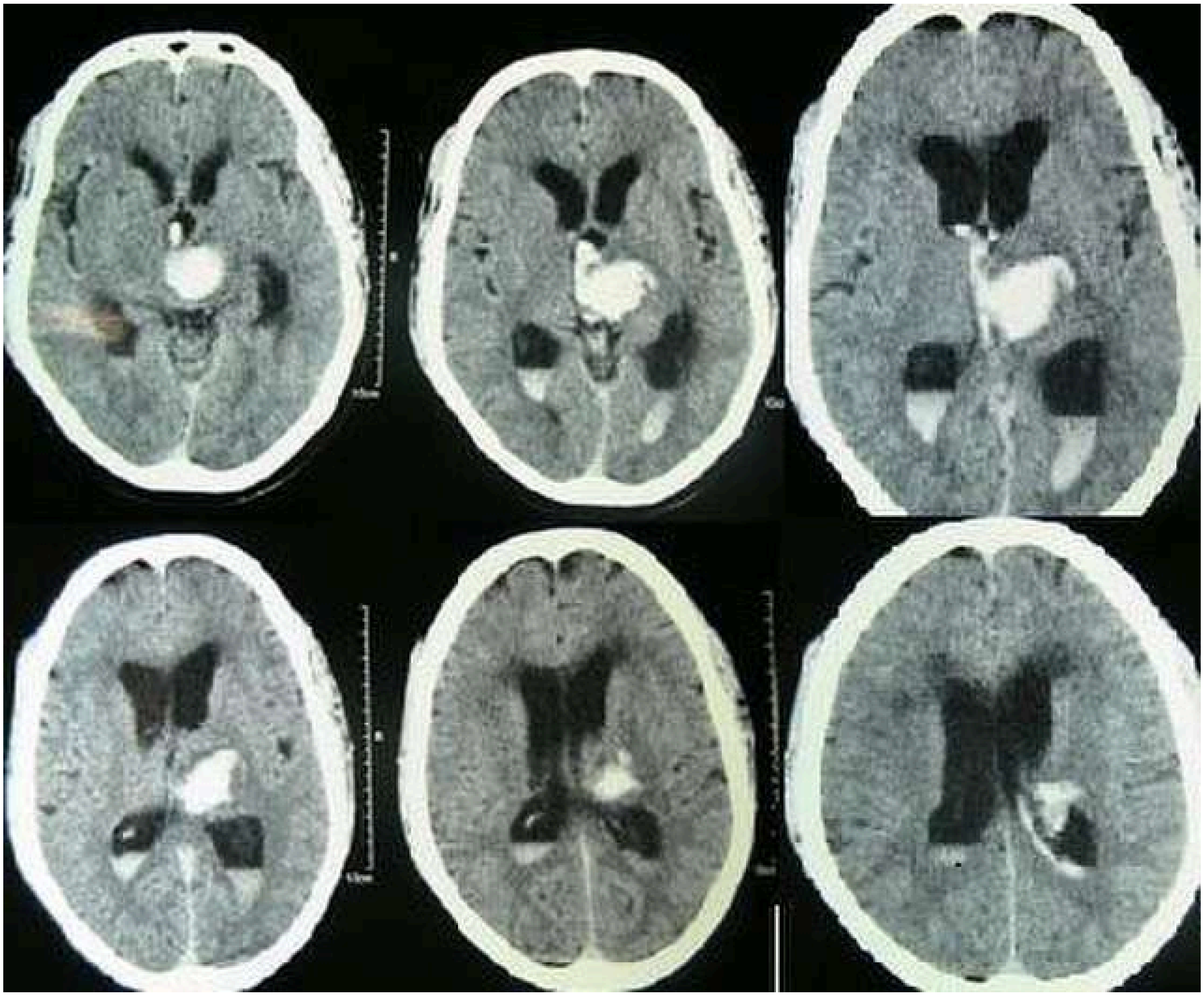
A 72-year-old woman patient presents to the emergency department with a sudden onset of severe headache, worse than any she's ever experienced, which started an hour ago. She also reports weakness on the right side of her body and difficulty speaking. There is no history of trauma.

Her medical history reveals longstanding hypertension, which she admits to not managing well in recent months. On examination, she appears drowsy. Her blood pressure is 195/110 mmHg. Her neurological examination reveals right-sided hemiparesis and expressive aphasia.

Urgent head imaging is ordered.



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What is the most likely diagnosis?

○ Brain tumour

○ Intracerebral and intraventricular hemorrhage

○ Intracerebral and subarachnoid haemorrhage

○ Ischaemic stroke

○ Subdural haemorrhage

Submit answer

Reference ranges

Score: **20%**

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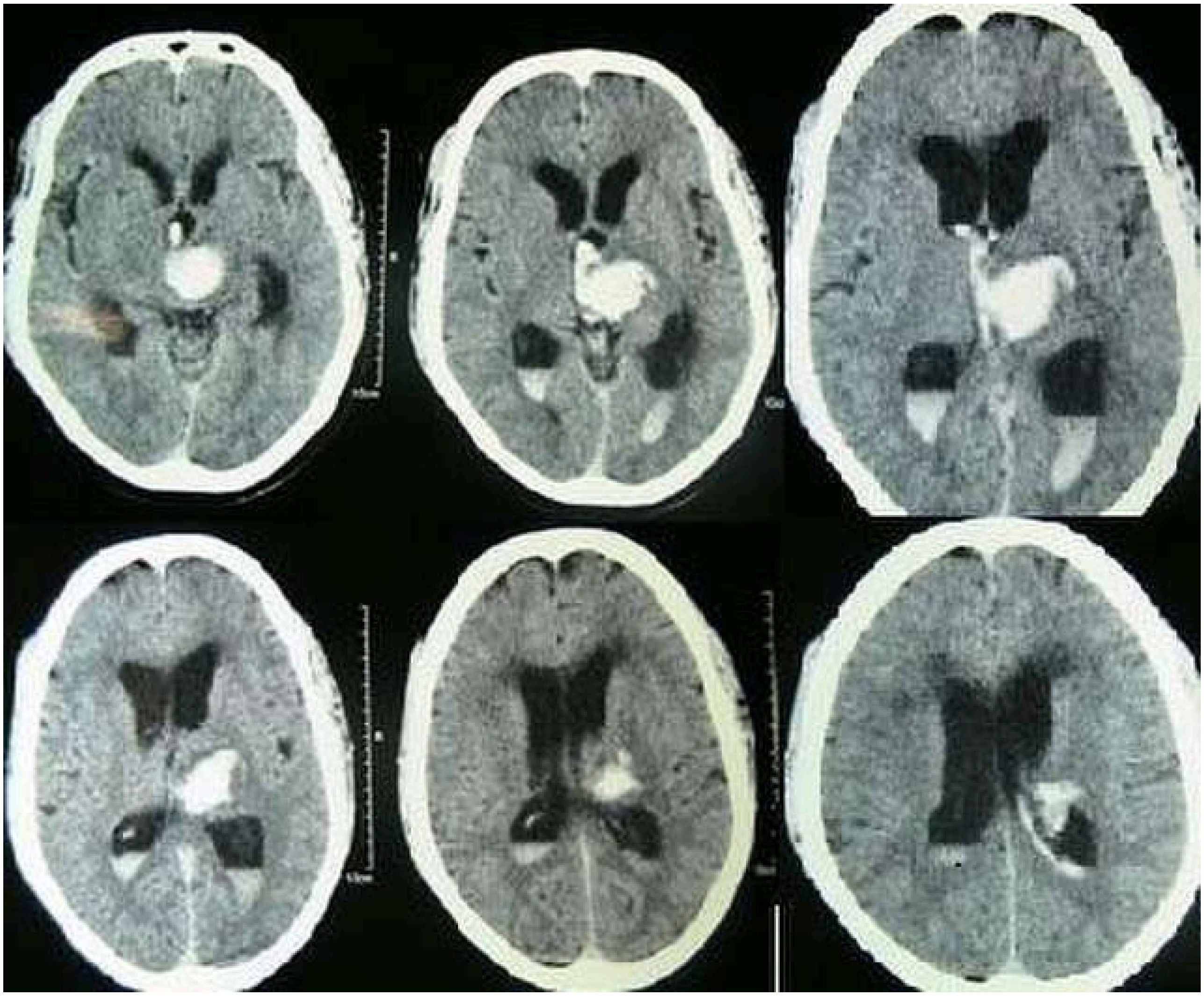

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What is the most likely diagnosis?

Brain tumour	1%
Intracerebral and intraventricular hemorrhage	82%
Intracerebral and subarachnoid haemorrhage	15%
Ischaemic stroke	1%
Subdural haemorrhage	1%

The correct answer is **intracerebral and intraventricular haemorrhage**. The CT shows bright hyper-dense intracerebral and intraventricular lesions, due to the presence of blood, which has a characteristic bright white appearance on CT imaging. An intracerebral hemorrhage is caused by the sudden rupture of a blood vessel within the brain. This leads to the accumulation of blood, causing damage to the surrounding brain tissue. Intraventricular hemorrhage represents bleeding inside the brain's ventricular system, which consists of interconnected spaces filled with cerebrospinal fluid (bleeding here is located within the lateral ventricles). Common presentations include a sudden severe headache, weakness, altered consciousness, and focal neurological deficits.

Brain tumour is incorrect. The rapid onset of symptoms in this case, paired with the CT findings and lack of previous history, are not in keeping with a brain tumour. Sometimes an intracerebral haemorrhage can be caused by an underlying brain tumour and further imaging is sometimes considered after the resolution of the haemorrhage.

Intracerebral and subarachnoid haemorrhage is an incorrect choice. While there are intraparenchymal and intraventricular haemorrhages on CT scans, no blood exists in the subarachnoid spaces. Subarachnoid haemorrhage typically results from the rupture of cerebral aneurysms, or, less commonly, from a sustained head trauma. Patients with subarachnoid haemorrhage typically present with a thunderclap headache, described as a sudden-onset headache usually occipital that is the worst headache of their life often accompanied by sensitivity to light (photophobia), signs of meningitis (meningism), and focal neurological deficits.

Ischaemic stroke although a potential differential, is ruled out in this case by the CT findings. The earliest CT sign visible for ischemic stroke is a hyperdense segment of a vessel, representing direct visualization of the intravascular thrombus/embolus and as such is visible immediately. Other signs include areas of low density in the grey and white matter of the territory. These changes may take time to develop and the CT may be normal initially. Clinically ischaemic stroke can be difficult to distinguish from haemorrhagic stroke.

Lastly, **subdural haemorrhage** is characterized by bleeding between the brain's surface and its outer covering, the dura mater, often caused by head trauma. They are thought to result from the stretching and tearing of bridging cortical veins as they traverse the subdural space to connect with a nearby dural sinus for drainage. It can lead to symptoms such as fluctuating levels of consciousness, headache, dizziness, and in severe cases coma. On CT, it presents with a crescent-shaped homogeneously hyperdense extra-axial collection that spreads diffusely over the affected hemisphere.

👍 👎 Discuss (3) Improve

Next question >

Head injury: types of traumatic brain injury ★

Basics

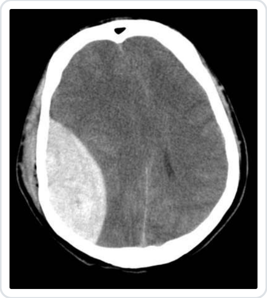
- primary brain injury may be focal (contusion/haematoma) or diffuse (diffuse axonal injury)
- diffuse axonal injury occurs as a result of mechanical shearing following deceleration, causing disruption and tearing of axons
- intra-cranial haematomas can be extradural, subdural or intracerebral, while contusions may occur adjacent to (coup) or contralateral (contre-coup) to the side of impact
- secondary brain injury occurs when cerebral oedema, ischaemia, infection, tonsillar or tentorial herniation exacerbates the original injury. The normal cerebral auto regulatory processes are disrupted following trauma rendering the brain more susceptible to blood flow changes and hypoxia
- the Cushings reflex (hypertension and bradycardia) often occurs late and is usually a pre terminal event

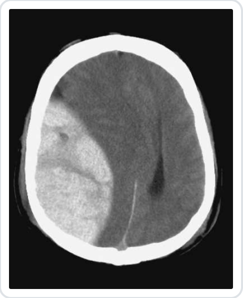
Type of injury	Notes
Extradural (epidural) haematoma	Bleeding into the space between the dura mater and the skull. Often results from acceleration-deceleration trauma or a blow to the side of the head. The majority of epidural haematomas occur in the temporal region where skull fractures cause a rupture of the middle meningeal artery. Features • features of raised intracranial pressure • some patients may exhibit a lucid interval
Subdural haematoma	Bleeding into the outermost meningeal layer. Most commonly occur around the frontal and parietal lobes. Risk factors include old age, alcoholism and anticoagulation.

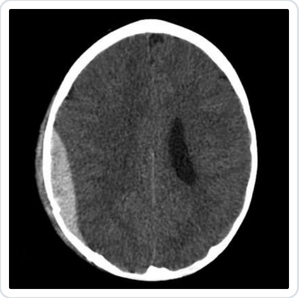
Type of injury	Notes
	Slower onset of symptoms than a epidural haematoma. There may be fluctuating confusion/consciousness
Subarachnoid haemorrhage	Classically causes a sudden occipital headache. Usually occurs spontaneously in the context of a ruptured cerebral aneurysm but may be seen in association with other injuries when a patient has sustained a traumatic brain injury
Intracerebral haematoma	An intracerebral (or intraparenchymal) haemorrhage is a collection of blood within the substance of the brain. Causes / risk factors include: hypertension, vascular lesion (e.g. aneurysm or arteriovenous malformation), cerebral amyloid angiopathy, trauma, brain tumour or infarct (particularly in stroke patients undergoing thrombolysis). Patients will present similarly to an ischaemic stroke (which is why it is crucial to obtain a CT in head in all stroke patients prior to thrombolysis) or with a decrease in consciousness. CT imaging will show a hyperdensity (bright lesion) within the substance of the brain. Treatment is often conservative under the care of stroke physicians, but large clots in patients with impaired consciousness may warrant surgical evacuation.

Image gallery

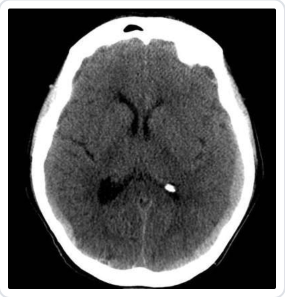
Extradural (epidural) haematoma:

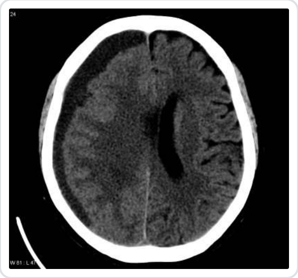


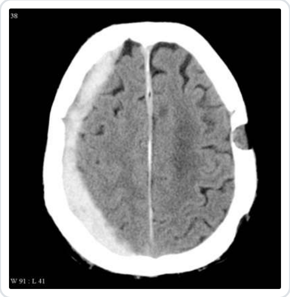




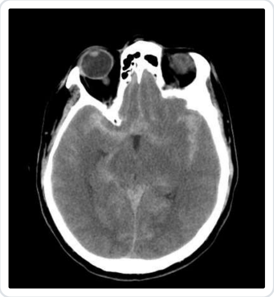
Subdural haematoma:

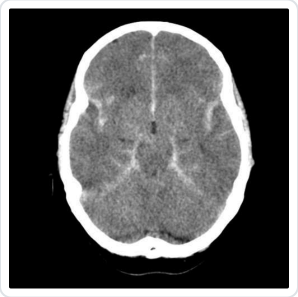






Subarachnoid haemorrhage:





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Next question

Textbooks

High-yield textbook

Extended textbook

Links

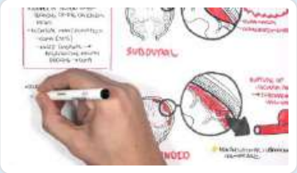
NICE

2014 Head injury guidelines

👍 11 👎 2

Suggest link Report broken link

Media



Intracranial Haemorrhage Types

Armando Hasudungan - YouTube 👍 6 👎 0

Intracranial
Bleeds
subdural
&
extradural

Intracranial bleeds - Subdural and Extradural haematoma

Soton Brain Hub - YouTube 👍 4 👎 0

TRAUMATIC
BRAIN INJURY
~ PATHOLOGY REVIEW ~
Osmosis.org

Traumatic brain injury: pathology review

Osmosis - YouTube 👍 4 👎 1

Report broken media

Score: **20.6%**

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Question 182 of 189

A 32-year-old woman presents with reduced sensation. She has noticed that slowly over six months she has not felt when hot water has splashed on her hands, despite blistering occurring afterwards. Her husband has become concerned and asked her to seek a medical opinion. She denies any other problems, including weakness, weight-loss or her activities of daily living being affected. She has a past medical history of asthma but only rarely needs her salbutamol inhaler. She has no other medications or allergies. On examination, she has sensory loss over her hands and arms when tested for temperature and pain. There is a dermatomal distribution affecting dermatomes C4 to C6 which is symmetrical. Cranial nerve and lower limb examination is normal. Spinal examination shows no tenderness. What is the most likely diagnosis?

- ○ Multiple sclerosis ×
- ○ Cervical disc prolapse ×
- ○ Vasculitis ×
- ○ Myasthenia gravis ×
- ○ Syringomyelia ×

Submit answer

Reference ranges

Score: **20%**

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Multiple sclerosis	3%
Cervical disc prolapse	4%
Vasculitis	2%
Myasthenia gravis	1%
Syringomyelia	90%

The correct answer is syringomyelia. The fact that she has primarily loss of spinothalamic function in a cervical nerve distribution with a distinct sensory loss makes syringomyelia the most probable diagnosis. Weakness can occur as well. Syringomyelia is caused by a tubular cyst within the central spinal cord, most commonly affecting the cervical region. It compresses corticospinal and spinothalamic tract as well as anterior horn cells. Presentation can be variable causing loss of temperature sensation, pain, paralysis, stiffness and weakness. Due to the common location of the cyst, this is most likely to affect hands, arms and shoulders. In half of the patients, there is only mild or no disability.

Multiple sclerosis is unlikely as there is no evidence of lesions separated by attacks. The absence of neck pain or tenderness makes a disc prolapse unlikely, whilst the absence of systemic features makes vasculitis unlikely. Myasthenia is associated with fatigability rather than a sensory loss.

Discuss (3) | Improve

Next question

Syringomyelia ★

Syringomyelia ('syrinx' for short) describes a collection of cerebrospinal fluid within the spinal cord.

Syringobulbia is a similar phenomenon in which there is a fluid-filled cavity within the medulla of the brainstem. This is often an extension of the syringomyelia but in rare cases can be an isolated finding.

Causes include:
- a Chiari malformation: strong association
- trauma
- tumours
- idiopathic

Features
- a 'cape-like' (neck, shoulders and arms)
  - loss of sensation to temperature but the preservation of light touch, proprioception and vibration
  - classic examples are of patients who accidentally burn their hands without realising
  - this is due to the crossing spinothalamic tracts in the anterior commissure of the spinal cord being the first tracts to be affected
- spastic weakness (predominantly of the lower limbs)
- neuropathic pain
- upgoing plantars
- autonomic features:
- Horner's syndrome due to compression of the sympathetic chain, but this is rare
  - bowel and bladder dysfunction
- scoliosis will occur over a matter of years if the syrinx is not treated

Investigations
- full spine MRI with contrast to exclude a tumour or tethered cord
- a brain MRI is also needed to exclude a Chiari malformation

Treatment will be directed at treating the cause of the syrinx. In patients with a persistent or symptomatic syrinx, a shunt into the syrinx can be placed.

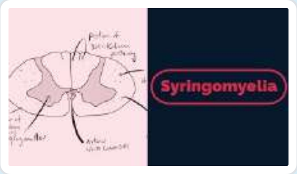
Next question >

Textbooks

High-yield textbook

Extended textbook

Media



Syringomyelia

Soton Brain Hub - YouTube 👍 11 👎 0

Report broken media

Score: **20.6%**

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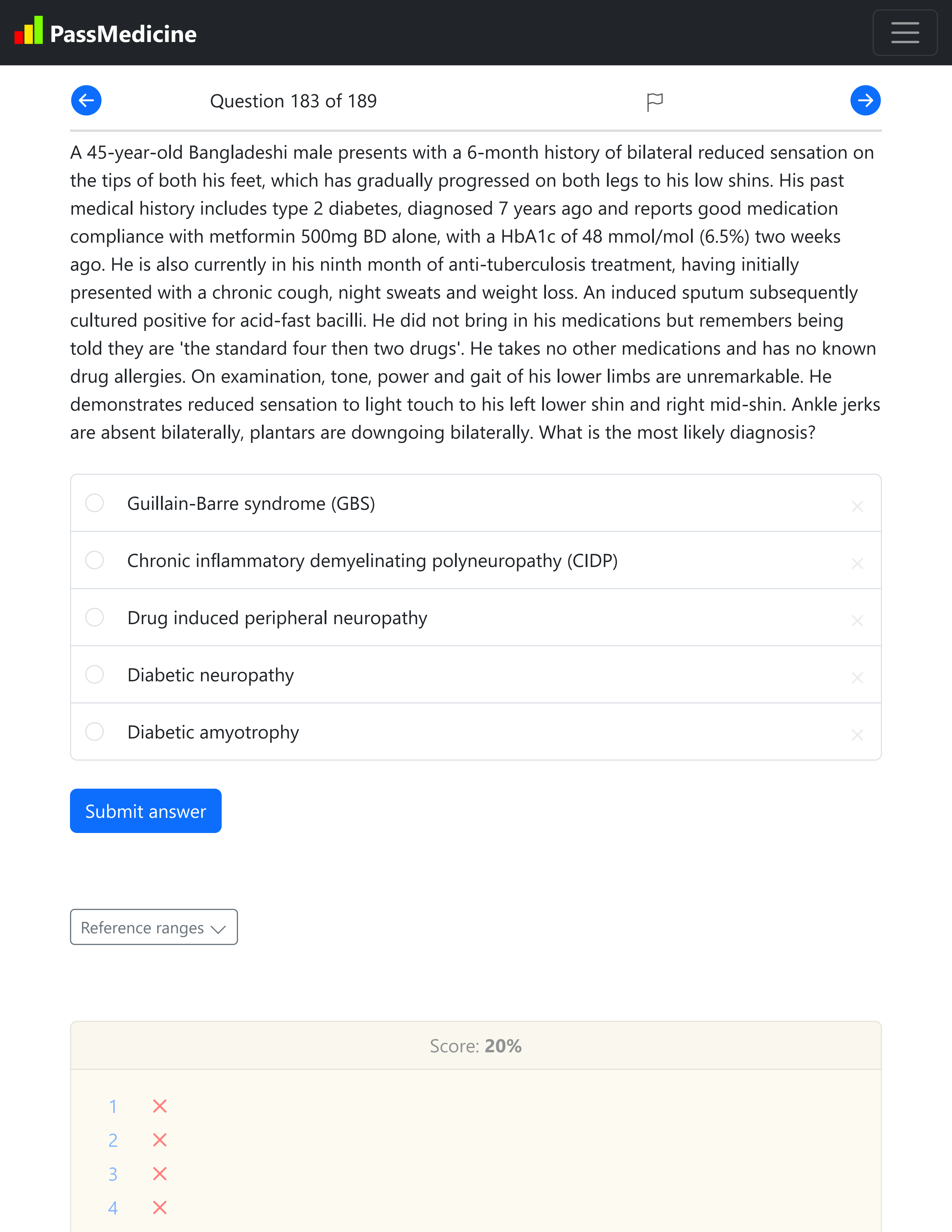
Back to top



Question 183 of 189

A 45-year-old Bangladeshi male presents with a 6-month history of bilateral reduced sensation on the tips of both his feet, which has gradually progressed on both legs to his low shins. His past medical history includes type 2 diabetes, diagnosed 7 years ago and reports good medication compliance with metformin 500mg BD alone, with a HbA1c of 48 mmol/mol (6.5%) two weeks ago. He is also currently in his ninth month of anti-tuberculosis treatment, having initially presented with a chronic cough, night sweats and weight loss. An induced sputum subsequently cultured positive for acid-fast bacilli. He did not bring in his medications but remembers being told they are 'the standard four then two drugs'. He takes no other medications and has no known drug allergies. On examination, tone, power and gait of his lower limbs are unremarkable. He demonstrates reduced sensation to light touch to his left lower shin and right mid-shin. Ankle jerks are absent bilaterally, plantars are downgoing bilaterally. What is the most likely diagnosis?

- ○ Guillain-Barre syndrome (GBS) ×
- ○ Chronic inflammatory demyelinating polyneuropathy (CIDP) ×
- ○ Drug induced peripheral neuropathy ×
- ○ Diabetic neuropathy ×
- ○ Diabetic amyotrophy ×

Submit answer

Reference ranges

Score: **20%**

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A 45-year-old Bangladeshi male presents with a 6-month history of bilateral reduced sensation on the tips of both his feet, which has gradually progressed on both legs to his low shins. His past medical history includes type 2 diabetes, diagnosed 7 years ago and reports good medication compliance with metformin 500mg BD alone, with a HbA1c of 48 mmol/mol (6.5%) two weeks ago. He is also currently in his ninth month of anti-tuberculosis treatment, having initially presented with a chronic cough, night sweats and weight loss. An induced sputum subsequently cultured positive for acid-fast bacilli. He did not bring in his medications but remembers being told they are 'the standard four then two drugs'. He takes no other medications and has no known drug allergies. On examination, tone, power and gait of his lower limbs are unremarkable. He demonstrates reduced sensation to light touch to his left lower shin and right mid-shin. Ankle jerks are absent bilaterally, plantars are downgoing bilaterally. What is the most likely diagnosis?

Guillain-Barre syndrome (GBS)	3%
Chronic inflammatory demyelinating polyneuropathy (CIDP)	12%
Drug induced peripheral neuropathy	81%
Diabetic neuropathy	3%
Diabetic amyotrophy	1%

The patient describes a chronic sensory deficit in a peripheral nerve distribution. This is most likely isoniazid-induced peripheral neuropathy, classically interfering with vitamin B6 via an unknown mechanism. As a result, pyridoxine should be regularly prescribed in all patients taking isoniazid. GBS and CIDP classically present as combined motor and sensory syndromes, of duration less than and more than 4 weeks respectively. While diabetes-related peripheral neuropathy is possible, his glycosylated haemoglobin measurement, an accurate measure of the patient's serum glucose levels over the past 3 months, reflects very good control.

Discuss (5) | Improve

Next question >

Drugs causing peripheral neuropathy ★

Drugs causing a peripheral neuropathy

- amiodarone
- isoniazid
- vincristine
- nitrofurantoin
- metronidazole

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Score: **20.6%**

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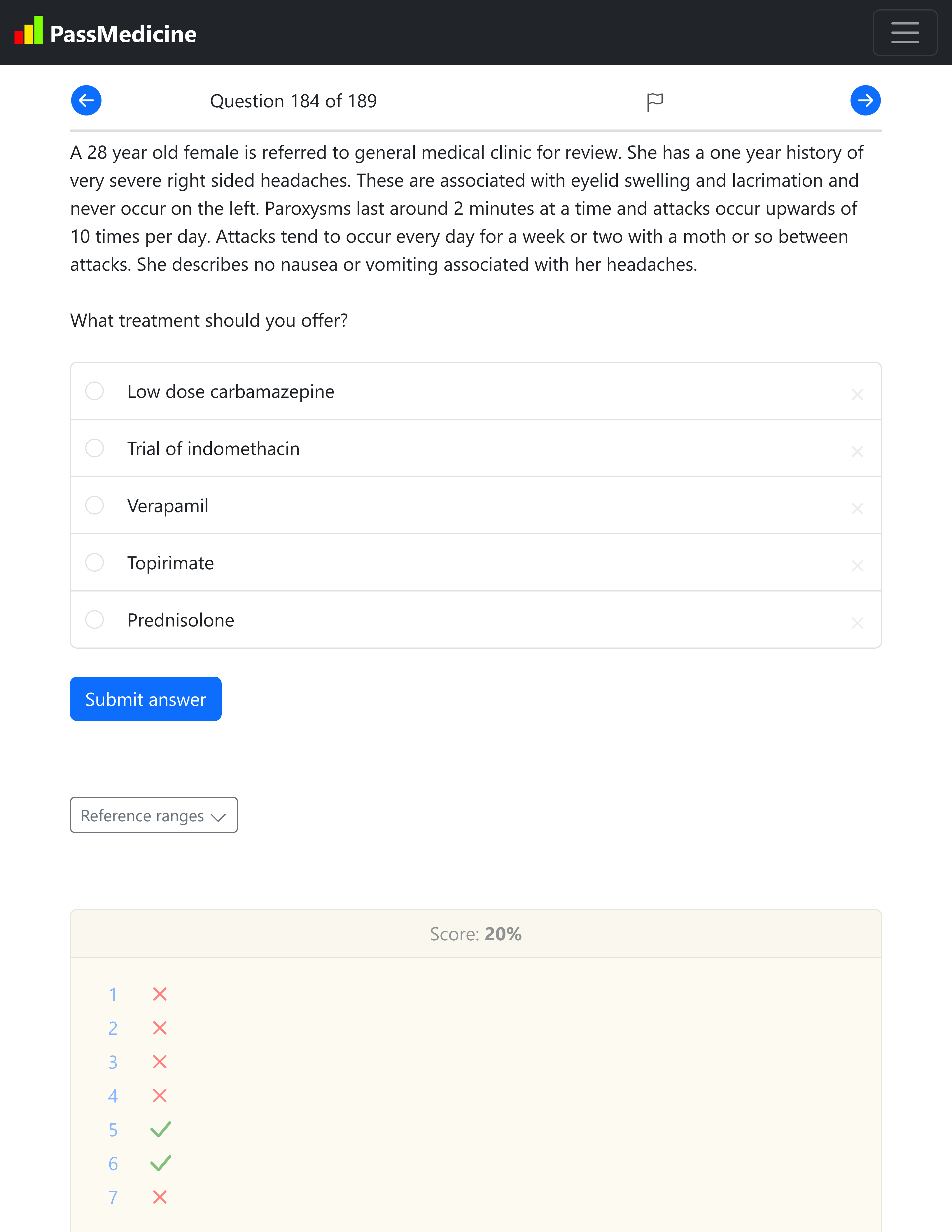
Back to top



Question 184 of 189

A 28 year old female is referred to general medical clinic for review. She has a one year history of very severe right sided headaches. These are associated with eyelid swelling and lacrimation and never occur on the left. Paroxysms last around 2 minutes at a time and attacks occur upwards of 10 times per day. Attacks tend to occur every day for a week or two with a moth or so between attacks. She describes no nausea or vomiting associated with her headaches.

What treatment should you offer?

- ○ Low dose carbamazepine
- ○ Trial of indomethacin
- ○ Verapamil
- ○ Topirimate
- ○ Prednisolone

Score: **20%**

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A 28 year old female is referred to general medical clinic for review. She has a one year history of very severe right sided headaches. These are associated with eyelid swelling and lacrimation and never occur on the left. Paroxysms last around 2 minutes at a time and attacks occur upwards of 10 times per day. Attacks tend to occur every day for a week or two with a moth or so between attacks. She describes no nausea or vomiting associated with her headaches.

What treatment should you offer?

Low dose carbamazepine	6%
Trial of indomethacin	58%
Verapamil	30%
Topirimate	6%
Prednisolone	1%

The history given above is one of severe, paroxysmal, unilateral headaches with associated autonomic features. The two most likely diagnoses would be cluster headache or paroxysmal hemicrania (PH), with the latter being more likely. It would be unusual for cluster headache attacks to last for less than 5 minutes and a daily frequency of greater than 10 is unusual for cluster headache but not PH. In addition, cluster headaches have a male predilection but PH tends to affect females.
Paroxysmal hemicrania (or chronic paroxysmal hemicrania if attacks occur for over one year) tends to be highly responsive to treatment with indomethacin, so much so that a trial of this may be considered a diagnostic strategy.

Low dose carbamazepine is the treatment of choice for trigeminal neuralgia. Verapamil is first line for prophylaxis of cluster headaches. Topiramate may be used in migraine prophylaxis and oral steroids have been used to prevent cluster headaches but have little evidence to support their use.

It may be argued that due to the relative rarity of PH compared with cluster headaches that even with the history above the former may be more likely, however due to the diagnostic response to indomethacin seen in PH it would be appropriate to trial this first.

Discuss (5) | Improve

Next question

Trigeminal autonomic cephalalgias

Trigeminal autonomic cephalalgias (TACs)

- cluster headache
- paroxysmal hemicrania
- hemicrania continua
- short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT syndrome)
- short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms (SUNA)

123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

NEUROLOGY
TRIGEMINAL AUTONOMIC
CEPHALALGIAS

Severe headaches - Understanding Trigeminal Autonomic Cephalgias - types, pathophysiology, treatment

Armando Hasudungan - YouTube 👍 10 👎 0

Report broken media

Score: **20.6%**

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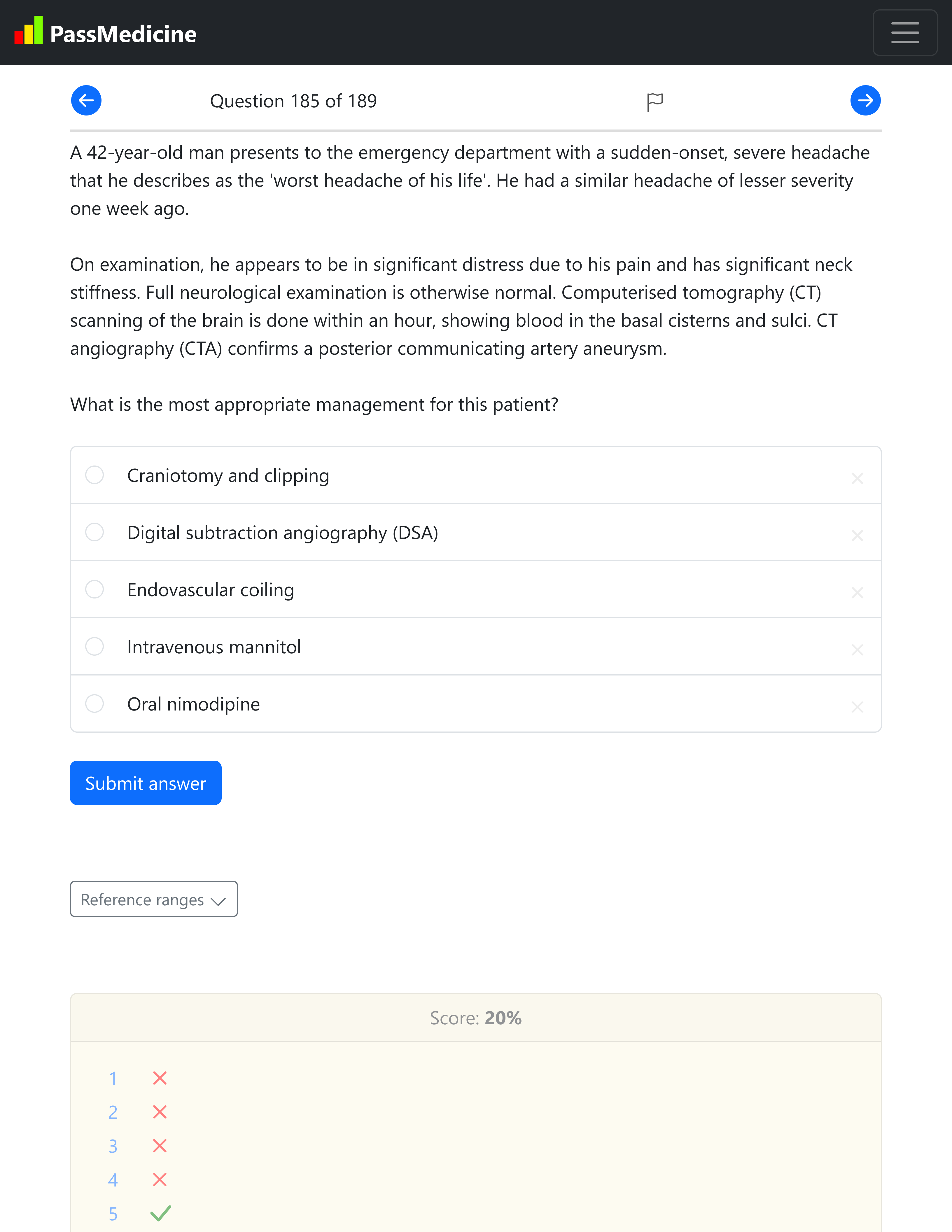

Question 185 of 189

A 42-year-old man presents to the emergency department with a sudden-onset, severe headache that he describes as the 'worst headache of his life'. He had a similar headache of lesser severity one week ago.

On examination, he appears to be in significant distress due to his pain and has significant neck stiffness. Full neurological examination is otherwise normal. Computerised tomography (CT) scanning of the brain is done within an hour, showing blood in the basal cisterns and sulci. CT angiography (CTA) confirms a posterior communicating artery aneurysm.

What is the most appropriate management for this patient?

- ○ Craniotomy and clipping ×
- ○ Digital subtraction angiography (DSA) ×
- ○ Endovascular coiling ×
- ○ Intravenous mannitol ×
- ○ Oral nimodipine ×

Score: **20%**

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A 48-year-old man presents to the emergency department with sudden onset, severe right eye pain and a headache. The pain started earlier this morning and worsened while watching television in a dark room. He also had 2 episodes of non-projectile vomiting. He has no significant past medical history except for migraine.

His vital signs are a temperature of 37 ºC, blood pressure 140/85 mmHg, pulse 55 beats/min and oxygen saturation 98% on room air. On examination, he is conscious and oriented but in distress due to severe pain. The pupils are unequal; the right pupil is semi-dilated and non-reactive to light.

Given the likely diagnosis, what is the definitive treatment for this patient?

- ○ Intravenous mannitol
- ○ Laser peripheral iridotomy
- ○ Laser trabeculoplasty
- ○ Oral propranolol
- ○ Tropicamide eye drops

Submit answer

Reference ranges

Score: **20%**

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A 48-year-old man presents to the emergency department with sudden onset, severe right eye pain and a headache. The pain started earlier this morning and worsened while watching television in a dark room. He also had 2 episodes of non-projectile vomiting. He has no significant past medical history except for migraine.

His vital signs are a temperature of 37 °C, blood pressure 140/85 mmHg, pulse 55 beats/min and oxygen saturation 98% on room air. On examination, he is conscious and oriented but in distress due to severe pain. The pupils are unequal; the right pupil is semi-dilated and non-reactive to light.

Given the likely diagnosis, what is the definitive treatment for this patient?

Intravenous mannitol	6%
🟩 Laser peripheral iridotomy	71%
Laser trabeculoplasty	14%
Oral propranolol	3%
🟥 Tropicamide eye drops	7%

Laser peripheral iridotomy is the definitive treatment for acute angle-closure glaucoma

Important for me Less important

This scenario describes a patient with acute angle-closure glaucoma, for which **peripheral laser iridotomy** is the definitive treatment option. A history of sudden-onset eye pain precipitated by mydriasis (watching TV in a dark room), vomiting, and pupillary findings in this scenario point towards the diagnosis. This is confirmed by measuring intra-ocular pressure via tonometry and visualisation of the angle by gonioscopy. Emergency management includes drugs that reduce the secretion of aqueous humour (acetazolamide and timolol) and drugs that increase the outflow of aqueous humour (pilocarpine and apraclonidine). Laser peripheral iridotomy is the definitive treatment option as it creates a tiny hole in the peripheral iris, allowing aqueous humour to flow to the angle.

Intravenous mannitol is not the definitive treatment for angle-closure glaucoma. Diuretics like mannitol and acetazolamide are sometimes used in medical management of acute angle-closure glaucoma to lower the intraocular pressure and symptomatic relief.

Laser trabeculoplasty lowers the intraocular pressure in open-angle glaucoma and has no role in treating acute angle-closure glaucoma. Unlike the patient's symptoms in this scenario, open-angle glaucoma exhibits no symptoms in the initial stage and only presents with problems in peripheral vision.

A sudden-onset headache and past history of migraine may suggest an acute migraine attack, which makes **oral propranolol** a reasonable option in this scenario, but a semi-dilated, non-reactive pupil points away from this.

Tropicamide eye drops will worsen this patient's symptoms as tropicamide causes pupillary block and worsens angle-closure glaucoma. Tropicamide is one of the causes of drug-induced acute angle-closure glaucoma.

👍 👎 Discuss Improve

Next question >

Acute angle-closure glaucoma ★

Glaucoma is a group of disorders characterised by optic neuropathy due, in the majority of patients, to raised intraocular pressure (IOP). It is now recognised that a minority of patients with raised IOP do not have glaucoma and vice versa.

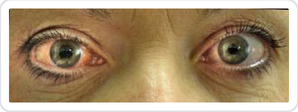
In acute angle-closure glaucoma (AACG) there is a rise in IOP secondary to an impairment of aqueous outflow. Factors predisposing to AACG include:

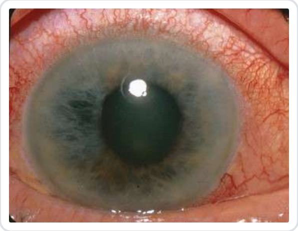
- hypermetropia (long-sightedness)
- pupillary dilatation
- lens growth associated with age

Features

- severe pain: may be ocular or headache
- decreased visual acuity
- symptoms worse with mydriasis (e.g. watching TV in a dark room)
- hard, red-eye
- haloes around lights

- semi-dilated non-reacting pupil
- corneal oedema results in dull or hazy cornea
- systemic upset may be seen, such as nausea and vomiting and even abdominal pain





Investigations

- tonometry to assess for elevated IOP
- gonioscopy (literally looking, *oscopy*, at the angle, *gonio*): a special lens for the slit lamp that allows visualisation of the angle

Management

The management of AACG is an emergency and should prompt urgent referral to an ophthalmologist. Emergency medical treatment is required to lower the IOP with more definitive surgical treatment given once the acute attack has settled.

There are no guidelines for the initial medical treatment emergency treatment. An example regime would be:

- combination of eye drops, for example:
    - a direct parasympathomimetic (e.g. pilocarpine, causes contraction of the ciliary muscle → opening the trabecular meshwork → increased outflow of the aqueous humour)
    - a beta-blocker (e.g. timolol, decreases aqueous humour production)
    - an alpha-2 agonist (e.g. apraclonidine, dual mechanism, decreasing aqueous humour production and increasing uveoscleral outflow)
- intravenous acetazolamide
    - reduces aqueous secretions
- some guidelines also recommend the use of topical steroids to reduce inflammation

Definitive management

- laser peripheral iridotomy
    - creates a tiny hole in the peripheral iris → aqueous humour flowing to the angle

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

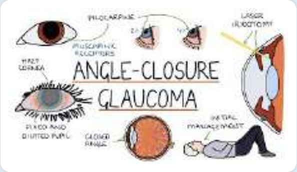
Patient.info 👍 27 👎 9

Acute angle closure glaucoma review

Suggest link

Report broken link

Media


PILOCARPINE
LASER IRIDOTOMY
MUSCARINIC RECEPTORS
HAZY CORNEA
ANGLE-CLOSURE
GLAUCOMA
FIXED AND DILATED PUPIL
CLOSED ANGLE
INITIAL MANAGEMENT

Acute angle closure glaucoma

Zero To Finals - YouTube 👍 4 👎 0

GLAUCOMA
Osmosis.org

Glaucoma (open-angle, closed-angle, and normal-tension)

Osmosis - YouTube 👍 6 👎 3

Report broken media

Score: **20.6%**

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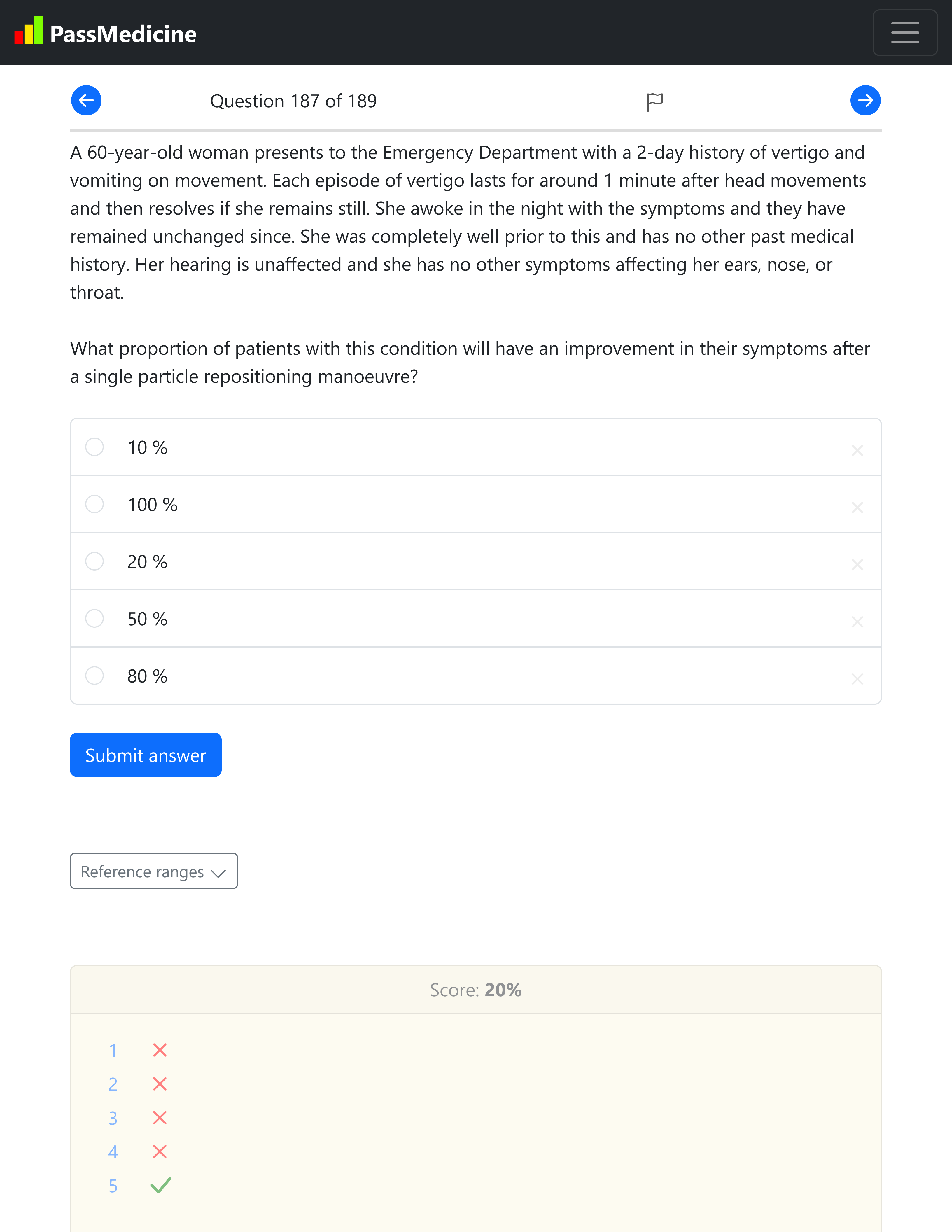

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Question 187 of 189

A 60-year-old woman presents to the Emergency Department with a 2-day history of vertigo and vomiting on movement. Each episode of vertigo lasts for around 1 minute after head movements and then resolves if she remains still. She awoke in the night with the symptoms and they have remained unchanged since. She was completely well prior to this and has no other past medical history. Her hearing is unaffected and she has no other symptoms affecting her ears, nose, or throat.

What proportion of patients with this condition will have an improvement in their symptoms after a single particle repositioning manoeuvre?

○ 10 %

○ 100 %

○ 20 %

○ 50 %

○ 80 %

Submit answer

Reference ranges

Score: **20%**

1 ✗
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4 ✗
5 ✓

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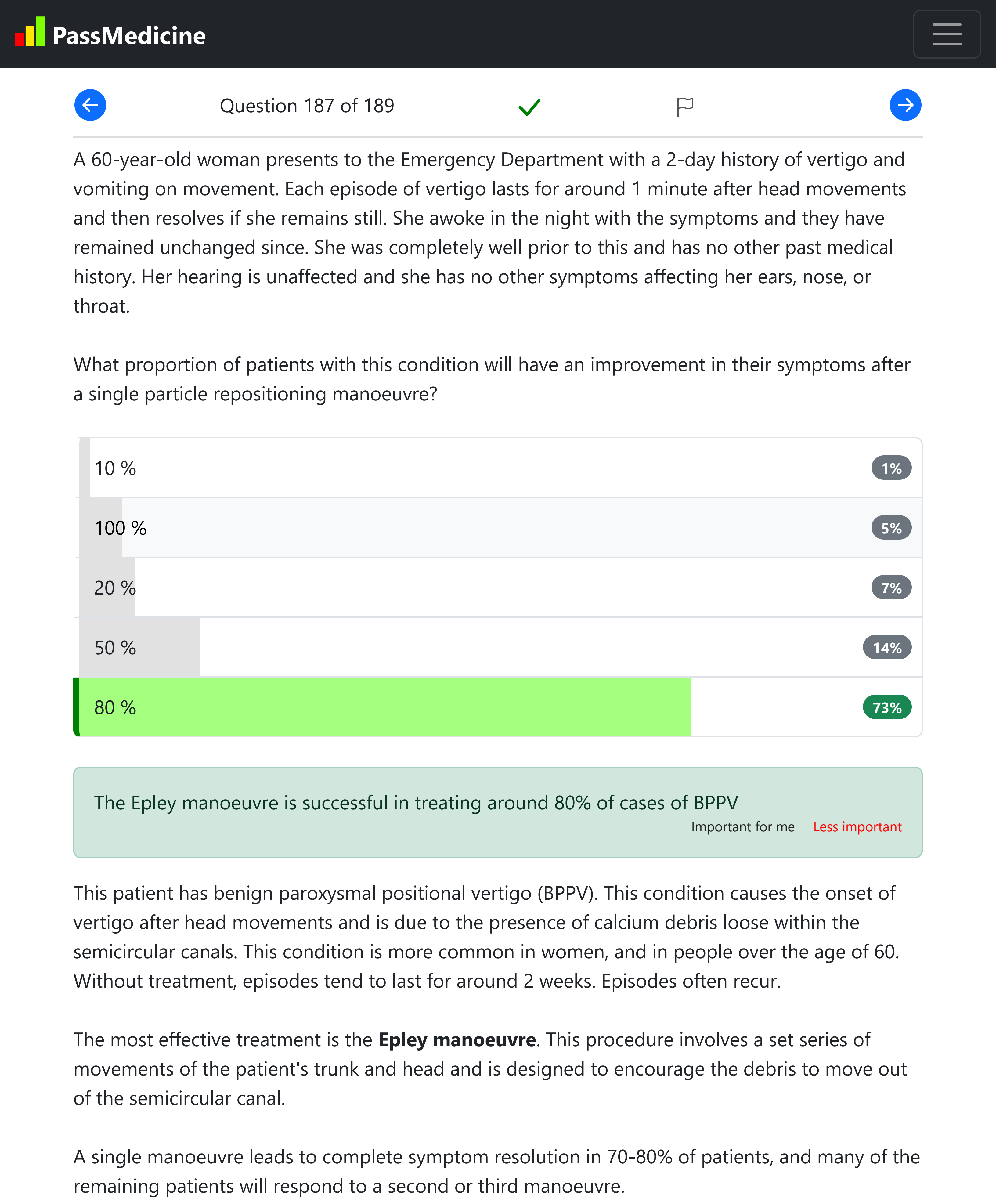


A 60-year-old woman presents to the Emergency Department with a 2-day history of vertigo and vomiting on movement. Each episode of vertigo lasts for around 1 minute after head movements and then resolves if she remains still. She awoke in the night with the symptoms and they have remained unchanged since. She was completely well prior to this and has no other past medical history. Her hearing is unaffected and she has no other symptoms affecting her ears, nose, or throat.

What proportion of patients with this condition will have an improvement in their symptoms after a single particle repositioning manoeuvre?

10 %	1%
100 %	5%
20 %	7%
50 %	14%
80 %	73%

The Epley manoeuvre is successful in treating around 80% of cases of BPPV

Important for me Less important

This patient has benign paroxysmal positional vertigo (BPPV). This condition causes the onset of vertigo after head movements and is due to the presence of calcium debris loose within the semicircular canals. This condition is more common in women, and in people over the age of 60. Without treatment, episodes tend to last for around 2 weeks. Episodes often recur.

The most effective treatment is the **Epley manoeuvre**. This procedure involves a set series of movements of the patient's trunk and head and is designed to encourage the debris to move out of the semicircular canal.

A single manoeuvre leads to complete symptom resolution in 70-80% of patients, and many of the remaining patients will respond to a second or third manoeuvre.

Discuss (1) Improve

Next question

Benign paroxysmal positional vertigo

Benign paroxysmal positional vertigo (BPPV) is one of the most common causes of vertigo encountered. It is characterised by the sudden onset of dizziness and vertigo triggered by changes in head position. The average age of onset is 55 years and it is less common in younger patients.

Features

- vertigo triggered by change in head position (e.g. rolling over in bed or gazing upwards)
- may be associated with nausea
- each episode typically lasts 10-20 seconds
- positive Dix-Hallpike manoeuvre
    - rapidly lower the patient to the supine position with an extended neck
    - a positive test recreates the symptoms of benign paroxysmal positional vertigo
    - rotatory nystagmus

BPPV has a good prognosis and usually resolves spontaneously after a few weeks to months. Symptomatic relief may be gained by:

- **Epley manoeuvre** (successful in around 80% of cases)
- teaching the patient exercises they can do themselves at home, termed vestibular rehabilitation, for example Brandt-Daroff exercises

Medication is often prescribed (e.g. Betahistine) but it tends to be of limited value.

Around half of people with BPPV will have a recurrence of symptoms 3-5 years after their diagnosis

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

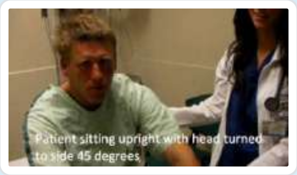
Clinical Knowledge Summaries 👍 33 👎 11

Benign paroxysmal positional vertigo guidelines

Suggest link

Report broken link

Media


Patient sitting upright with head turned to side 45 degrees

Hallpike Test and Epley Maneuver

Individual - YouTube 👍 14 👎 0

Report broken media

Score: **20.6%**

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189 ✓

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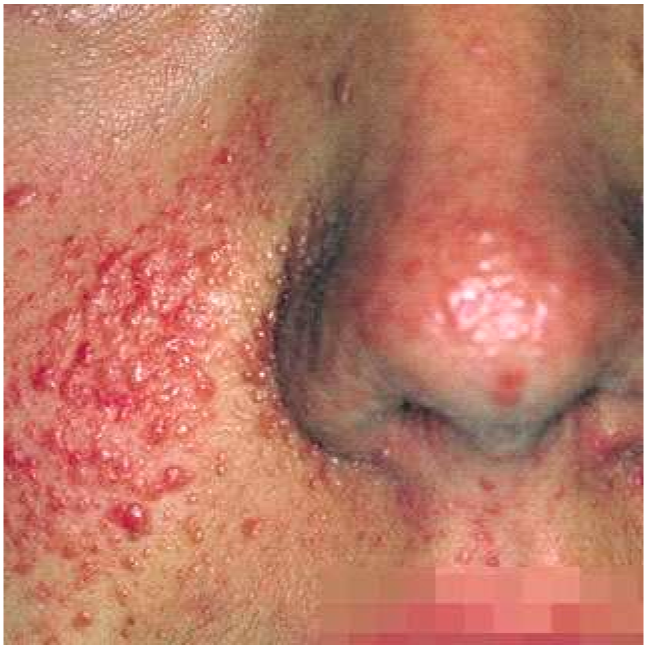
The patient below is being treated for epilepsy:



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What is the most likely underlying diagnosis?

- ○ HIV
- ○ Neurofibromatosis
- ○ Arteriovenous malformation
- ○ Tuberous sclerosis
- ○ Lennox-Gastaut syndrome

Submit answer

Reference ranges

Score: **20.5%**

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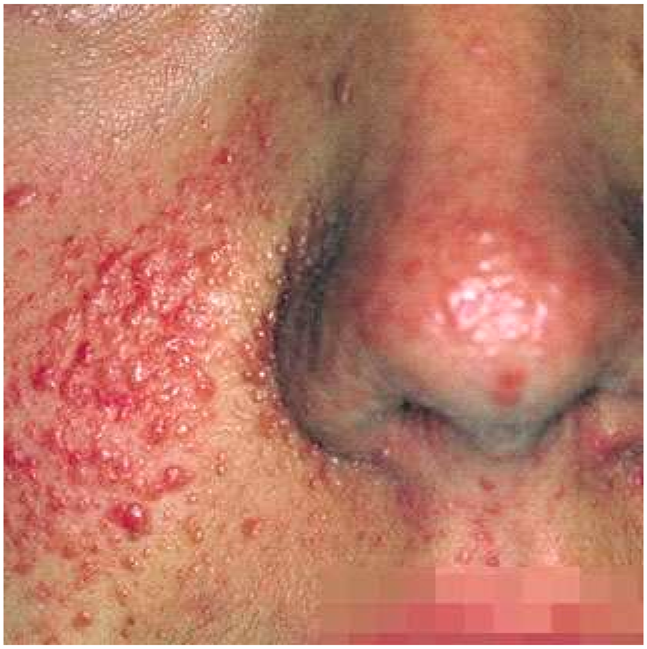
The patient below is being treated for epilepsy:



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What is the most likely underlying diagnosis?

HIV	2%
Neurofibromatosis	6%
Arteriovenous malformation	4%
Tuberous sclerosis	73%
Lennox-Gastaut syndrome	14%

Tuberous sclerosis - adenoma sebaceum

Important for me Less important

The correct answer is **Tuberous sclerosis**. Tuberous sclerosis is a genetic disorder that causes non-cancerous (benign) tumours to grow in different parts of the body. It often affects the brain and can lead to conditions such as epilepsy. The image shows characteristic adenoma sebaceum on the nose.

HIV is not the most likely underlying diagnosis in this case. While HIV can have neurological manifestations due to direct effects of the virus or secondary to opportunistic infections, it does not typically present with benign brain tumours. Neurological complications of HIV usually include cognitive impairment, myelopathy, neuropathy, and potentially opportunistic infections of the CNS like toxoplasmosis or progressive multifocal leukoencephalopathy (PML).

The next option, **Neurofibromatosis**, is a genetic disorder causing tumours to form on nerve tissue. These tumours can develop anywhere in your nervous system, including your brain and spinal cord. However, these tumours are typically neurofibromas and would have a different appearance on imaging compared to those seen in tuberous sclerosis.

Arteriovenous malformation (AVM) is an abnormal connection between arteries and veins, bypassing the capillary system. This vascular disorder disrupts normal blood flow and oxygen circulation. Brain AVMs could possibly cause seizures but they would create a different pattern on imaging studies than what is presented here.

Finally, **Lennox-Gastaut syndrome** is a type of childhood-onset epilepsy that results in multiple seizure types and cognitive decline. However, it does not cause benign brain tumours as seen in the provided image.

In conclusion, based on UK guidelines for managing patients with epilepsy and considering the characteristics shown in the image provided - benign brain tumours - Tuberous Sclerosis represents the most likely underlying diagnosis for this patient's condition.

👍 | 👎 | 💬 Discuss (1) | Improve

Next question ›

Tuberous sclerosis ★

Tuberous sclerosis (TS) is a genetic condition of autosomal dominant inheritance. Like neurofibromatosis, the majority of features seen in TS are neurocutaneous.

Cutaneous features

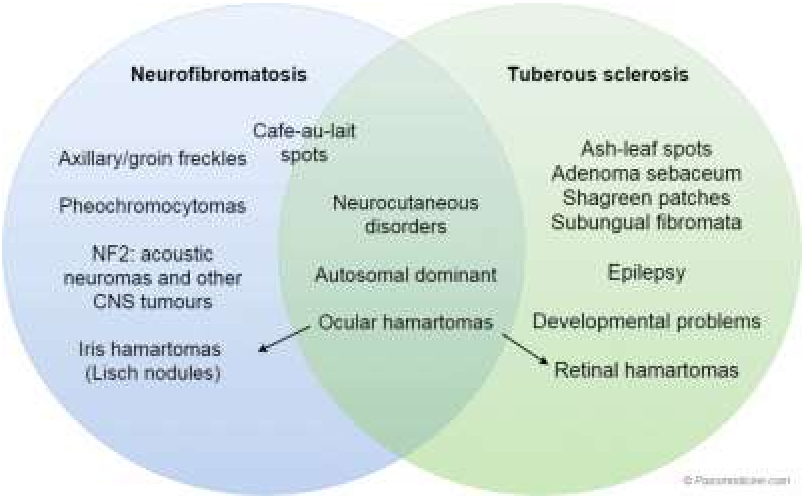
- depigmented 'ash-leaf' spots which fluoresce under UV light
- roughened patches of skin over lumbar spine (Shagreen patches)
- adenoma sebaceum (angiofibromas): butterfly distribution over nose
- fibromata beneath nails (subungual fibromata)
- cafe-au-lait spots* may be seen

Neurological features

- developmental delay
- epilepsy (infantile spasms or partial)
- intellectual impairment

Also

- retinal hamartomas: dense white areas on retina (phakomata)
- rhabdomyomas of the heart
- gliomatous changes can occur in the brain lesions
- polycystic kidneys, renal angiomyolipomata
- lymphangioleiomyomatosis: multiple lung cysts


Neurofibromatosis
Tuberous sclerosis
Cafe-au-lait spots
Axillary/groin freckles
Pheochromocytomas
NF2: acoustic neuromas and other CNS tumours
Iris hamartomas (Lisch nodules)
Neurocutaneous disorders
Autosomal dominant
Ocular hamartomas
Ash-leaf spots
Adenoma sebaceum
Shagreen patches
Subungual fibromata
Epilepsy
Developmental problems
Retinal hamartomas
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Comparison of neurofibromatosis and tuberous sclerosis. Note that whilst they are both autosomal dominant neurocutaneous disorders there is little overlap otherwise

*these of course are more commonly associated with neurofibromatosis. However a 1998 study of 106 children with TS found cafe-au-lait spots in 28% of patients

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 6 👎 12

Tuberous sclerosis review

Suggest link

Report broken link

Media

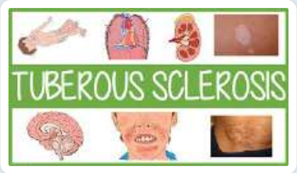
TUBEROUS SCLEROSIS
Osmosis.org

Tuberous sclerosis

Osmosis - YouTube 👍 11 👎 0

Angiomyolipoma

Osmosis - YouTube 👍 3 👎 0


TUBEROUS SCLEROSIS

Tuberous Sclerosis Complex

Townsend Teaching - YouTube 👍 3 👎 0

Tuberous
Sclerosis

Tuberous Sclerosis Complex (TSC)

Pixorize - YouTube 👍 2 👎 2

Report broken media

Score: **20.6%**

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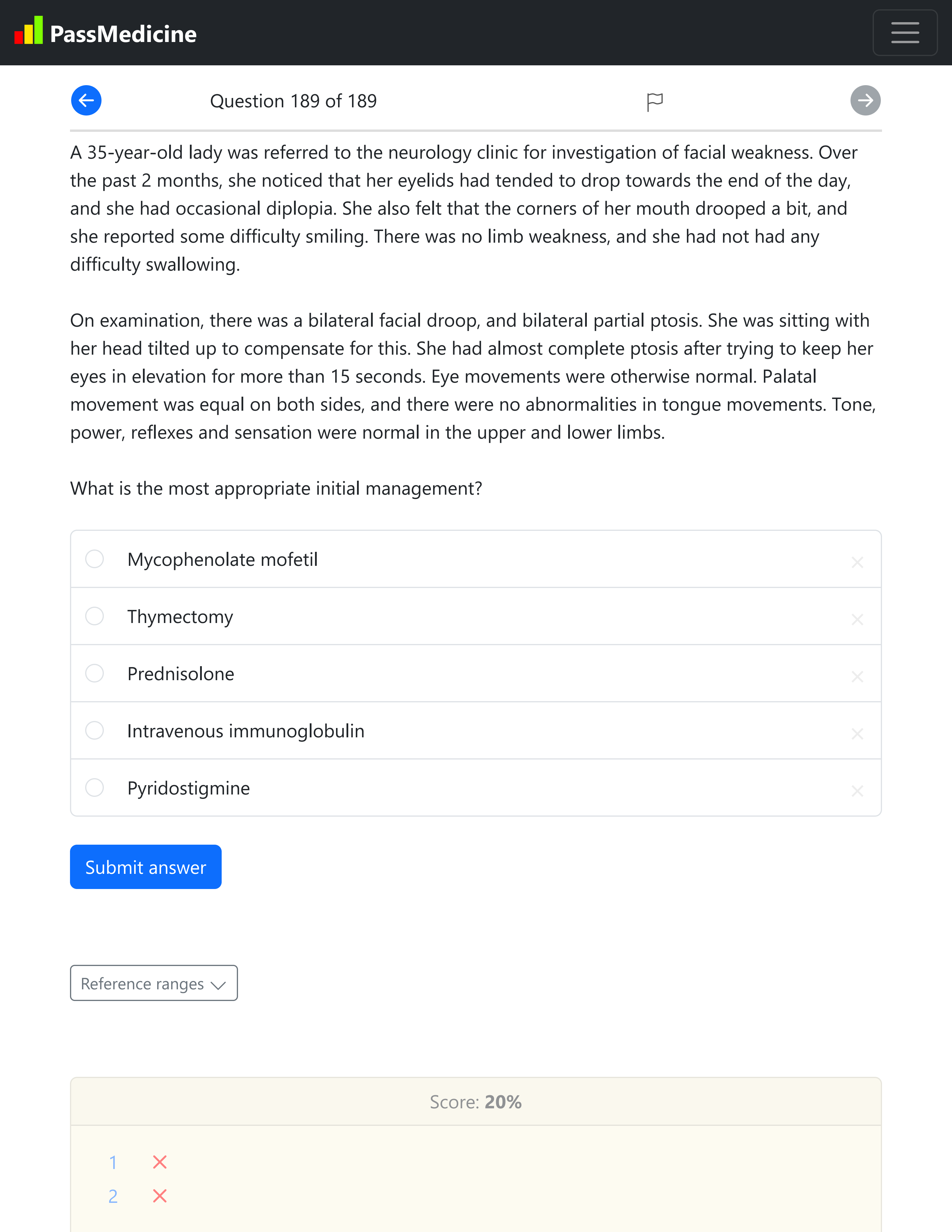

Question 189 of 189

A 35-year-old lady was referred to the neurology clinic for investigation of facial weakness. Over the past 2 months, she noticed that her eyelids had tended to drop towards the end of the day, and she had occasional diplopia. She also felt that the corners of her mouth drooped a bit, and she reported some difficulty smiling. There was no limb weakness, and she had not had any difficulty swallowing.

On examination, there was a bilateral facial droop, and bilateral partial ptosis. She was sitting with her head tilted up to compensate for this. She had almost complete ptosis after trying to keep her eyes in elevation for more than 15 seconds. Eye movements were otherwise normal. Palatal movement was equal on both sides, and there were no abnormalities in tongue movements. Tone, power, reflexes and sensation were normal in the upper and lower limbs.

What is the most appropriate initial management?

○ Mycophenolate mofetil ×

○ Thymectomy ×

○ Prednisolone ×

○ Intravenous immunoglobulin ×

○ Pyridostigmine ×

Score: **20%**

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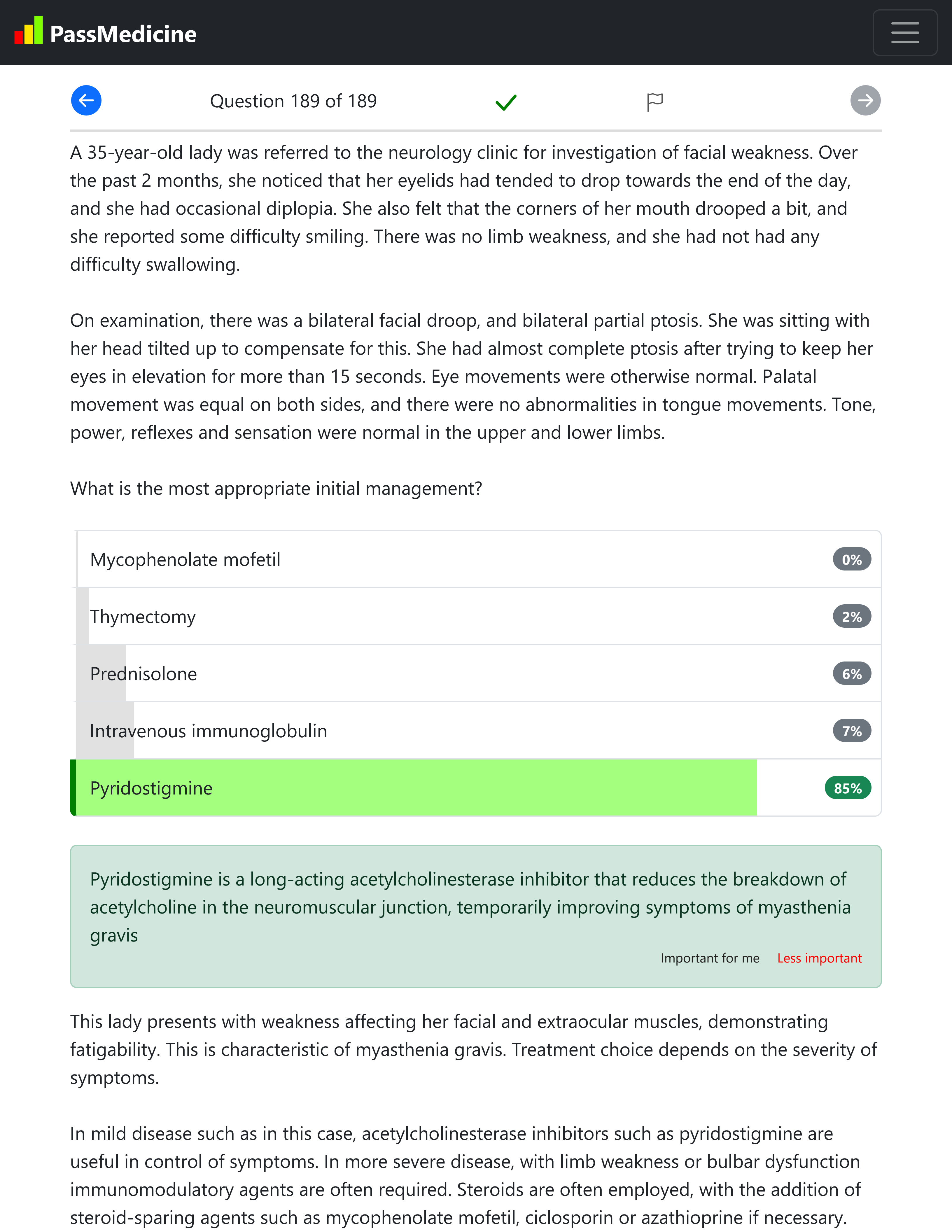

← Question 189 of 189 ✓ →

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On examination, there was a bilateral facial droop, and bilateral partial ptosis. She was sitting with her head tilted up to compensate for this. She had almost complete ptosis after trying to keep her eyes in elevation for more than 15 seconds. Eye movements were otherwise normal. Palatal movement was equal on both sides, and there were no abnormalities in tongue movements. Tone, power, reflexes and sensation were normal in the upper and lower limbs.

What is the most appropriate initial management?

Mycophenolate mofetil	0%
Thymectomy	2%
Prednisolone	6%
Intravenous immunoglobulin	7%
Pyridostigmine	85%

Pyridostigmine is a long-acting acetylcholinesterase inhibitor that reduces the breakdown of acetylcholine in the neuromuscular junction, temporarily improving symptoms of myasthenia gravis

Important for me Less important

This lady presents with weakness affecting her facial and extraocular muscles, demonstrating fatigability. This is characteristic of myasthenia gravis. Treatment choice depends on the severity of symptoms.

In mild disease such as in this case, acetylcholinesterase inhibitors such as pyridostigmine are useful in control of symptoms. In more severe disease, with limb weakness or bulbar dysfunction immunomodulatory agents are often required. Steroids are often employed, with the addition of steroid-sparing agents such as mycophenolate mofetil, ciclosporin or azathioprine if necessary.

Intravenous immunoglobulin and plasma exchange are useful in myasthenic crises. Thymectomy improves symptoms in cases associated with thymoma, and may also be beneficial in young patients with recent onset of symptoms, but would not be used as an initial treatment option.

👍 👎 💬 Discuss (3) Improve

Myasthenia gravis

Myasthenia gravis is an autoimmune disorder resulting in insufficient functioning acetylcholine receptors. Antibodies to acetylcholine receptors are seen in 85-90% of cases*. Myasthenia is more common in women (2:1)

The key feature is muscle fatigability - muscles become progressively weaker during periods of activity and slowly improve after periods of rest:
- extraocular muscle weakness: diplopia
- proximal muscle weakness: face, neck, limb girdle
- ptosis
- dysphagia

Associations
- thymomas in 15%
- autoimmune disorders: pernicious anaemia, autoimmune thyroid disorders, rheumatoid, SLE
- thymic hyperplasia in 50-70%

Investigations
- single fibre electromyography: high sensitivity (92-100%)
- CT thorax to exclude thymoma
- CK normal
- antibodies to acetylcholine receptors
  - positive in around 85-90% of patients
  - n the remaining patients, about about 40% are positive for anti-muscle-specific tyrosine kinase antibodies
- Tensilon test: IV edrophonium reduces muscle weakness temporarily - not commonly used any more due to the risk of cardiac arrhythmia

Management
- long-acting acetylcholinesterase inhibitors
  - pyridostigmine is first-line

- immunosuppression is usually not started at diagnosis, but the majority of patients eventually require it in addition to long-acting acetylcholinesterase inhibitors:
    - prednisolone initially
    - azathioprine, cyclosporine, mycophenolate mofetil may also be used
- thymectomy

Management of myasthenic crisis
- plasmapheresis
- intravenous immunoglobulins

*antibodies are less commonly seen in disease limited to the ocular muscles

+ Q 123

Textbooks

High-yield textbook

Extended textbook

Links

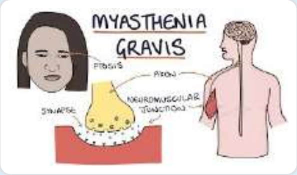
Postgraduate Medical Journal 👍 13 👎 3

Review of myasthenia gravis

Suggest link

Report broken link

Media


MYASTHENIA GRAVIS
PTOSIS
AXON
SYNAPSE
NEUROMUSCULAR JUNCTION

Myasthenia gravis

Zero to Finals - YouTube 👍 12 👎 1

NEUROLOGY
MYASTHENIA GRAVIS

Myasthenia gravis

Armando Hasudungan - YouTube 👍 1 👎 1

MYASTHENIA GRAVIS

Myasthenia gravis - causes, symptoms, treatment, pathology

Osmosis - YouTube 👍 2 👎 2

Report broken media

Score: **20.6%**

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